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Cerebral Complications of Nonaccidental Head Injury in Childhood

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Patterns of cerebral parenchymal injury and their relationship to outcome morbidity are evaluated in this retrospective study of 14 children with confirmed nonaccidental head injury (NAHI). The mean age at time of injury was 12 months 6 days, mean Children's Coma Score was 5.36, and mean postinjury follow-up was 17 months 12 days. All patients had acute subdural hematoma (interhemispheric or convexity) on initial CT imaging. Two major groups of children were identified from initial CT scans; those with diffuse cerebral hypoattenuation (n = 7) and those with focal cerebral hypoattenuation (n = 7). The two groups differed significantly by age (diffuse group, mean age 5 months 9 days ± 36 days; focal group, mean age 19 months 3 days ± 6 months 9 days; $P < 0.01$) and ultimate type and extent of parenchymal damage. Outcome was generally poor in both groups (mean Children's Outcome Score of III/IV). Cerebral infarction developed in all survivors. Most common were hemispheric necrosis after hemispheric swelling subjacent to an ipsilateral convexity acute subdural hematoma (n = 5); distribution of the posterior cerebral artery (n = 4) or callosomarginal branch of the anterior cerebral artery (n = 4); and borderzone infarctions (n = 4). Of 14 children, 11 (79%) had early posttraumatic seizures (EPTS). Clinical progression of symptoms was confirmed in nine patients (mean Childrens Coma Score was 4.0 ± 0.33). None had a lucid interval. This is the first study using strict inclusion criteria that documents the range of infarction patterns and potential age-dependent differences in postinjury response cascades after nonaccidental head injury.

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Introduction

Nonaccidental head injury (NAHI) is a significant cause of mortality and morbidity in the infant and young child and is characterized by a variety of injury patterns, including diffuse axonal injury, other shearing injuries of the brain, subdural hematoma, evidence of blunt force trauma, and ocular findings [1-3]. Few analyses of the types and extent of cerebral parenchymal injuries in NAHI have been completed [4-7]. In particular, little emphasis has been placed on vascular territory cerebral parenchymal damage associated with NAHI [8,9].

The inclusive term *nonaccidental head injury* is used herein for head injury caused by acts of commission by a caregiver that require significant force and result in craniospinal injury. NAHI encompasses rotational and translational biomechanical forces, as well as other contributing factors, such as smothering, strangulation, or cardiorespiratory arrest, occurring at the time of injury. The terms *shaken baby syndrome* and *shaken-impact syndrome* imply a simplicity and uniformity of mechanism that focus on biomechanical forces and ignore the important contribution of hypoxic-ischemic injury to postinjury response cascades.

The purpose of this study is to evaluate whether infarction after NAHI is primarily unrelated to strangulation injury and whether young children with severe NAHI exhibit a lucid interval after injury events. Historical data, clinical features, and neuroradiologic findings in a series of 14 infants and children with confirmed NAHI presenting with diffuse or focal cerebral hypoattenuation on computed tomography (CT) imaging are reported.

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Patients and Methods

Patients. Inclusion criteria included the following: (1) confirmation of nonaccidental etiology, by witnessed events, confession, felony conviction, or the absence of trauma history in the presence of clear and convincing evidence of major head trauma (e.g., blunt force trauma or evidence of associated strangulation); (2) evidence of either focal or diffuse cerebral hypoattenuation on initial CT imaging; (3) complete medical records, including documentation of ophthalmologic evaluation and radiographic skeletal surveys; (4) imaging studies from admission and after hospital discharge; and (5) neurologic follow-up data available for a minimum of 3 months after hospital discharge.

Children were excluded if the diagnosis of NAHI could not be conclusively established, records or imaging studies were insufficient, they lacked cerebral hypoattenuation on initial imaging, or they had other acute or chronic illnesses diagnosed. Children with meningitis, sepsis, and coagulopathy were also excluded. This process yielded 14 children selected from 52 children admitted to Childrens Hospital Los Angeles from 1991 to 1994 with suspected NAHI.

Study Groups. Group assignment was based on initial CT scan criteria only. Patients were included in the diffuse hypoattenuation group (Group 1) if they displayed: (1) diffuse, bilateral parenchymal hypoattenuation with loss of gray-white matter differentiation; (2) small or normal ventricles; (3) no midline shift greater than 3 mm; and (4) no intraparenchymal hemorrhages. Seven of the 14 children fulfilled these criteria. The remaining seven patients with (1) one or more regions of focal parenchymal hypoattenuation with loss of gray-white matter differentiation in the affected region; (2) focal sulcal effacement; and (3) midline shift of 2 mm or greater were included in the focal hypodensity group (Group 2).

