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# Infant Acute Life-Threatening Event—Dysphagic Choking Versus Nonaccidental Injury

Patrick D. Barnes, MD,\* John Galaznik, MD,<sup>†</sup> Horace Gardner, MD,<sup>‡</sup> and Mark Shuman, MD<sup>§</sup>

A 4-month-old male infant presented to the emergency room with a history of choking while bottle feeding at home, and was found by emergency medical services (EMS) to be apneic and pulseless. He subsequently developed disseminated intravascular coagulopathy and died. Computed tomography (CT) and magnetic resonance imaging (MRI) showed subdural hemorrhages (SDHs), subarachnoid hemorrhage (SAH), and retinal hemorrhages (RHs), along with findings of hypoxic-ischemic encephalopathy (HIE). The caretaker account appeared to be inconsistent with the clinical and imaging features, and a diagnosis of nonaccidental injury with “shaken baby syndrome” was made. The autopsy revealed diffuse anoxic central nervous system (CNS) changes with marked edema, SAH, and SDH, but no evidence of “CNS trauma”. Although NAI could not be ruled out, the autopsy findings provided further evidence that the child’s injury could result from a dysphagic choking type of acute life threatening event (ALTE) as consistently described by the caretaker.

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Nonaccidental injury, including “shaken baby syndrome,” is a “diagnosis” that is often considered in infants presenting with an acute life-threatening event (ALTE). Emergency physicians, family practitioners, and pediatricians are often the first to evaluate a child in this situation. Pediatric neurologists and neuroradiologists are often consulted in such cases. It has been previously accepted that in the absence of a history of significant trauma (ie, motor vehicle accident or 2-story fall), the “triad” of (1) infant encephalopathy, (2) SDH or SAH, and (3) RH is diagnostic of nonaccidental trauma/shaken baby syndrome based upon a rotational acceleration-deceleration trauma mechanism. This empirical formula has been challenged by evidence-based medicine and legal standards.<sup>1-12</sup>

We present a case of an infant with a dysphagic choking type of ALTE resulting in HIE identified at autopsy.<sup>13-16</sup> The case was also labeled as nonaccidental trauma/shaken baby syndrome.

## Case Report

### Clinical Course

A 4.5-month-old male infant with no previous health problems was being fed in bed with a formula bottle propped on his chest. The father left the room and returned to find the baby choking and blue. He turned the baby and tried to clear his airway. Nothing changed, so he patted him on the back. He then tried to blow in his mouth and “saw the baby’s stomach swell.” He then attempted to “pump his chest” trying to dislodge whatever the baby was choking on. The caregiver then attempted cardiopulmonary resuscitation (CPR), but had no prior training. After calling 911 for EMS, he said he then “ran next door to seek help from this neighbor” who also attempted CPR. EMS arrived and found the baby apneic and pulseless. The EMS records indicate that the infant was without a pulse for 30 minutes and anoxic/hypoxic for at least 40 minutes before effective respiration and circulation were restored.

On arrival to the emergency room (ER), the baby inadvertently extubated and was again asystolic. Clinically the infant was having bleeding from IV sites and his ears. Initial clotting studies at +4 hours were prothrombin time, 49.3; partial thromboplastin time, 198.4; and fibrinogen <20 mg/dL (209-440), documenting disseminated intravascular coagulopathy. By +50 minutes in the pediatric intensive care unit, the baby was hypothermic (88.3 F) and acidotic (pH, 6.726; base excess, -27.9) with anterior fontanelle bulging, but no external evidence of trauma. The child was being reperfused

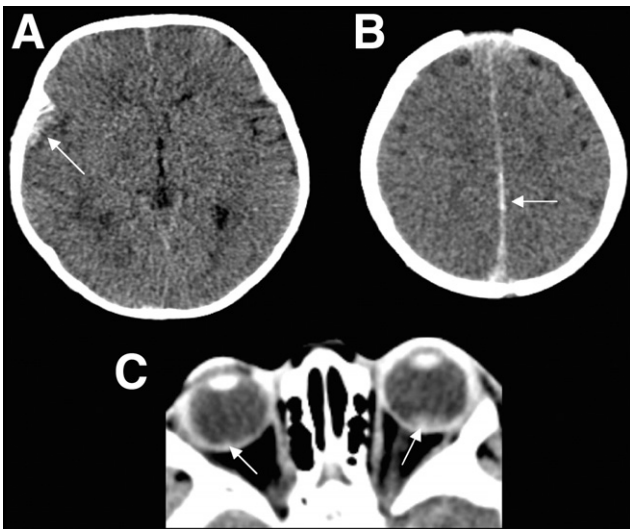
From the \*Department of Radiology, Lucile Salter Packard Children’s Hospital at Stanford, Palo Alto, CA.

