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Neck injuries in young pediatric homicide victims

Clinical article

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Object. In this study, the authors estimate the prevalence of injuries to the soft tissue of the neck, cervical vertebrae, and cervical spinal cord among victims of abusive head trauma to better understand these injuries and their relationship to other pathophysiological findings commonly found in children with fatal abusive head trauma.

Methods. The population included all homicide victims 2 years of age and younger from the city of Philadelphia, Pennyslvania, who underwent a comprehensive postmortem examination at the Office of the Medical Examiner between 1995 and 2003. A retrospective review of all available postmortem records was performed, and data regarding numerous pathological findings, as well as the patient's clinical history and demographic information, were abstracted. Data were described using means and standard deviations for continuous variables, and frequency and ranges for categorical variables. Chi-square analyses were used to test for the association of neck injuries with different types of brain injury.

Results. The sample included 52 children, 41 (79%) of whom died of abusive head trauma. Of these, 29 (71%) had primary cervical cord injuries: in 21 there were parenchymal injuries, in 24 meningeal hemorrhages, and in 16, nerve root avulsion/dorsal root ganglion hemorrhage were evident. Six children with abusive head trauma had no evidence of an impact to the head, and all 6 had primary cervical spinal cord injury (SCI). No child had a spinal fracture. Six of 29 children (21%) with primary cervical SCIs had soft-tissue (ligamentous or muscular) injuries to the neck, and 14 (48%) had brainstem injuries. There was a significant association of primary cervical SCI with cerebral edema ($p = 0.036$) but not with hypoxia-ischemia, infarction, or herniation.

Conclusions. Cervical SCI is a frequent but not universal finding in young children with fatal abusive head trauma. In the present study, parenchymal and/or root injury usually occurred without evidence of muscular or ligamentous damage, or of bone dislocation or fracture. Moreover, associated brainstem injuries were not always seen. Although there was a significant association of primary cervical cord injury with cerebral edema, there was no direct relationship to brainstem herniation, hypoxia-ischemia, or infarction. This suggests that cervical spinal trauma is only 1 factor in the pathogenesis of these lesions. *(DOI: 10.3171/2008.11.PEDS0835)*

Key Words • **abusive head trauma** • **cervical cord injury** • **neck injury**

Since John Caffey first identified maltreatment as the cause of unexplained skeletal and brain injuries in infants in 1946,[11] considerable research has been published describing the epidemiology, patterns, and mechanisms of injury associated with abusive head trauma in young children. Although underrecognition still exists,[24] abusive head trauma affects ~ 17 in 100,000 US children annually.[27] The exact mechanism of injury in abusive head trauma, and the relative contributions of shaking and impact continue to be debated. Although some researchers report that shaking alone is sufficient to cause injury,[8,9,12,23,31,43] others conclude that blunt impact is necessary to cause significant primary brain trauma.[14]

Nevertheless, the clinical manifestations and outcomes of abusive head trauma have been well described. Many studies have documented that the majority of young infants who suffer abusive head trauma have extraparenchymal hemorrhage,[5,13,14,34,35] > 80% have retinal hemorrhages,[14,35] and 30–50% have skeletal injuries of various ages.[7,13,34] Between 60 and 85% show evidence of impact injury, mani-

Abbreviations used in this paper: OME = Office of the Medical Examiner; SCI = spinal cord injury; SDH = subdural hemorrhage.

fested by skull fractures or cranial soft-tissue contusions, some of which are not apparent until postmortem examination.[8,14,17,34] The mortality rate after abusive head trauma approaches 20%,[5,30] and in those who survive, > 75% will have permanent neurological impairments.[2,3,5,30]