Data Collection. Clinical records, radiologic images, and medical examiner records were collected retrospectively. Children's Coma Scores (CCS) and Children's Outcome Scores (COS) were determined from available clinical data [10,11]. Developmental delay was defined as mild, moderate, or severe by Denver II Developmental testing. Initial noncontrast CT scans were obtained in all patients using a GE 9800 scanner (General Electric, Milwaukee, WI) with contiguous 5-mm thick axial sections. In four patients, magnetic resonance imaging (MRI) scans (1.5 T Siemens 635 P system; Siemens, Iselin, NJ) were also obtained during the follow-up period. Timing of the first CT scan varied from less than 4 hours after onset of symptoms in six patients who presented with rapidly deteriorating clinical status, to 73 hours after onset of symptoms (mean = 16.7 hours).

Infarction patterns were defined according to the following criteria. Diffuse cerebral infarction was diagnosed if bilateral borderzone infarction and diffuse cerebral atrophy was present on follow-up CT or MRI neuroimaging. Patients were classified as having arterial infarctions if abnormalities were limited to one or two major arterial territories or hemispheric necrosis if the entire hemisphere was involved on follow-up CT or MRI neuroimaging.

Statistical Analysis. Groups were compared using the nonparametric Mann-Whitney U test. All data are presented as mean ± SEM except where indicated. Significance was defined as $P < 0.05$.

Results

These 14 patients represent 26.9% of children admitted with suspected NAHI during the study period. Historical data, clinical features, and radiographic skeletal findings are summarized in Table 1; neuroradiologic findings are summarized in Table 2; and outcome data are summarized in Table 3.

Patient Demographics

The mean age of the 14 selected children at the time of injury was 12 months 6 days (range 53 days to 4 years 6 months), with 10 of 14 less than 10 months of age (mean 6 months 9 days ± 3 months 3 days). Eight children were boys (57%) and six were girls (43%). Three children died. The perpetrator was identified in 12 of 14 cases. The father was the perpetrator in five cases, the mother's boyfriend in two cases, a child care provider in four cases, and an uncle in one case.

Presenting Findings

Unconsciousness, apnea, respiratory difficulty, and seizures were the presenting findings in 13 (93%) of 14 children. Mean CCS was 5.36 (range 3-11). Of 14 children, 10 (71%) had retinal hemorrhages. These were both preretinal and intraretinal.

Historical descriptions of injury events accurately described clinical behavior after the injury, such as apnea, limpness, or seizures, but understated the actual injury events (e.g., physical evidence of multiple impact sites with history of shaking only). The clinical progression of symptoms could be accurately determined in nine of 14 children. The mean CCS score of these patients was 4. Loss of consciousness developed during injury events and persisted in all of these patients. None exhibited a lucid interval after injury events.

Strangulation was a proven component of injury events in three children and strongly suspected in a fourth. Only one of these patients developed an infarct pattern that could be directly attributed to strangulation injury.

Early Posttraumatic Seizures

Eleven children (79%) had early posttraumatic seizures (EPTS) starting before or during the first 72 hours after admission. Eight children had partial seizures with secondary generalization and three had generalized tonic or tonic-clonic seizures. Children with generalized seizures had a significantly worse outcome compared with children with partial seizures with secondary generalization ($P < 0.05$). Seizures were difficult to control in eight children, consisting of partial seizures in six patients and generalized seizures in two patients.

Impact Evidence

Unequivocal clinical or radiologic evidence of impact (facial bruising, scalp contusions, subgaleal hematomas, or skull fractures) was observed in eight (57%) of 14 children. Two patients had minor facial trauma and were not included in the group of definitive impact. Skull fractures were present in two of 14 patients.

