Nonaccidental Head Injury Is the Most Common Cause of Subdural Bleeding in Infants <1 Year of Age

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KEY WORDS

abusive head trauma, subdural bleeding, nonaccidental head injury, infant, forensic neuropathology

ABBREVIATIONS

SDB—subdural bleeding
NAHI—nonaccidental head injury
SIDS—sudden infant death syndrome

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WHAT'S KNOWN ON THIS SUBJECT: SDB in infants and toddlers usually is considered a typical sign of NAHI. This view has been challenged by the hypothesis that SDB might be a consequence of hypoxia or brain swelling instead of traumatic tearing of bridging veins.

WHAT THIS STUDY ADDS: In our study population, unexplained SDB according to the unified hypothesis was an extreme rarity. Furthermore, traumatic SDB occurring in the context of NAHI was the most common subgroup of possible causes of infantile SDB.

abstract

OBJECTIVE: Subdural bleeding (SDB) in infants is considered an essential symptom of nonaccidental head injury (NAHI). Recently, this view has been challenged by the "unified hypothesis," which claims that SDB in infants is related to hypoxia and brain swelling rather than to traumatic shearing of bridging veins. We analyzed a large series of infants' autopsies for the presence and causes of SDB, which should be a common event according to the unified hypothesis.

METHODS: Autopsy, clinical, and legal information for infants <1 year of age from a single institution over 50 years were analyzed regarding cause of death, presence, morphology, and cause of SDB, and brain weight.

RESULTS: From a total of 16 661 autopsies during the study period, 715 (4.3%) involved infants <1 year of age. Fifty (7.0%) of those had SDB. NAHI was identified in 17 patients. The most common cause of SDB was trauma (15 cases [30.0%]), with NAHI accounting for 14 cases. SDB was present in 82.4% of patients with NAHI but only 5.2% of infants with other causes of death. Four patients (8.0%) had unexplained SDB with no discernible cause of bleeding. Statistical analysis did not reveal any correlation between the presence of SDB and brain weight.

CONCLUSIONS: In the study population, unexplained SDB in infants was an extreme rarity. Moreover, a correlation between brain swelling and the presence of SDB could not be drawn. Our data argue strongly against the unified hypothesis and strengthen the association between SDB and NAHI in infancy. *Pediatrics* 2009;124:1587–1594

Severe nonaccidental head injury (NAHI) in infants is most commonly characterized by the combination of acute encephalopathy, subdural bleeding (SDB), and retinal hemorrhage occurring in a context of inappropriate or inconsistent history.[1–3] Lethality and morbidity are serious, with 12% to 30% of infants dying and 60% to 70% of survivors experiencing significant neurologic deficits.[4–7] There is ongoing controversy regarding the precise mechanisms of brain injury, the cause of death, the degree of force necessary, and whether direct impact is needed.[8–15]

SDB is a relatively uncommon event in infants, with reported incidences of 20 to 25 cases per 100 000 infants <1 year of age.[4,16–18] It is generally thought that the majority of SDB cases involving infants are attributable to NAHI in which abrupt acceleration/deceleration forces applied to the skull and its contents lead to tearing of bridging veins.[18–20] Other causes for infantile SDB include severe accidental trauma, infections, coagulation disorders, traumatic labor, and rare metabolic disorders.[21] Because every case of unexplained SDB in infants raises immediate concerns about the possibility of NAHI, the differential diagnosis of SDB in infants is crucial.[21,22]

The classic theory of SDB, its pathogenesis, and its association with NAHI has been challenged by the "unified hypothesis." This theory postulates that SDB in infants may result from a combination of severe hypoxia, brain swelling, and increased central venous pressure, causing blood to leak from intracranial veins into the subdural space.[23,24] Because of the enormous medicolegal implications of the concept of NAHI, the unified hypothesis has become the center of an ongoing debate.[1,23,25–30] We hypothesized that, if hypoxia and brain swelling lead to SDB, as claimed by the unified hypothesis, then SDB should be a common finding in infants' autopsies, in which both hypoxia and brain swelling are noted frequently. Therefore, we investigated a large series of infants' autopsies for the presence, morphologic features, and causes of SDB. Statistical analysis of associations between the presence of NAHI, SDB resulting from other causes, and brain swelling (determined on the basis of brain weight) was performed.