Recent research on abusive head trauma has explored the relationship between abusive head trauma and hypoxic cerebral injury. Hypoxic-ischemic injury is common in patients with abusive head trauma[7,17,18,28,41] and it is theorized that trauma-induced apnea leads to cerebral hypoxia and/or ischemia.[17,18,25,28] Some investigators have speculated that secondary injury consequent to hypoxia-ischemia, edema, or infarction may be a stronger determinant of a patient's ultimate neurological outcome than the primary injury or the associated presence of subdural and subarachnoid hemorrhage, or diffuse axonal injury.[15,25,28]

Although patterns of brain injury in infants with abusive head trauma have been well described, there has been far less attention directed toward the prevalence and clinical significance of neck injuries in these infants. Because many investigators have postulated that a shaking whiplash event, with or without head impact, may be the primary mechanism in many infants with inflicted brain injuries,[12,14,23] it is reasonable to hypothesize that the necks of these young infants may also be injured. Additionally, infants and young children are at an increased risk of flexion, distraction, and rotational injuries to the cervical spine due to their horizontally oriented facet joints, incompletely formed uncovertebral joints, increased laxity of the associated spinous ligaments, immature paraspinous musculature, and a relatively large head-to-body ratio.[19] Some researchers have suggested that with purely rotational injuries (such as shaking), injury to the neck and cervical spinal cord should be universal.[1]

Despite concerns about the contributions of neck injury to the sequelae of abusive head trauma, the few case series in the literature have been limited in sample size and scope of data reported. We therefore report the postmortem findings in a cohort of young homicide victims in whom complete pathological analyses of the brains and spinal columns were performed. We have focused on estimating the prevalence of injuries to the soft tissue of the neck, the cervical vertebrae, and the cervical spinal cord to better understand these injuries and their relationship to other pathophysiological findings common in children who have sustained fatal abusive head trauma.

Methods

The target population for this study was all homicide victims 2 years of age and younger from the city of Philadelphia, Pennsylvania, in whom a comprehensive postmortem examination was done at the OME between 1995 and 2003. Children were eligible if the chief medical examiner in consultation with the senior neuropathologist onsite at the OME determined the manner of death to be homicide; all available information provided to the OME was used before reaching this decision. Findings were retrospectively reviewed for analysis. In our review, we included certification of identification reports, case registration summaries (including case histories), general autopsy reports, and neuropathology reports. Limited medical records, including imaging studies, were available in some cases. The general autopsy was completed by a forensic pathologist at the OME, and a full neuropathological examination was performed by a forensic neuropathologist (L.B.R.-A.). Examination of the nervous system included removal of the brain, spinal cord, and eyes in continuity, followed by gross and microscopic examination according to the standard techniques described elsewhere.[26] Demographic information, clinical history, and injury data were collected. Demographic variables included sex, race, and age at death. Race was categorized as Caucasian, African-American, Asian, Hispanic, Native American, or other, as recorded in the case registration summaries.

In addition to the documentation of cause and manner of death (homicide), the clinical history included the time interval between injury and death, and an assessment of the mechanism of death. Where possible, the time interval was calculated in days from reported symptom onset to death, as recorded in the case registration summaries. The mechanism of death was determined and reported by the chief medical examiner based on assessment of the available investigative data and results of the postmortem examination. Based on this report, we separated the cases into 2 categories: abusive head trauma and death consequent to non-CNS injury, including asphyxiation and abdominal trauma.

Table 1 lists all the pathological findings that were recorded. Neck injury data included the presence of cervical spine dislocation or fracture, ligament or muscle injury, or any soft-tissue injury of the neck (such as hematoma or bruising). Primary cervical SCIs included any cervical cord contusion, laceration, or transection; vertebral artery injury; nerve root avulsion/dorsal root ganglia hemorrhage; and meningeal hemorrhage (epidural, intradural, subdural, and/or subarachnoid). Given its proximity to the cervical cord, any traumatic injury to the brainstem (such as a laceration or hemorrhage) was also recorded.