Table 1. Clinical data

Patient No./ Sex/Age	History			Physical Examination				
	Trauma	Clinical	EPTS	Neurologic Focality	CCS*	Bruises/STS/Other	RH	Skeletal Survey
1/M/6 mo 23 days	Alleged fall <3 ft w/o symptoms	12 hr irritability	LPSG	L hemiparesis; R gaze deviation	10	None	+	∅ fracture
2/F/3 mo 15 days	None	"Made noise; went limp": CRA at scene	RPSG	R > L hypotonia; L gaze deviation	8	LSQ hematoma	+	L recent tibial fracture with minimal periosteal reaction
3/M/4 mo 8 days	None	Infant not breathing after being in bathroom × 5 min w/father	LPSG	L hemiparesis; R gaze deviation	4	Chin, shoulder, and abdomen—healing	+	Healed distal L radius; Salter II fracture
4/M/9 mo 8 days	None	Sudden onset A, LOC, and P	None	Opisthotonic posturing; L pupil fixed and dilated	3	5 × 5 cm occipital	+	>6 cm diastatic occipital skull fracture to foramen magnum
5/M/54 mo	Witnessed strangulation, shaking, thrown onto edge of marble table	Sudden onset A, LOC, flaccid	LPSG	Extensor posturing at scene; L hemiplegia in emergency room	3	Neck L > R; facial petechiae	+	>4 cm extensive occipital skull fracture
6/F/1 mo 23 days	None	Father "suddenly realized she wasn't breathing"	LPSG	None	8	Chin and bilateral UE—petechiae	+	∅ fracture
7/F/27 mo	Alleged <2 ft fall 6 days earlier w/o symptoms; confession "shaking and driving head into wall"	Sudden onset A, LOC, and P	GTC	None	4	Bilateral periorbital	+	∅ fracture
8/F/14 mo 15 days	None	She suddenly became "limp, pale, and made odd sounds"	None	R hemiparesis; extensor posturing, L gaze deviation	6	Facial (handprint) bilateral thighs and buttocks	−	∅ fracture
9/M/12 mo 23 days	None	Sudden onset V, LOC, and limpness	RPSG	R sided extensor posturing	4	None	−	∅ fracture
10/F/5 mo 23 days	Confession: shaking	A and LOC	GTC	Extensor posturing	4	Forehead (faint)	+	∅ fracture
11/M/9 mo 16 days	Alleged fall <3 ft 1 day earlier w/o symptoms	Onset crying with uncle; irritable and weak cry × 12 hr; then seizures	LPSG	L hemiparesis	9	R cheek; R neck near supraclavicular fossa; both pinnae	+	∅ fracture
12/M/3 mo	None	A and limpness	LPSG	None	5	L cheek (small); abrasion forehead (small)	−	Subacute rib fracture R 6 and 7
13/F/9 mo 15 days	Alleged fall against edge of crib 12 hr earlier w/o symptoms	Sudden onset LOC, pallor, and limpness	GTC	Extensor posturing; R eye medially deviated	4	Old and new frenulum tears	+	∅ fracture
14/M/9 mo 13 days	None	A, LOC, pallor; CRA at scene	None	None	3	Acute frenulum tear; T 90.8° R	−	Healing femur fracture

* Total maximum, 11; total minimum, 3; ocular response maximum, 4; verbal response maximum, 3; motor response maximum, 4; 8-10 on CCS is equivalent to 5-8 on Glascow Coma Score (GCS); 3-7 on CCS is equivalent to 3-4 on GCS.
+ = present.
− = absent.

Abbreviations:
A = Apnea
CCS = Children's Coma Score
CRA = Cardiorespiratory arrest
EPTS = Early posttraumatic seizures
GTC = Generalized tonic clonic seizures
LOC = Loss of consciousness
P = Posturing
PSG = Partial seizures with secondary generalization
RH = Retinal hemorrhage
STS = Soft tissue swelling
UE = Upper extremities
V = Vomiting

Table 2. Imaging data

Patient No.	Initial Imaging Pattern: Hypodensity	Sulcal Effacement	Loss GWM Differentiation	MLS	ASDH	End Results: PCA Infarct	Hemispheric Necrosis	Borderzone Infarct	Other Infarct
Group 1	Diffuse cerebral hypoattenuation								
2	Diffuse	+	+	−	C-bilateral (scanty); IH (−)	L	−	−	L branch MCA
3	Diffuse	−	+	−	C (−); IH (3 mm)	Bilateral	−	+	DCA
6	Diffuse	+	+	−	C-bilateral (scanty); IH (2 mm)	R	−	+	R > L volume loss
10	Diffuse	+	+	−	C (−); IH (2 mm)	−	−	+	DCA, severe
12	Diffuse	+	+	−	C-bilateral (scanty R > L); IH (2 mm)	−	−	+	DCA, R > L, severe
13	Diffuse	+	+	−	C (−); IH (1-2 mm)	−	−	−	Diffuse brain necrosis (autopsy)
14	Diffuse	+	+	−	C (−); IH (2-3 mm)	−	−	−	Diffuse brain necrosis (autopsy)
Group 2	Focal cerebral hypoattenuation								
1	R frontal	+ R frontal	+ (R)	2 mm	C-R (2 mm); IH (2 mm)	R	−	−	−
4	L hemispheric	+ L hemispheric	+ (L)	12 mm	C-L (8 mm); IH (4 mm)	−	L	−	R frontal + R superior cerebellar necrosis (autopsy)
5	R hemispheric	+ R hemispheric	+ (R)	14 mm	C-R (10 mm); IH (3 mm)	−	R	−	L branch ACA
7	L hemispheric R superior cerebellar	+ L hemispheric	+ (L)	20 mm	C-L (16 mm); IH (3 mm)	−	L	−	R branch ACA
8	L hemispheric	+ L hemispheric	+ (L)	4 mm	C-L (4 mm); IH (2 mm)	−	L	−	−
9	L hemispheric	+ L hemisphere	+ (L)	−	C-L (4 mm); IH (2 mm)	−	L	−	R branch ACA
11	R frontal	+ R frontal	+ (R)	−	C-bilateral (scanty); IH (2 mm)	−	−	−	R ICA

+ = present.
− = absent.