METHODS

Records of autopsies performed at the Institute of Neuropathology (Hamburg, Germany) between 1956 and 2005 were reviewed, and infants <1 year of age were identified. The records were analyzed with regard to clinical data and pathologic and neuropathologic findings, including brain weight, cause of death, histologic features, and the presence of SDB and NAHI. If available, records from investigative authorities, court proceedings, and expert witnesses were used. Patients whose records were lacking data indispensable for this study were excluded.

The causes of death were grouped into the following categories: malformation, perinatal conditions, infection, metabolic disorder, NAHI, other nonnatural deaths, sudden infant death syndrome (SIDS) and unclassified sudden infant death, other causes, or unclear causes. The diagnoses of SIDS and unclassified sudden death were assigned by using the most-recent diagnostic criteria published by an expert panel of pediatric and forensic pathologists and pediatricians.[31] The category of other causes included known medical conditions considered too rare to constitute a category of their own. The cause of death was deemed unclear in cases in which the available information would not allow categorization.

NAHI was identified on the basis of a confession by the perpetrator, a conviction for child abuse in a criminal court (with or without a confession), or a unanimous conclusion of all authors at a case conference, with the use of the following criteria for a positive decision: signs of serious external injury (hematomas or lacerations), unexplained fractures (long bones, ribs, or skull), SDB, traumatic intracerebral pathologic conditions (simple contusions or gliding contusions), retinal hemorrhage,[32] and/or an alleged history overtly inadequate to the clinical picture. At least 3 criteria were needed for the diagnosis of NAHI in every case without a confession.

Patients with SDB were analyzed with respect to the cause of death and underlying diseases. Each SDB case was assigned to 1 of the following categories of possible causes[21]: traumatic, primary, or secondary disorders of hemostasis (hematologic disorders, sepsis, multiorgan failure, or major abdominal or thoracic surgery), perinatal (occurring in close temporal proximity to labor and/or in the context of intracranial bleeding of immaturity), infections, metabolic or biochemical disorders (eg, glutaric aciduria, galactosemia, or hypernatremia), or unexplained (any remaining SDB case not readily explained by 1 of the preceding mechanisms). SDB that occurred secondary to intracerebral hemorrhage or as a consequence of a neurosurgical procedure was not included. The age (fresh or old) and morphologic features (unilateral or bilateral and focal or diffuse) of the SDB were determined. SDB was considered old when it was described macroscopically as brown or yellow or with neomembranes present or when granulation tissue and/or hemosiderin was noted histologically. Fresh SDB was diagnosed when the SDB was described macroscopically as dark red and it could be easily washed from the dura. Pure intra-

dural bleeding was diagnosed when the dura was described macroscopically as reddish, with no blood clot visible, or significant amounts of erythrocytes were visible microscopically.

For all patients with SDB or NAHI, histologic slides, if available, were reviewed independently by 2 neuropathologists (Drs Matschke and Glatzel). Processing of samples was performed according to published protocols and included multiple sections from the cerebrum, dura mater, and eyeballs stained with hematoxylin and eosin, van Gieson's stain, Masson's trichrome stain, Kluver's stain, Nissl's stain, Turnbull's stain for siderin, and selected immunohistochemical stains.[33]

The brain weight was determined with a sensitivity of 1.0 g, after severance of the spinal cord at the level of the cervicomedullary junction and removal of the dura mater. Weighing was performed either immediately after removal of the brain (fresh brain weight) or after fixation in buffered formalin for an average time of 3 weeks (formalin-fixed brain weight). Because formalin fixation leads to a weight gain of 10% in pediatric brains,[34] this value was subtracted from the raw data to yield adjusted fresh brain weights. For standard values of infants' brain weights, metadata compiled from several literature sources were used.[35] For statistical analyses, only fresh or adjusted fresh brain weights were used. Brain weights for patients with significant alterations of brain weight attributable to preexistent pathologic conditions (malformations, massive bleeding, significant old hypoxic-ischemic lesions, or necrosis) and for brains that had been significantly destroyed artificially or through autolysis or putrefaction were omitted from statistical analysis.