Primary traumatic brain lesions included contusions or lacerations, parenchymal hemorrhage, and meningeal hemorrhage. Cerebral contusions and lacerations were further classified as superficial contusions or lacerations (defined as olfactory bulb/tract injury or cortical contusions/lacerations) and deep contusions and lacerations (defined as axonal injury, gliding injury, injury to the corpus callosum, or ventricular tears/lacerations). The presence or absence of cerebral edema, cerebral hypoxia-ischemia, a cerebral infarction, and herniation were determined as follows according to the usual neuropathological criteria[22] and were noted for each patient.

Cerebral Edema. The diagnosis of cerebral edema was determined by comparing the brain weight of the homicide victim with the expected value for a child of that age. Absolute figures cannot be given here because brain weight changes from birth to 24 months of life. Beginning with the brain weight determination, the pathologist then examined the specimen for sulcal effacement consequent to gyral widening and flattening. Microscopic diagnosis of cerebral edema rests on the presence of status spongiosis, acute swelling of oligodendroglia, and exaggeration of perivascular and pericellular shrinkage artifact.

TABLE 1: Summary of pathological findings in 52 infant homicide victims*

Neck	Cervical Spinal Cord	Retinal Hemorrhage	Cranial Findings	Secondary Cerebral Findings	Extracranial Findings
ligamentous injury	parenchymal cord injury meningeal hemorrhage nerve root avulsion/dorsal root	bilat unilat	meningeal hemorrhage epidural subdural subarachnoid	cerebral edema brainstem herniation hypoxia-ischemia infarction	extracranial fx rib fx
muscle injury			intracerebral bleeding		concomitant visceral injury
other soft-tissue injury			cerebral contusions/lacerations superficial deep		facial bruising body bruising
cervical spine dislocation or fracture			DAI		
			evidence of BFT skull fx galeal/subgaleal hematoma bruises to scalp superficial cerebral contusions/lacerations		
			brainstem trauma		

* BFT = blunt force trauma; DAI = diffuse axonal injury; fx = fractures.

Hypoxia-Ischemia. The diagnosis of hypoxia-ischemia was based on established gross and microscopic features of the specimen. These often, but not always, included severe superficial and deep congestion, deep pink to purple discoloration of the gray matter (consequent to the pathophysiological mechanism of cerebral autoregulation), and microscopic identification of acute neuronal necrosis.

Cerebral Infarction. Cerebral infarction may take various forms that differ according to whether the infarction is acute, subacute, or chronic, and whether it is in a vascular distribution, such as in the middle cerebral artery, a border zone lesion, or laminary necrosis.

Herniation. Evaluation of a brain for evidence of herniation is a routine part of gross examination. Criteria for determination of herniation included grooving of unci and/or parahippocampal gyri, compression of the third cranial nerve in association with herniation of these structures, cingulate herniation beneath the falx cerebri, cerebellar tonsillar grooving, the presence or absence of associated brainstem swelling, presence of Duret hemorrhages, identification of the Kernohan notch phenomenon, and rarely, herniation of the temporal poles over the sphenoid ridge into the anterior fossa.

Meningeal hemorrhage was characterized as epidural, subdural, and/or subarachnoid if present. Evidence of blunt or impact trauma to the head, such as skull fractures, galeal or subgaleal hematomas, superficial contusions and lacerations, and any scalp hematomas or bruising was recorded. Based on injury identification, children with abusive head trauma were then classified as either having visible evidence of impact or no visible evidence of impact. The presence of retinal hemorrhages, determined through examination of the eyes, was also recorded.

Collected extracranial injury data included the presence of other injuries, such as rib fractures, extremity fractures, abdominal trauma (such as lacerations or contusions to internal organs or the presence of a hemoperitoneum), and cutaneous bruising or hematomas.

All data were abstracted from the autopsy reports and entered into an Access database (Microsoft Corporation). Data were then imported into STATA version 8.2 software (STATA Corporation) for analysis. Data were described using means and standard deviations for continuous variables, and frequencies and ranges for categorical variables. Chi-square analyses were used to test for the association of neck injuries with different types of brain injury.