<sup>†</sup>Student Health Center, College of Community Health Sciences, University of Alabama, Tuscaloosa, AL.

<sup>‡</sup>Ophthalmologist, Manitou Springs, CO.

<sup>§</sup>Forensic Pathology, Miami-Dade County Medical Examiner, Miami, FL.

Address reprint requests to Patrick D. Barnes, Department of Radiology, Stanford University Medical Center, Lucile Packard Children’s Hospital, 725 Welch Road, Palo Alto, CA 94304. E-mail: pbarnes@stanford.edu



**Figure 1** Initial brain CT (A-C) at + 1.5 hours shows bilateral cerebral low densities with decreased gray-white matter differentiation (edema) plus small high density convexity, interhemispheric, and peritentorial SAH and SDH (A, B, arrows). Also seen are asymmetric posterior intraocular hemorrhages about the optic nerve head (C, arrows).

with a blood pressure of 148/103. The clinical examination revealed a neurologically devastated infant consistent with severe brain swelling as well as a marked clotting disorder. The first eye examination at + 4 hours by a nonophthalmologist revealed bilateral RH. At + 16 hours, the ophthalmologist observed extensive bilateral RH and retinal elevation. The child died + 66 hours after organ harvesting.

### Imaging Evaluation

The initial brain CT (+ 1.5 hours) showed bilateral cerebral edema, along with small convexity, interhemispheric, and peritentorial SAH and SDH (Fig 1). There was slight widening of the sutures but no scalp or skull abnormality. Asymmetric posterior intraocular hemorrhages about the optic nerve head were also seen, left greater than right. The follow-up CT (+ 12 hours) showed progression of the cerebral edema and increased SAH/SDH plus increased ocular hemorrhages with blood levels (Fig 2). Also, because of the distribution of high densities along the dural venous sinuses, venous thrombosis could not be ruled out. The initial brain and orbit MRI (+ 19 hours) showed marked cerebral, cerebellar, and cervical spinal cord edema and restricted diffusion consistent with HIE and tonsillar herniation (Fig 3). A right frontal parasagittal extracerebral blood level was also present along with small hemorrhages (or thrombosis) in the same distribution as on the prior CT examinations. Bilateral ocular hemorrhages with blood levels were seen along with retinal detachments and retro-ocular, perioptic edema. The orbit and sinus CT (+ 44 h) showed bilateral pansinus and otomastoid congestion, bilateral ocular hemorrhages, and a question of orbital and cavernous sinus thromboses (not shown). These findings clearly progressed compared with the early CT examinations. The skeletal survey was negative except for a question of some right-sided rib fractures.