Discrete groups were compared with the χ^2 test or Fisher's exact test, if adequate, and means of continuous variables were compared with t tests for independent samples. Correlation coefficients for correlations between brain weight and age were calculated for 3 groups, that is, patients with NAHI (group 1), patients with SDB resulting from causes other than NAHI (group 2), and all other patients (ie, with neither NAHI nor SDB) (group 3). A linear regression analysis of brain weight was performed by using age as the independent variable and including gender, NAHI, and SDB without NAHI, to ascertain potential relationships to brain weight apart from age.

RESULTS

Study Population

During the study period, a total of 16 661 autopsies were performed at our institution. After exclusion of stillbirths, infants <1 year of age constituted 715 of the total (4.3%). Of those, 392 were male and 307 female; for the remaining 16 patients, information allowing the determination of gender was lacking. The mean age at death was 75.4 days (median: 28 days).

Cause of Death

The most common cause of death was a malformation (289 patients [40.4%]) (Fig 1). Death occurred as a result of perinatal complications for 174 patients (24.3%), 87 patients (12.2%) had fatal infections, 32 (4.5%) died as a result of metabolic diseases, and 24 (3.4%) were assigned to the SIDS/unclassified sudden death group. Seventeen patients (2.4%) had fatal NAHI and 15 (2.1%) had other nonnatural causes of death. Sixty-seven patients (9.4%) had definite causes of death too rare to constitute a category of their own (other causes). For 10 patients (1.4%), a cause of death could not be determined with certainty (unclear cause).

Cause of SDB

SDB was present in 50 patients (7.0% of all autopsies). Forty-six of these cases (92.0%) could be assigned to 1 of the categories of known causes. Fifteen SDB cases (30.0%) were attributable to trauma (1 patient with accidental injury and the remaining 14 with NAHI). The 2 next most common causes were disorders of hemostasis and SDB occurring in the perinatal setting, with 13 patients each (28.0%). Four patients (8.0%) had SDB as a complication of a local infection, and SDB as a symptom of an underlying metabolic disease was extremely rare (1 patient). Four patients (8.0%) had unexplained SDB, including 1 unclassified sudden death case involving a 3-month-old boy with a small area of intradural hemorrhage and a SIDS case involving a 2-month-old girl with focal old SDB. The remaining 2 patients both had bilateral diffuse SDB. For a 7-month-old boy, "subdural bleeding bilaterally, most probably due to vascular malformation," was reported. Histologic testing of the alleged lesion had not been performed. The clinical history revealed that 2 other children in the family had died as infants, as a result of unknown causes. The mother was described as suffering from "confusional states," and the father was unknown. Because infantile SDB attributable to vascular malformation (especially bilaterally) is extremely rare and because the history in this case was strongly suggestive of child abuse, the possibility of NAHI was considered. Because of the poor documentation, the patient was assigned to the unclear death and unexplained SDB groups. The second patient with unexplained bilateral diffuse SDB was a 7-month-old boy who experienced "sudden apnea and loss of consciousness [and] trepanation due to subdural bleeding bilaterally." Additional clinical or pathologic data were lack-