Abbreviations:
ACA = Anterior cerebral artery
C = Convexity
DCA = Diffuse cortical atrophy
ICA = Internal carotid artery
IH = Interhemispheric
MCA = Middle cerebral artery
MLS = Midline shift
PCA = Posterior cerebral artery

Postmortem Examination Data

Postmortem examination results extended the clinical and imaging findings in all cases. All three children who died had significant additional evidence of impact found at postmortem examination. In two of these cases the extent and significance of impact were not suspected by clinical examination alone (both children only had frenulum tears by external examination). Extensive acute optic nerve sheath subdural hematoma was present in all children who died. Evidence of strangulation in one patient was only found at the postmortem examination.

Diffuse vs Focal Cerebral Hypoattenuation

The mean age of children in Group 1 (diffuse cerebral hypoattenuation) was 5.3 months (median 4 months 8 days; n = 7), which was significantly younger than the mean age of 19 months 3 days in Group 2 (focal cerebral hypoattenuation) (median 12 months 23 days; n = 7) ($P < 0.01$) (Table 4). Not unexpectedly, they differed by admission neurologic examination. Children with diffuse brain swelling presented in coma with or without extensor posturing, with one exception. Children admitted with focal brain swelling uniformly had focal cranial nerve or motor findings on initial neurologic examination, in addition to coma (n = 5) or irritability (n = 2).

The two groups did not differ significantly by admission CCS, COS, or frequency of EPTS. All but one patient in Group 2 (focal brain swelling) had partial seizures with secondary generalization; two patients in Group 1 (diffuse brain swelling) also had partial seizures with secondary generalization. In general, the presence or absence of focality on neurologic examination predicted the type of

Table 3. Outcome

Patient No.	Time Monitored (mo)	Developmental Delay	Microcephaly	Motor Deficit	Visual Impairment	PTE	Children's Outcome Score*
1	15	Mild	+	−	HH	−	II
2	10	Moderate	+	L hemiparesis	HH	−	III
3	23	Moderate	+	−	CB	−	II
4	NA	Deceased					V
5	34	Severe	−	L hemiparesis	HH	−	III
6	13	Severe	+	Spastic quadriparesis, L > R	CB	+	IV
7	28	Severe	+	Spastic quadriparesis	CB	−	III
8	10	Moderate	−	R hemiparesis	HH	−	III
9	28	Severe/almost vegetative	+	Spastic quadriparesis	CB	−	III/IV
10	14	Severe/vegetative	+	Spastic quadriparesis	CB	+	IV
11	4	Mild	−	L hemiparesis		−	III
12	12	Severe	+	Spastic quadriparesis	CB	+	IV
13	NA	Deceased					V
14	NA	Deceased					V

* I = Excellent recovery; II = moderate but nondisabling defect; III = either a severe motor or cognitive defect; IV = vegetative; V = death; + = present; − = absent.

Abbreviations:
CB = Cortical blindness
HH = Homonymous hemianopia
NA = Not applicable
PTE = Posttraumatic epilepsy

seizure. Three of five survivors presenting with diffuse brain swelling developed posttraumatic epilepsy (60%) compared with none of those with focal swelling.

Typical for children with severe head injury, both groups had hyperglycemia at admission. Although the admission blood glucose was higher in children with diffuse brain swelling (278.1 ± 20.7 mg/dL vs 179.8 ± 20.7 mg/dL), it was not statistically significant. Children with an initial blood glucose greater than 200 mg/dL tended to have a worse outcome. The presenting leukocyte count ranged from 6,900 to 54,800 with a median of 16,500. Children tended to be anemic on admission.