This study was reviewed and approved by the Institutional Review Board of the Children's Hospital of Philadelphia.

Results

There were a total of 52 homicide victims 2 years of age or younger in the city of Philadelphia, Pennsylvania, between 1995 and 2003 who underwent postmortem examinations at the OME. Of this group, 41 (79%; 95% CI 65.3–88.9) died of abusive head trauma, and 10 died of other mechanisms, including 3 who died of asphyxiation and 7 who died of blunt abdominal or body trauma. An additional child died of complex injuries sustained when her mother jumped from a second story window with the infant in her arms. Although the young child had significant neurotrauma along with other injuries, including organ laceration and multiple fractures, the case was unique and therefore classified separately.

Young children with abusive head trauma were significantly younger at death than those killed by other means ($p = 0.036$). Infants with abusive head trauma were

TABLE 2: Population demographics of 52 infant homicide victims*

Characteristic	Abusive Head Trauma (41 children)	Other MOD (11 children)	p Value
sex (%)			
male	15 (36.6)	6 (54.6)	0.28
female	26 (63.4)	5 (45.4)	
age at death (%)			
<1 yr	22 (53.7)	2 (18.2)	0.036
1–2 yrs	19 (46.3)	9 (81.8)	
race (%)			
African-American	31 (75.6)	7 (63.6)	0.243
Caucasian	8 (19.6)	2 (18.2)	
Hispanic	1 (2.4)	2 (18.2)	
Asian	1 (2.4)	0 (0)	
length of time btwn injury & death (%)			
≤1 day	22 (57.9)†	9 (81.8)	0.147
≥2 days	16 (42.1)†	2 (18.2)	

* MOD = mechanism of death.

† Due to insufficient data, 3 children were not included in this group.

more often female and survived longer after injury, but these differences were not statistically significant. There were no racial differences between the children who died of head trauma and those who died of other mechanisms (Table 2).

Medical records, some including imaging results, were available for 22 of the 52 children. Four were dead on or shortly after arrival at the hospital, and none had undergone MR imaging of the neck or cervical spinal cord. Six children had CT scans of the cervical spine, all of which were negative for fractures, subluxation, or soft-tissue swelling. Magnetic resonance imaging of the brain was done in 5, yielding abnormal results in all cases with varying degrees of hemorrhage, infarction, and hypoxic injury. Of these 5, the cause of death in 4 was abusive head trauma, and the fifth child died of asphyxiation.

Neck Injuries

Twenty-nine of 41 children with abusive head trauma (71%; 95% CI 54–84) had primary injuries to the cervical spinal cord; 2 had cervical SCIs alone without associated primary traumatic brain injuries, and 1 of these was pronounced dead on arrival to the emergency department. No clinical history was available for this child, who also had multiple other injuries, including rib fractures, a femur fracture, liver lacerations, multiple subgaleal hemorrhages of scalp, and diffuse superficial contusions and abrasions to the chest and head. No cervical spinal cord images were obtained in this child. The second child died 3 days after presenting with extreme lethargy and respiratory distress. She initially received a diagnosis of acidemia and encephalopathy from a possible inborn error of metabolism, and was found on postmortem examination to have only a traumatic cervical SCI. There was insufficient clinical data to determine whether this patient had any limb movement or respiratory effort before death.

Among the 29 children with cervical SCIs, there were some similarities in pathological findings: 21 (72%) had parenchymal injuries, such as cord contusions, lacerations, or transections; 24 (83%) had meningeal hemorrhages; and 16 (55%) had nerve root avulsions or dorsal root ganglion hemorrhages. Five children had parenchymal injuries without meningeal hemorrhaging, 8 had meningeal hemorrhaging without parenchymal injuries, and 16 had both parenchymal and meningeal injuries. Of the 16 with nerve root avulsions or dorsal root ganglion hemorrhaging, 14 also had meningeal hemorrhages. Ten children had meningeal hemorrhaging without nerve root avulsion or dorsal root ganglion hemorrhaging.