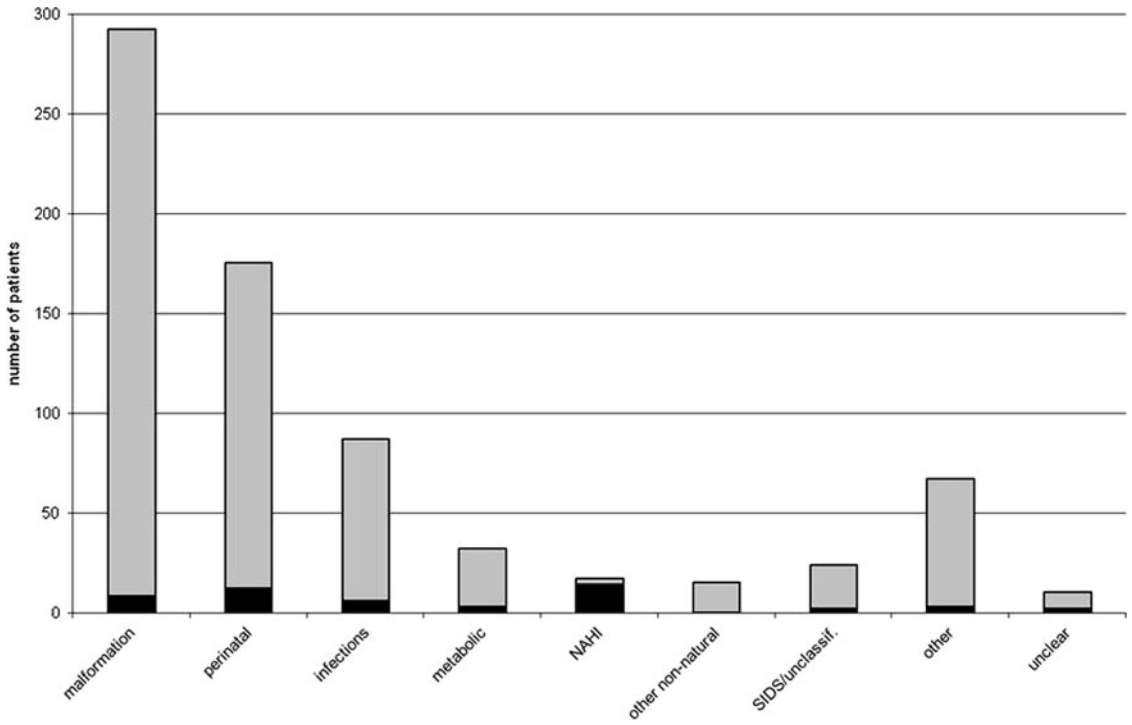

FIGURE 1
Cause of death in 715 infants' autopsies. The proportion of patients with SDB is shaded black in each category bar.

ing, and histologic testing had not been performed. Again, the possibility of NAHI was considered and the patient was assigned to the unclear death and unexplained SDB categories.

Morphologic Features of SDB

Twenty-one patients (42.0%) had focal SDB. The SDB was bilateral diffuse for 15 patients (30.0%) and was unilateral diffuse for 6 (12.0%). Eight patients (16.0%) had strictly intradural SDB. Thirty-three SDB cases were considered fresh, 12 old, and 5 both. Among the 15 patients with bilateral diffuse SDB, 10 cases were attributable to NAHI (66.7%). Accordingly, the most common morphologic type for traumatic SDB was bilateral diffuse (Fig 2).

Morphologic Findings and Characteristics of Patients With NAHI

The 17 patients identified as having NAHI were classified on the basis of a court conviction without confession of the defendant (1 patient), confession by the perpetrator (7 patients), and agreement at the case conference (9 patients). Nine patients were female and 8 were male. Patients with NAHI ($n = 17$; mean age: 140.4 days) were, on average, approximately twice as old as patients with SDB resulting from other causes ($n = 36$; mean age: 75.9 days; $P = .02$) and patients with neither NAHI nor SDB ($n = 662$; mean age: 73.8 days; $P = .004$) (Fig 3). Fourteen patients with NAHI (82.4%) had SDB. Of those, 12 had fresh diffuse SDB either bilaterally ($n = 10$) or unilaterally ($n = 2$), 1 had fresh intradural hemorrhage, and 1 had old SDB detected microscopically (Fig 4). For 3 patients (17.6%), SDB was absent. Histologic slides from brain tissue were available for all 17 patients with NAHI; for 5 patients, the slides also included sections from the dura mater. Histologic analysis of the slides revealed morphologic signs of recent hypoxic-ischemic encephalopathy for 5 patients, stigmata of brain death for 2 patients, and intraparenchymal hemorrhage consistent with diffuse axonal injury[36] for 2 patients. The clinical and morphologic findings, as well as the criteria for the identification of NAHI for each of the 17 patients, are summarized in Table 1.

Brain Weight

Brain weight data were available for 447 patients. In agreement with the literature,[35] there was no statistically significant difference in the brain weights of male versus female patients (mean: 397.8 vs 420.4 g; $P = .31$). Accordingly, data for male and female patients were not separated for statistical analysis. Of the 447 patients, 9 had NAHI, 20 had SDB occurring outside the setting of NAHI, and the remaining 418 had neither SDB nor NAHI. Pearson's correlation coefficients for correlations between age and brain weight were high and similar for all 3 groups (group 1: $r = 0.76$; group 2: $r = 0.70$; group 3: $r = 0.78$). Accordingly, linear regression