Table 4. Group comparison

	Group 1: Diffuse Cerebral Hypoattenuation (n = 7)	Group 2: Focal Cerebral Hypoattenuation (n = 7)
Age	5 mo 9 days ± 1 mo 6 days	19 mo 3 days ± 6 mo 9 days ($P < 0.01$)
CCS	5 mo 3 days ± 24 days	5.6 ± 0.3 (NS)
COS	IV (3.9 ± 0.4)	III (3.2 ± 0.3) (NS)
Initial glucose (mg/dL)	278.1 ± 72.8	179.8 ± 20.7 (NS)
Seizures		
EPTS	6	5
PTE	3	0
Microcephaly	5 (100%)	3 (50%)
FU imaging patterns	n = 5 survivors	n = 6 survivors
Hemispheric Necrosis	0	5
PCA	3	1
Branch ACA	0	4
Branch MCA	1	0
Branch ICA	0	1
Borderzone	4	0
DCA	4	0

Abbreviations:
ACA = Anterior cerebral artery
CCS = Children's Coma Score
COS = Children's Outcome Score
DCA = Diffuse cortical atrophy
EPTS = Early posttraumatic seizures
FU = Follow-up
ICA = Internal cortical artery
MCA = Middle cerebral artery
PCA = Posterior cerebral artery
PTE = Posttraumatic epilepsy

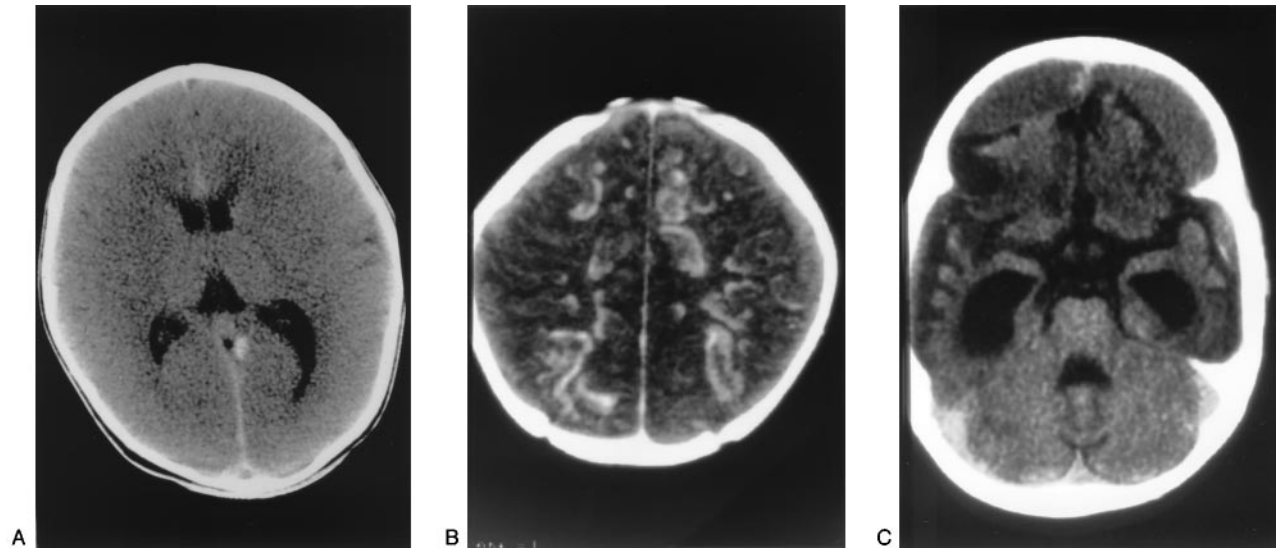

Figure 1. (A) Nonenhanced axial CT scan demonstrating mild diffuse cerebral hypoattenuation with loss of gray-white matter interface and sulcal effacement. There is a small amount of acute interhemispheric blood (Patient 10). (B) Nonenhanced axial CT scan 10 days after injury reveals bilateral infarction in the borderzone areas between anterior and middle cerebral arteries with extensive cortical laminar necrosis. (C) Nonenhanced axial CT scan 5 months after injury. There is diffuse cortical atrophy with significant volume loss, secondary ventriculomegaphy, and bilateral chronic subdural hematomata.

Although hematocrits ranged from 23.8% to 45%, the median hematocrit was 27%.

Neuroimaging

Follow-up scans were obtained in the 11 surviving patients 6 weeks to 34 months after injury. Patterns of cerebral parenchymal injury differed between groups and are summarized in Table 4. Four of the five survivors in Group 1 (initial diffuse parenchymal hypoattenuation) developed borderzone infarctions between the anterior cerebral and middle cerebral artery distributions associated with diffuse cortical atrophy (Fig 1). In this group, three children developed infarction either unilaterally or bilaterally in the posterior cerebral artery distribution compared with one child in Group 2.