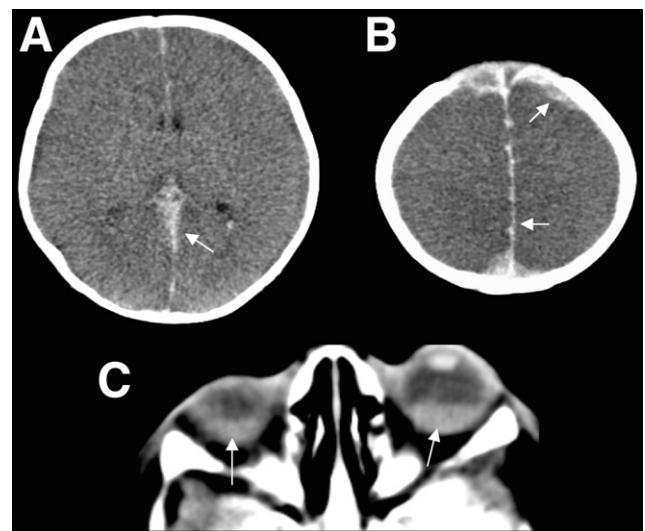
### Autopsy Findings

Fresh posterolateral fractures of the right 5th and 6th ribs were demonstrated about 2 inches in lateral to the costochondral junctions. No neck injury was demonstrated. There was wide suture separation and markedly bulging fontanelles, but no skull fracture. No autopsy was done of the eyes or orbits. On opening of the dura mater, there were bilateral small SDHs extending along the upper sagittal portions of the cerebral hemispheres plus thin layers of SDH over the right and left middle cranial fossae. SAH was also present, more so in the interhemispheric fissure, the upper portion of the cerebral convexities, and along the sylvian fissures. The brain weighed 925 g and was symmetrical. There was marked uncal grooving and cerebellar coning with herniation of the cerebellar tonsils into the upper cerebellar canal. Multiple coronal sections of the cerebrum, brain, cord, and cerebellum revealed an otherwise intact architecture but with marked swelling and diffuse softening, as seen with brain death. There was complete effacement of the ventricular system. Histological examination demonstrated “diffuse anoxic changes with marked interstitial edema.” “No additional pathological findings” were seen on other sections.

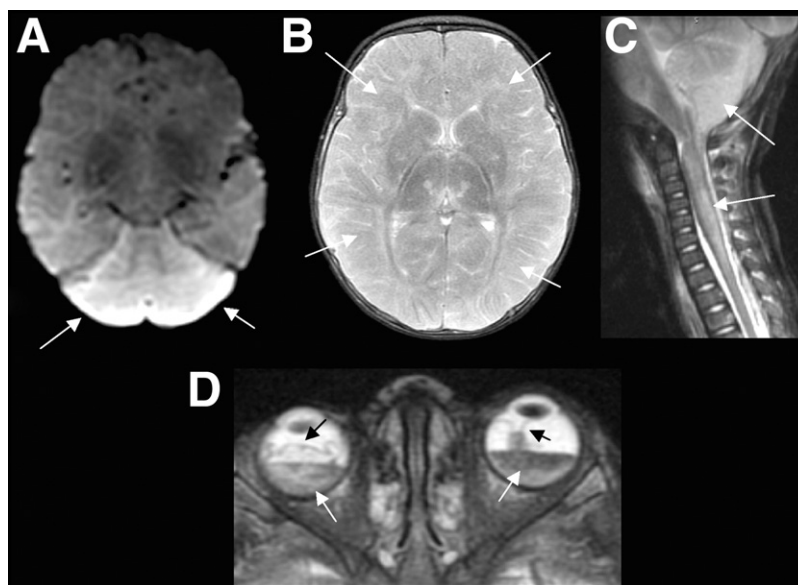
### Discussion

#### Acute Life-Threatening Event

ALTE has been defined as “an episode that is frightening to the observer and that is characterized by some combination of apnea (central or occasionally obstructive), color change (usually cyanotic or pallid but occasionally erythematous or plethoric), a marked change in muscle tone (usually marked limpness), choking, or gagging.”<sup>13-16</sup> Although the published data on the natural history of infants who present with ALTE are incomplete, many will never experience another event and will develop normally. Apnea refers to a cessation of



**Figure 2** Follow-up CT (A-C) at + 12 hours shows worsening edema with progression of the cerebral low densities and further loss of gray-white matter differentiation. There is some increase in the SAH/SDH (A, B, arrows) plus increased ocular hemorrhages with blood levels (C, arrows).



**Figure 3** MRI at + 19 hours with axial ADC map (A), axial T2 (B), sagittal T2 (C), and axial orbit T2 (D) shows marked T2- hyperintense cerebral, cerebellar, and cervical spinal cord edema (A-C, arrows), cerebral restricted diffusion (A, cerebral hypointensities relative to cerebellar hyperintensities), and tonsillar herniation. Bilateral ocular hemorrhages with blood levels (D, white arrows) are also seen along with retinal detachments (D, black arrows).

airflow and may result from central or obstructive causes. Central apnea is defined as the absence of respiratory effort caused by a lack of output from the central respiratory centers or by neuromuscular insufficiency. It results from a disruption in the generation or propagation of respiratory signals in the brainstem and descending neuromuscular pathways. Some causes include prematurity, head and neck trauma, and central hypoventilation syndrome. Chest wall movement will be absent and no breath sounds will be evident on auscultation of the chest.