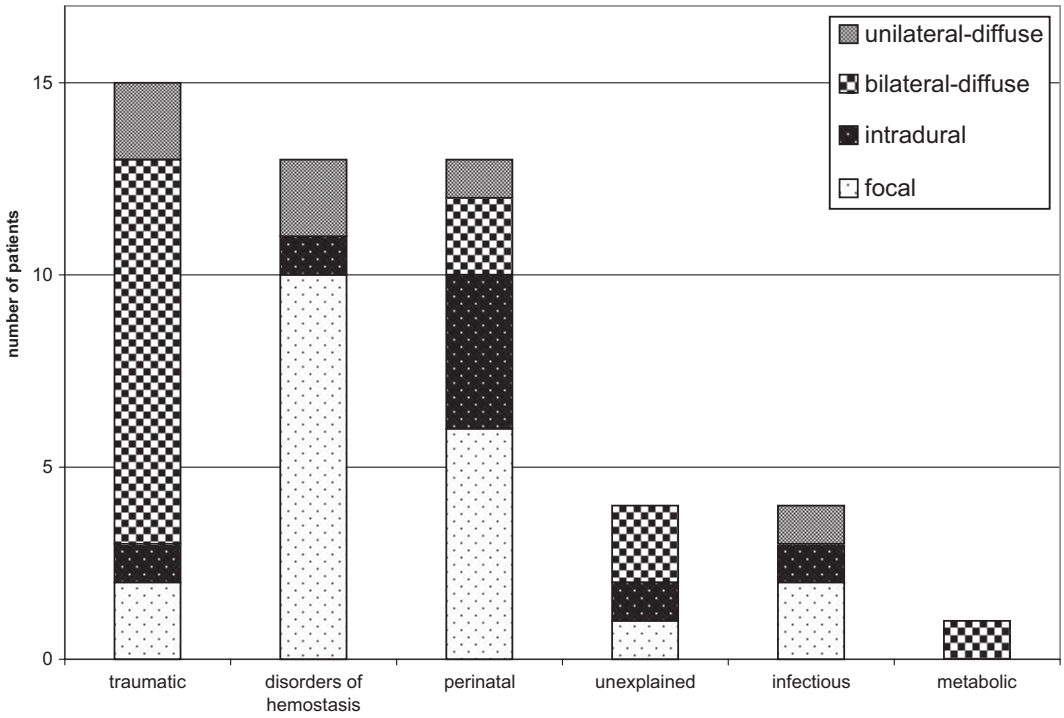

FIGURE 2
Cause and morphologic features of SDB in 50 infants' autopsies.