By contrast, initial focal low-density patterns of children in Group 2 were either hemispheric (n = 5) or unilateral frontal (n = 2) (Fig 2A). The five children who had an initial CT finding of hemispheric low density all

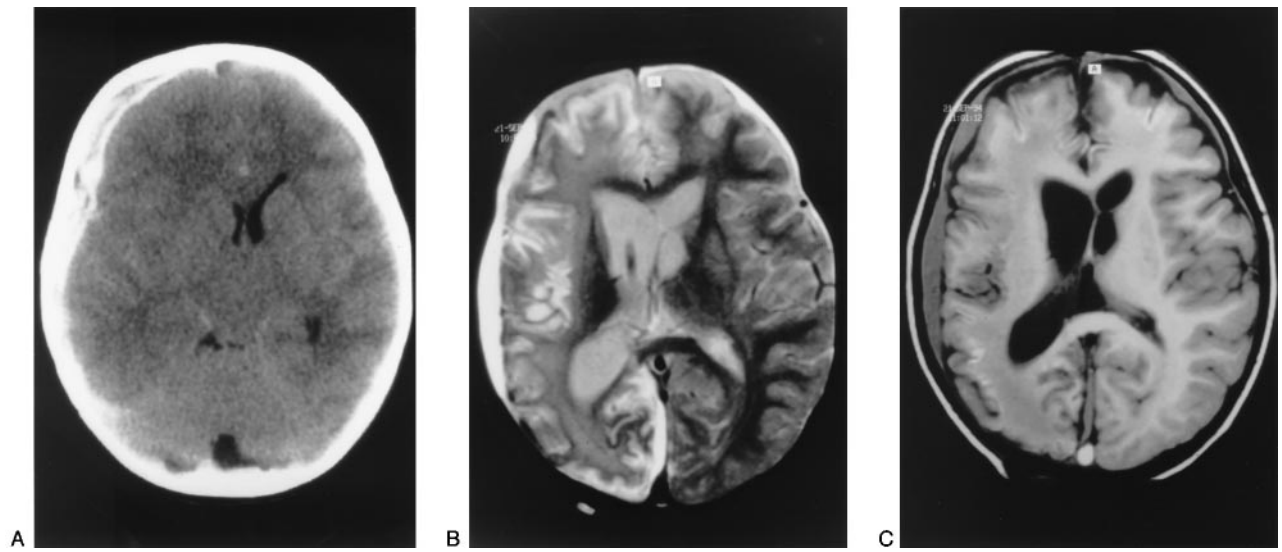

Figure 2. (A) Nonenhanced axial CT scan demonstrating a right acute subdural hematoma with ipsilateral hemispheric swelling (Patient 5). The right lateral ventricle is effaced and there is midline shift. Gray-white matter differentiation is decreased (right > left). Early acute hydrocephalus of the left temporal horn is also present. (B, C) Axial T_1-(1.5 T, TR = 500 ms; TE = 10 ms) and T_2-weighted (1.5 T, TR = 3,300 ms; TE = 90 ms) magnetic resonance images from the same patient 3 months after injury. The scans demonstrate right hemispheric and left frontal infarcts. There is significant posttraumatic encephalomalacia of the right hemisphere, with associated ventriculomegaly.

had an ipsilateral convexity acute subdural hematoma (ASDH) of varying size. Only one of these children underwent evacuation of the hematoma. These children uniformly developed hemispheric necrosis of the involved hemisphere (Fig 2B,C). Four also developed infarctions of the callosomarginal branch of the anterior cerebral artery (Fig 2B,C).

Less common infarction patterns involved the superior cerebellar (one in Group 2), internal carotid (one in Group 2), and the terminal branch of the middle cerebral arteries (one in Group 1). No child developed isolated infarction in the distribution of the middle cerebral artery, superior sagittal sinus thrombosis, or hemorrhagic infarction in an involved distribution.

Outcome Morbidity

The 11 survivors were monitored for 5-31 months after injury (mean length of follow-up was 17 months 12 days). Children with initial CT findings of diffuse parenchymal hypodensity had greater morbidity than those with focal parenchymal hypodensity. Mean COS was IV in Group 1 (diffuse) compared with III for Group 2 (focal) (Table 4). Developmental delay was present in all 11 survivors and tended to be more severe in those children with diffuse brain injury. Likewise, microcephaly, a finding in eight (73%) of 11 surviving children, developed in 100% of Group 1 (diffuse) and 50% of Group 2 (focal) patients.

Motor and visual deficits correlated with the initial neurologic examination and with the initial imaging pattern. Hemiparesis or quadriparesis developed in nine (82%) of 11 children. Significant visual impairment was present in all but one survivor. Visual impairment consisted of either a homonymous hemianopia or cortical blindness (retention of pupillary light reflexes in the absence of vision). These visual deficits were not related to retinal hemorrhages.