Obstructive apnea is defined as breaths associated with paradoxical inverse movements of the chest wall and abdomen, with a corresponding decrease in oxygen saturation. It results from breathing through an occluded airway despite neuromuscular respiratory efforts. Some examples include Pierre Robin syndrome, adenotonsillar hypertrophy, aspirated foreign body, airway collapse (eg, laryngomalacia, cysts), and vocal cord paralysis. Mixed apnea has both central and obstructive features. There may be a central predisposition (eg, prematurity) with an obstructive challenge (eg, upper respiratory infection), or an obstructive predisposition (eg, Pierre Robin) with a central challenge (eg, sedation). Other examples include gastroesophageal reflux (GER), respiratory syncytial viral infection, and pertussis. In such cases, there may be complicated laryngobronchospasm or laryngeal chemoreceptor reflex central apnea.

### Dysphagic Choking

The history is the single most important component in the evaluation of infants with ALTE.<sup>13-16</sup> Central apnea, for example, with trauma (including nonaccidental) injury will appear as an effortless pause, whereas obstructive apnea in an awake or awakened infant presents as a sudden onset of

choking, gasping, coughing, or gagging, with or without an appearance of distress. The appearance of gastric contents may proceed, accompany, or follow the event and may be a causative or an associated phenomenon. A thorough history from a primary witness and the physical examination are keys to this diagnosis. Review of the 911 audio records may clearly reveal an infant in respiratory distress.

Choking, coughing, and gagging are normal protective responses to stimulation of the posterior nasopharynx, hypopharynx, larynx, and lower airway. Mechanical obstruction may occur from foreign material or from reflexive soft-tissue occlusion designed to prevent passage of the material. The forceful effort to expel the offending material prevents effective ventilation. These forceful efforts may cause plethora and erythema of the face and head because the increased intrathoracic pressures that are generated cause increased blood flow and venous congestion superiorly. Sustained efforts may result in hypoxia and limpness caused by vagal stimulation. Typically, the coughing, gagging, and retching responses are self-limited when the offending stimulus is removed. When paroxysmal and sustained, such responses may result in severe injury or death, as may be classically seen with pertussis.<sup>13-21</sup>

The sequence of events in dysphagic apnea starts with aspiration of a feed or reflux causing paroxysmal coughing/choking. This produces a dramatic rise in intrathoracic pressure, which is transmitted directly to the intracranial contents during which intracranial vasculature becomes overdilated and damaged. If the buildup in venous pressure is sufficient, there may be disruption of the blood brain barrier along with SDH, SAH, and RH.<sup>17-21</sup> Hemorrhagic retinopathy occurs in a variety of situations in which raised intrathoracic pressure is communicated to intracranial vessels.<sup>21-27</sup> Because both the arterial and the venous circulation

of the eye follow those of the brain, intraluminal pressure in retinal vessels follows the intraluminal pressure of the intracranial vasculature. In paroxysmal coughing, intracranial vascular pressures continually rise as blood accumulates both by increased arterial pressures driving blood into the cranium and by the markedly elevated superior vena cava pressures opposing venous outflow from the cranium. These escalating pressures create the physiological conditions necessary for SDH and SAH to occur, as well as RH, particularly in the infant.<sup>17</sup> Laryngeal constriction caused by the same glottic/laryngeal irritation may then progress to upper airway closure with bronchospasm and obstructive apnea. The resulting prolonged severe hypoxia causes the already damaged brain to swell further. Computer modeling of the pathophysiological processes in a dysphagic choking event resulting in paroxysms of coughing/choking, similar to that reported with pertussis, demonstrates that conditions necessary for the generation of SDH, SAH, and RH may be present.<sup>17</sup> The clinical, imaging, and autopsy findings in this case are consistent with the computer model's predictions.