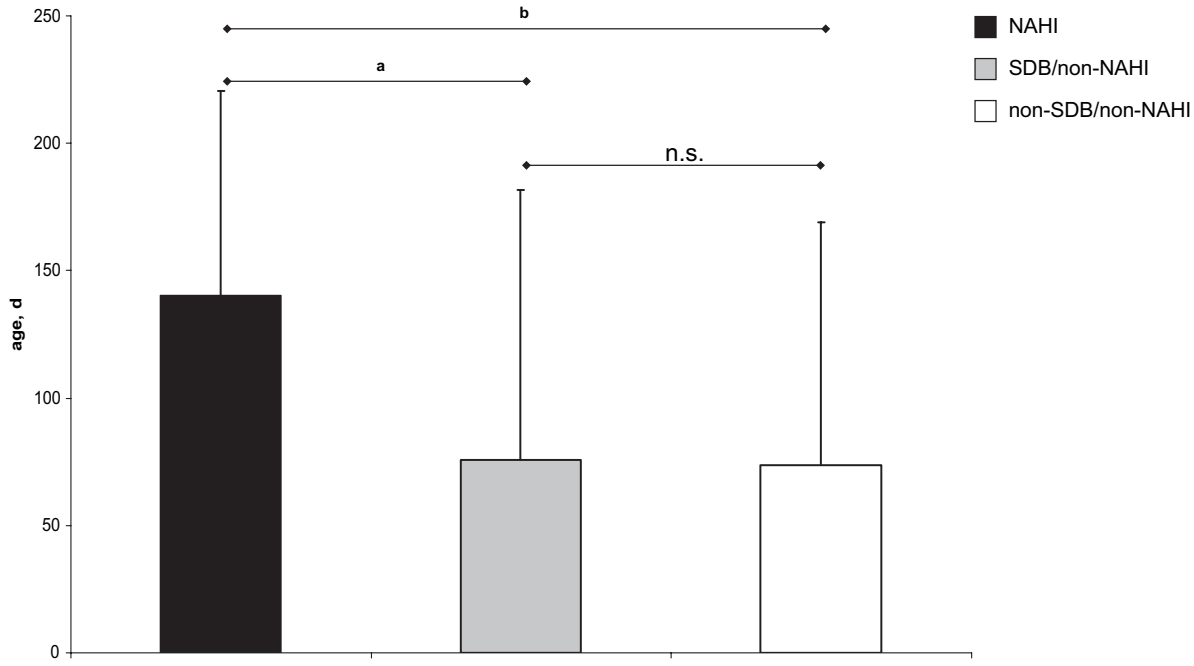

FIGURE 3
Ages of patients with NAHI ($n = 17$), patients with SDB occurring outside the setting of NAHI (SDB/non-NAHI; $n = 36$), and patients without SDB and NAHI (non-SDB/non-NAHI; $n = 662$). Whiskers indicate SDs; ns indicates not significant. [a] $P = .02$; [b] $P = .004$.

analysis showed no significant difference in the association between brain weight and age for the 3 groups. Because an increase in brain weight accompanies brain swelling and we did not detect any correlation between brain weight and SDB, brain swelling cannot be considered an independent risk factor for SDB.