Discussion

Infarction may be more common after NAHI than previously recognized. Infarction occurred in 27% of children admitted for suspected NAHI during the study period. The actual frequency of cerebral infarction in children sustaining NAHI remains unknown. In older children and adults, vascular distribution infarction after head injury is rare, regardless of etiology. In 1,332 patients 8-91 years of age with unintentional head injury, cerebral infarction occurred in 25 (2%) [12].

We identified two groups of children on the basis of the initial CT findings; those with diffuse brain swelling and those with focal brain swelling. Younger infants were more likely to present with diffuse brain swelling with or without bilateral ASDH; older infants and children presented with focal brain swelling and ipsilateral ASDH. Initial imaging patterns in our series largely predicted the pattern of subsequent brain injury. Only patients with diffuse brain swelling developed cortical necrosis and borderzone infarctions. By contrast, five of six survivors in Group 2 (focal brain swelling) developed hemispheric necrosis. Eighty percent of children with hemispheric swelling had secondary infarction in the distribution of the callosomarginal branch of the anterior cerebral artery from subfalcine herniation. In contrast to a larger series of older children and adults, in whom middle cerebral artery infarction was equally likely to occur as posterior or anterior cerebral artery infarction, we found only one terminal branch middle cerebral artery infarct in our study group [12]. Arterial territory cerebral infarcts primarily developed from vascular compression secondary to mass effect and transtentorial with or without transfalcine herniation, not secondary to strangulation injury to the carotid.

The greater tendency for younger infants to develop diffuse brain swelling as a pathophysiologic response to injury has not been explained. Whether or not this apparent age-related difference between younger children and older individuals is a function primarily of age, type of injury (nonaccidental vs unintentional), or unique physiologic features of the immature brain and craniospinal axis is unknown and warrants further research [13,14]. These physiologic features include differences in biomechanical properties, postinjury cerebral autoregulation, and vasoreactivity and ischemic tolerance [13-15].

Cerebral ischemia was a prominent feature in the infants in this series, as evidenced by cortical laminar necrosis and infarctions in the borderzone between the anterior and middle cerebral artery [16,17]. Immediate postinjury apnea and hypotension combined with a delay in seeking medical care may also potentiate secondary injury cascades more in younger children with NAHI [1,14,18,19]. Although ischemia and hypoxia have been suggested to be the primary determinants of poor outcome after traumatic brain injury at any age, recent studies have found that children less than 24 months old are at higher risk of early hypoperfusion [1,14,18].

Hemispheric swelling with or without ipsilateral ASDH is not uncommon after closed head injury and is not specific for abuse [20]. Hemispheric swelling and secondary necrosis was the most common pattern observed in our series, associated in all cases with an ipsilateral acute convexity subdural hematoma of varying extent. The etiology of hemispheric swelling and secondary necrosis subjacent to an ASDH is not understood. Clinical and experimental observations suggest that swelling occurs in response to local pressure effects, ischemia, and postischemic response cascades. Some authors consider ischemia to be the primary mechanism for subjacent hemispheric brain swelling [21,22]. Rats with induced ASDH had decreased brain water in the underlying hemisphere when injected with kynurenic acid (a broad-spectrum excitatory amino acid antagonist), suggesting that excitatory amino acids might also be involved in this process [23]. Com-

promise of cerebral blood flow in the involved hemisphere may further contribute to evolving edema [24,25].

Previously, strangulation injury has been proposed as the causative mechanism underlying hemispheric necrosis when seen in the context of abusive injury [9,26]. Yet in only one of four published cases was there pathologic confirmation of strangulation injury to the carotid [9,26]. Of the three patients with evidence of strangulation in this series, only one had imaging findings (internal carotid artery distribution hypoattenuation) correlating with the pattern of neck injuries.

Given the well-documented association of ASDH and hemispheric necrosis, a causal relationship of infarction to strangulation injury per se would be unproved, although strangulation should always be considered in the evaluation of focal or diffuse brain swelling in children with NAHI, particularly if there is external evidence of trauma. Blunt force head or neck trauma and the subsequent development of vascular distribution cerebral infarcts has been reported in a number of case reports in adults [27,28]. Traumatic vascular injury resulting from strangulation should therefore only be diagnosed if there is well-defined clinical, imaging, or postmortem examination evidence, such as absence of carotid pulsations contralateral to an observed hemiplegia, absent venous pulsations in the ipsilateral retina, or positive findings with carotid or transcranial color Doppler, magnetic resonance angiography, or cerebral angiography [29]. Examination of carotid arteries and soft tissues of the neck may be useful in the event of death, and postmortem carotid and vertebral angiography has been suggested as another adjunctive tool [30].