### Non-Accidental Injury/Shaken Baby Syndrome

In this case, the initial "diagnosis" of nonaccidental injury/shaken baby syndrome was based upon the heretofore classic "triad" of SDH, RH, and encephalopathy, along with a history presumed to be inconsistent with the injuries. CNS findings that mimic nonaccidental injury/shaken baby syndrome have been reported in accidental trauma and in many medical conditions.<sup>28-35</sup> The latter includes infection, coagulopathy, metabolic disorders, and others.<sup>33-35</sup> More recent reports also show that there is no specific pattern of intracranial hemorrhage that is diagnostic of nonaccidental injury/shaken baby syndrome, to include interhemispheric SDH and mixed density SDH.<sup>28-31</sup> Furthermore, recent evidence-based medical reviews (and legal challenges) of the past nonaccidental injury/shaken baby syndrome literature reveal that the vast majority of these publications failed to achieve quality of evidence ratings that would merit the use of the "triad" as a standard or guideline for proof of nonaccidental injury/shaken baby syndrome.<sup>1-12</sup> Although initial concern for nonaccidental injury is important and must be reported, medical personnel must carefully correlate such findings with the history to establish a correct sequence of events, including predisposing factors.<sup>33-35</sup> The initiation of the criminal process before a complete and thorough child protection and medical evaluation can lead to a rush in judgment. The injuries in this particular case were attributed to shaken baby syndrome before the brain injuries were completely evaluated. The father of the victim was charged with fatally shaking the child. Given the fact that the law requires physicians to report suspected nonaccidental injury, there is the danger of assuming nonaccidental injury in all cases of SDH and RH. As a result, further medical and imaging workup may not be pursued (eg, MRI of the brain and cervical spine). The American Academy of Pediatrics, as others, strongly endorses the use of MRI in cases of suspected nonaccidental injury.<sup>33-36</sup>

### Hypoxic-Ischemic Vs Traumatic Brain Injury

The gross and microscopic examination in this case showed only the effects of severe HIE, venous hypertension (HTN), and coagulopathy.<sup>37-39</sup> This is consistent with the history of infantile dysphagic choking as consistently provided by the caretaker. There was no evidence of "primary" traumatic injury (ie, contusion or shear injury). There was neither any traumatic neck or spinal cord injury nor any evidence of traumatic scalp or skull injury. Such injury would be expected to produce central apnea, not obstructive apnea. The only evidence of traumatic injury were the acute unilateral rib fractures that could certainly be related to the multiple CPR attempts.

### Subdural, Subarachnoid, and RH

There are many potential causes for the SDH, SAH, and RH. These include trauma, infection, coagulopathy, increased intracranial pressure (ICP), ischemic endothelial damage, and reperfusion.<sup>33-35</sup> There was no impact or other traumatic injury of the CNS or head and neck in this case, nor was there any indication of infection. Coagulopathy was present as supported by laboratory findings. This is a known phenomenon that may be initiated by tissue injury because of trauma or hypoxia-ischemia.<sup>40,41</sup> After the capillary beds are open and leaking, further increases in ICP from brain edema and CPR may exacerbate this process. Geddes et al suggest that additional factors, such as venous and arterial HTN may exacerbate hemorrhage in the ischemic, swollen brain with increased ICP.<sup>39</sup> They propose both increased oozing from hypoxic veins in the setting of venous HTN secondary to severe edema, and increased hemorrhage from episodic or sustained arterial HTN (eg, with reperfusion) that may occur as a part of Cushing's triad or be neurogenic in origin. Additionally, choking, vomiting, or paroxysmal coughing (eg, pertussis) may also result in SDH and RH.<sup>17</sup> Furthermore, the distribution of SDH or SAH along the interhemispheric fissure is not pathognomonic for nonaccidental injury, as previously reported, and has been shown to occur in cases of accidental trauma and HIE.<sup>28-32,35</sup> The definitive fundoscopic documentation of RH was not made until the child was in the pediatric intensive care unit. Given the course of events to that point, the RH may be a result of multiple factors as described earlier regarding SDH and SAH. RH is a known manifestation of increased ICP. There is no single type or pattern of RH that is pathognomonic of nonaccidental injury/shaken baby syndrome, and RH is reported in several conditions.<sup>42-50</sup>

### Conclusions

Physicians have an obligation to completely and timely evaluate suspected nonaccidental injury, including its mimics. The imaging findings alone cannot distinguish nonaccidental injury from accidental injury, or from the medical mimics. A complete and thorough medical evaluation, using evidence-based medicine principles, is necessary in parallel with the child protection assessment. A multidisciplinary approach to this evaluation is also important, including the involvement of qualified specialists. Such an approach may help to differentiate between appro-



appropriate child protection vs the improper break up of a family or a wrongful indictment and conviction. When evaluating cases of fatal ALTE in which SDH, RH, and encephalopathy (eg, HIE) are found, a clinical history of, or evidence for, dysphagic choking may be significant regarding etiology.

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