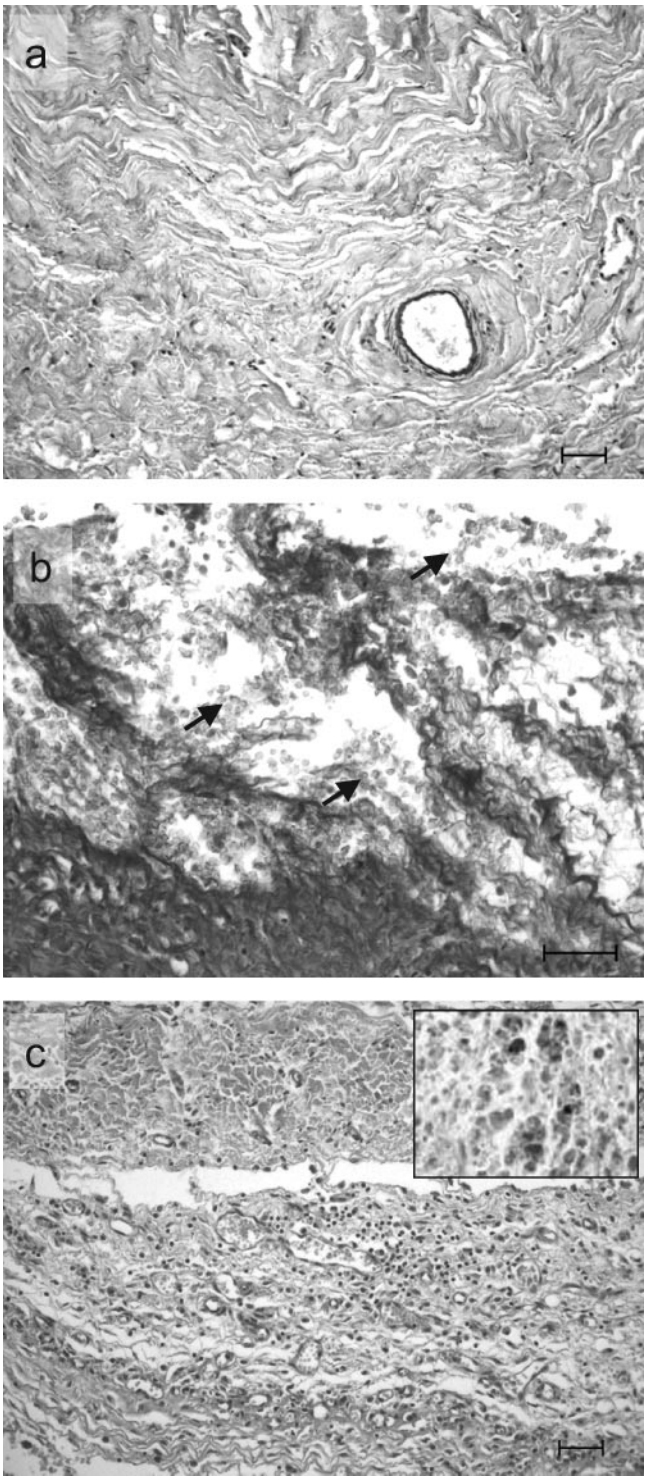

FIGURE 4

Histologic features of the dura mater. A, Normal dura mater consisting of bundles of collagen fibers and scattered vessels. Elastica van Gieson's stain; scale bar indicates 50 μm. B, Fresh intradural SDB with intact erythrocytes (arrows) between collagen fibers in a confirmed case of abusive head trauma (5-month-old boy). Elastica van Gieson's stain; scale bar indicates 50 μm. C, Old SDB with chronic granulation tissue (lower half) underneath the dura (upper half), with numerous fragile capillaries, sparse chronic round cell infiltration, and numerous siderophages (inset), in a confirmed case of abusive head trauma (8-month-old girl). Hematoxylin and eosin and Turnbull's stain for siderin (inset); scale bar indicates 50 μm.

DISCUSSION

In 2003, Geddes et al[24] published an article that initiated an intense debate on the concept of NAHI and abusive head trauma in infants. After identifying microscopic hemorrhage within the dura in 36 of 50 cases of intrauterine, neonatal, or infant death, Geddes et al[24] created a unified hypothesis, suggesting that SDB in infants was the result not of traumatic shearing of subdural veins but of a combination of severe hypoxia, brain swelling, and increased central venous pressure causing blood to leak from intracranial veins into the subdural space. Geddes and Plunkett[23] further concluded that "we need to reconsider the diagnostic criteria, if not the existence, of shaken baby syndrome." Because of the enormous medicolegal implications, especially concerning the necessity of impact and/or violence necessary to produce SDB in infants (or in claiming that SDB might result from any event that initiated apnea or hypoxia in infants), the hypothesis has become the center of an ongoing debate, "excoriated by most, but embraced by few."[1,23,25–30]

If hypoxia and/or brain swelling has a possible role in the pathogenesis of infantile SDB, according to the unified hypothesis, then the incidence of SDB in infants' autopsies should be very high. Most infantile deaths occurring in a clinical setting are attributable to congenital heart or lung disease, birth asphyxia, septicemia, or bronchopneumonia,[24] conditions that are all capable of leading to hypoxic-ischemic injury with consequent brain swelling. Our study population reflected the spectrum of infant deaths occurring in a clinical setting, with most patients being profoundly hypoxic before death. In our cohort of 715 infants' autopsies, however, we were able to identify only 50 patients (7.0%) with SDB, which made SDB in infants' autopsies an uncommon event. In addition, the statistical associa-

TABLE 1 Clinical and Morphologic Details for 17 Patients With NAHI

Patient No.	Age, mo	Gender	Initial Clinical History	External Injury	Fractures	Hemorrhage				Intracerebral Pathologic Findings	Identification of NAHI
						Epidural	Subdural	Subarachnoid	Retinal		
1	4	Female	Suddenly stopped breathing while playing	None	Long bones, ribs	No	Bilateral diffuse	Microscopic	Yes	Microscopic bleeding	Case conference
2	8	Female	Accidentally bumped head against car door	None	Skull	Yes	Bilateral diffuse	No	Yes	None	Confession of perpetrator
3	4	Female	Accidentally slipped out of baby buggy	Hematomas	Skull	No	Bilateral diffuse	No	No	None	Court conviction
4	5	Male	Suddenly went limp and turned blue	Hematomas	None	No	Intradural	Yes	Yes	Contusions	Confession of perpetrator
5	1.5	Male	Found lifeless	Hematomas	Long bones, ribs	No	None	No	NE	Contusions	Confession of perpetrator
6	3	Male	Found lifeless	Hematomas	Skull	No	Microscopic, old	Microscopic, old	NE	None	Case conference
7	10	Female	Accidentally bumped head against edge of bed	Hematomas	None	No	Bilateral diffuse	Yes	Yes	None	Case conference
8	4	Female	Fell out of baby buggy	Hematomas	Long bones, ribs	No	Unilateral diffuse	Yes	NE	Contusions	Case conference
9	3	Female	Suddenly stopped breathing	Hematomas	None	No	Unilateral diffuse	Yes	No	Contusions	Case conference
10	5	Male	Fell from couch	Hematomas	NE	Yes	Bilateral diffuse	Yes	Yes	None	Case conference
11	4	Male	Slipped from father's arms	Hematomas	None	No	Bilateral diffuse	Yes	NE	None	Case conference
12	6	Male	Suddenly went limp and blue	Hematomas	Skull	No	Bilateral diffuse	Microscopic, old	NE	None	Case conference
13	9	Female	Suddenly stopped breathing	Hematomas	NE	No	Bilateral diffuse	No	NE	Microscopic bleeding	Case conference
14	6	Male	NA	NE	Skull	No	None	No	NE	Contusions, lacerations	Confession of perpetrator
15	2	Female	Perpetrator tried to stop crying by beating and shaking	Hematomas	NE	Yes	Bilateral diffuse	Yes	NE	Hemorrhagic necroses	Confession of perpetrator
16	4	Male	Bumped head accidentally	NE	NE	No	Bilateral diffuse	No	NE	None	Confession of perpetrator
17	1	Female	Bumped head against bed	None	NE	No	Bilateral diffuse	No	NE	None	Confession of perpetrator