Comparing studies of children with NAHI is difficult because of the considerable differences in inclusion and exclusion criteria, as well as study purpose. Of the case series that have noted initial imaging patterns of cerebral swelling after NAHI, only Cohen et al. [6] reported follow-up imaging findings. In 37 children with NAHI, 65% had imaging evidence of brain swelling, focal in 75%. Of the 24 patients with follow-up CT scans, 25% had generalized atrophy, and 71% had focal regions of atrophy. In the most recent follow-up study of NAHI to date, Duhaime et al. [31] described the clinical outcome of 14 patients 5 years 6 months to 15 years 6 months after injury but did not provide current neuroimaging, psychologic, or behavioral data. Zimmerman et al. [8] noted a 50% incidence of infarction in 14 children after NAHI, but, like Cohen et al. [6], did not discuss details of infarct patterns in relationship to age.

The relationship of onset of clinical symptoms to injury was determined in nine patients in this series. These severely injured patients (mean CCS 4) were rendered immediately and continuously unconscious from the time of assault. These children did not have a lucid interval, which is typical for severe concussive syndromes and consistent with animal and human data [32]. Frequently, histories are given of minor falls one or more days before admission that are unassociated with any intervening symptoms, followed by the abrupt onset of respiratory difficulty and unresponsiveness just before presentation. This series supports the hypothesis that infants and young children severely injured by nonaccidental mechanisms do not experience a lucid interval nor do they recover to their premorbid state of function after injury.

EPTS occur more frequently after NAHI than after head injury from unintentional mechanisms at any age [33-35]. Eleven (79%) of 14 children in this series had EPTS, consistent with previous reports in which frequency of EPTS in NAHI ranged from 30% to 100% (mean 68%) [1,7,19,26,36,37]. None of these studies delineated the number and type of infarctions, however, nor did they use consistent inclusion criteria.

The basis for this observation is probably multifactorial. First, a substantial proportion of children with NAHI have associated hypoxic-ischemic injury. Second, the immature central nervous system has greater susceptibility to ischemic injury and to seizures in general [14,19,38]. Third, infants have increased synaptic and excitatory amino acid receptor density [38,39]. Suppressive synaptic networks are incompletely developed and gamma-aminobutyric acid receptors are structurally and functionally different in the immature central nervous system [39]. In addition, excitatory amino acids released by injured neurons and glia excessively activate immature *N*-methyl-D-aspartate and AMPA receptors in the hippocampus, contributing to posttraumatic seizure activity, as well as neuronal injury and cell death [40].

Although the pattern of brain swelling in the authors' series did not predict EPTS risk (children with diffuse or focal brain swelling in this series were equally likely to have EPTS), it may predict outcome and the development of posttraumatic epilepsy. Generalized seizures correlated with diffuse brain injury and negatively with outcome. Sixty percent of children with diffuse brain swelling developed posttraumatic epilepsy, compared with no patients with focal brain swelling. Partial seizures predominated in this series, as in adults with traumatic brain injury, and were generally difficult to control. The high incidence and poor control of EPTS in NAHI supports the early use of prophylactic antiepileptic medication, particularly if an infarction syndrome is suspected. There is a need for prospective randomized studies to further examine this issue.

Survivors in this series uniformly exhibited sequelae, not unexpected given the inclusion criteria. Outcome paralleled extent and pattern of brain swelling, infarction, and degree of hypoxic-ischemic insult. Cognitive delay, cortical visual loss, and focal neurologic findings were common findings. Younger children with diffuse brain swelling tended to have a worse outcome, both developmentally and by COS, consistent with previous reports of worse functional outcome in younger than in older children after traumatic brain injury and in children with NAHI vs unintentional injury (Table 4) [14,19,31]. Slow-

ing of the rate of growth of head size (reflective of brain growth) became apparent within 2 months after injury and correlated with extent of injury and the eventual development of microcephaly in 73% of survivors. All children presenting with diffuse brain swelling developed microcephaly compared with 50% of patients with focal (hemispheric or anterior circulation) swelling, probably a reflection of the extent of hypoxic-ischemic injury.

In summary, we have described age-related differences in morbidity and a range of cerebral injury patterns in a population of children with NAHI identified using strict inclusion criteria. Young children with severe NAHI in this series did not have a lucid interval. Cerebral infarction should be included as one of the recognized consequences of NAHI, particularly because early identification of evolving infarction may prove useful in developing better intervention strategies. Given the individual and societal costs of childhood head trauma, further research is needed focusing on the pathogenesis and treatment of these injuries [14,41].

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