NE indicates not examined; NA, not available.

tion between brain weight and age was the same for patients with SDB occurring outside the setting of NAHI (SDB/non-NAHI) and for patients with neither SDB nor NAHI (non-SDB/non-NAHI), which argues against unique brain swelling in the SDB group. Therefore, our data give no evidence for the unified hypothesis, corroborating a recent morphologic study of 82 fetuses, infants, and toddlers with apnea/hypoxia, in which no case with macroscopic evidence of SDB could be found.[37]

Furthermore, the majority of SDB cases in our series could be assigned to categories of known causes, leaving only a small proportion of unexplained SDB cases. Overall, the most common cause of SDB in our series was head injury (30% of all cases), with NAHI accounting for >90% of all traumatic SDB cases. These data become even more convincing with separate examination of the 15 cases with bilateral diffuse SDB, the morphologic type considered typical for NAHI,[2] because two thirds of those cases were attributable to NAHI (Fig 2). In our series, NAHI was the most common single cause for SDB in infants, especially when bilateral diffuse and at older ages, followed by SDB in clotting disorders and SDB in the perinatal setting (Fig 2). These results are in agreement with the few clinical or epidemiological studies available to date.[4,18,19,38]

At present, there is no proven or accepted pathophysiologic explanation for a possible role of hypoxia in the pathogenesis of SDB.[25] Therefore, hypoxia-induced SDB would have been classified as unexplained in our series, although only 4 cases were classified as such. Two of those cases involved only focal intradural bleeding, and each had a definite cause of death (SIDS and unclassified sudden death[31]). In both cases, the significance of the focal SDB with respect to the cause or circumstances of death was negligible. The remaining 2 cases of unexplained SDB dated from 1960 and 1972, and there was no definable cause of death. Both cases involved unexplained, fresh, diffuse, bilateral SDB, and both deaths might have been attributable to NAHI. Because neither histologic findings nor adequate documentation was available, these 2 cases were classified as unclear. Therefore, unexplained SDB is exceedingly rare in infants, and the existence of hypothetical, hypoxia-induced SDB, as suggested by the unified hypothesis, may be seriously questioned.

One drawback of our study may be the relative dearth of histologic studies of dura mater, because dura mater is studied histologically only if abnormalities are identified macroscopically. Consequently, the occurrence of SDB could be ascertained only macroscopically for the majority of our patients. It is possible that an unknown number of cases with microscopic intradural bleeding might have gone undetected. This might explain the considerably higher frequency of SDB in the study by Geddes et al,[24] because those authors specifically analyzed the dura histologi-

cally for all of their 50 patients. However, their study population included abortions, intrauterine deaths, and stillbirths, with only 30 live-born patients. In only 13 of those cases did the authors consider the dural hemorrhage severe. Of those 13 cases, 12 were either perinatal or neonatal deaths. Because NAHI is an extreme rarity in very young infants and typically occurs at a mean age of 4.6 months,[7] only 1 patient with SDB in the study by Geddes et al[24] might be of an age to be considered as having NAHI.

CONCLUSIONS

Our analysis of a large number of infants' autopsies showed that (1) SDB in infants is a rare event, (2) most SDB in infants is attributable to trauma, with NAHI substantially outnumbering accidental injuries, and (3) most SDB in infants can be explained. Our data indicate that NAHI and not hypoxia or brain swelling is the most common cause of SDB in infants.

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