Only 6 (21%) of 29 children with primary cervical SCIs had soft-tissue injuries to the neck. Among these, 4 had muscle, 3 had ligamentous, 2 had other soft-tissue injuries, and 3 had both muscle and ligamentous injuries. Soft-tissue injury to the neck was uncommon overall, as only 10 (19%) of 52 homicide victims had such an injury. Of these 10, 9 were victims of abusive head trauma and only 1 was the victim of another mechanism of death (asphyxiation). Among the 9 children with abusive head trauma and soft-tissue injuries to the neck, 6 had muscle injuries, 3 had ligamentous injuries, and another 3 had other soft-tissue injuries to the neck. Overall, therefore, 9 (22%) of 41 children had abusive head trauma, 1 (9%) of 11 children had other mechanisms of death (not head trauma), and 1 of 3 victims of asphyxiation had soft-tissue injuries to the neck. There were no children with cervical spine fractures or dislocations (Table 3).

We examined the relationship between the cervical spinal cord and the mechanism of abusive head trauma (impact vs no evidence of impact). We found a trend toward universal SCI in children without blunt impact; of the 6 children without visible signs of impact, all had primary cervical spinal cord and regional injuries, compared with 23 (65.7%) of the 35 with evidence of blunt trauma ($p = 0.088$).

TABLE 3: Pathological findings in the necks of infant homicide victims

Finding	MOD		
	Abusive Head Trauma (41 infants)	Cervical SCI (29 infants)	Other (11 infants)
any soft-tissue injury to neck (%)	9 (22)	6 (20.7)	1 (9.1)
muscle injury	6 (14.6)	4 (13.8)	1 (9.1)
ligamentous injury	3 (7.3)	3 (10.3)	0 (0)
other soft-tissue injury	3 (7.3)	2 (6.9)	0 (0)
cervical spine dislocation or fracture	0 (0)	0 (0)	0 (0)

The presence of primary cervical SCI was examined for any association with cerebral edema, infarction, hypoxia-ischemia, and herniation among the children with abusive head trauma. No association was found between primary cervical SCI and hypoxia-ischemia ($p = 0.853$), infarction ($p = 0.44$), or herniation ($p = 0.16$). There were insufficient clinical data to correlate these findings with clinical manifestations typically associated with SCI, such as apnea or paralysis. However, there was a significant association between primary cervical cord injury and cerebral edema ($p = 0.036$; Table 4).

Head Injuries

Thirty-seven (90%) of 41 infants and young children with abusive head trauma had intracranial meningeal hemorrhaging. This included SDHs in 34 (92%), and subarachnoid hemorrhages in 34 (92%). Epidural hemorrhage was found in 8 children (21%); all epidural hemorrhages were reported as small and none were clinically apparent. Seven of the 8 were associated with overlying skull fractures. Twenty-seven of the 41 children (66%) had evidence of intracerebral bleeding. In 1 child, the condition of the brain was such that the presence of cerebral contusions and lacerations could not be ascertained, but of the remaining 40 children, 32 (80%) had some cerebral contusion or laceration, whether superficial or deep. Twenty-six (65%) of 40 had superficial cerebral contusions and lacerations, and 23 (58%) of 40 had deep cerebral contusions and lacerations. Six children (15%) had diffuse traumatic axonal injury.

Among the 41 children with abusive head trauma, 35 (85%) had evidence of blunt head trauma. Evidence of blunt trauma included skull fractures in 13 (37%), galeal or subgaleal hematomas in 24 (69%), scalp bruising in 19 (54%), and superficial cerebral contusions and lacerations in 23 (54%).

Eleven of the 41 children (27%) with fatal abusive head trauma had extracranial fractures (such as rib or extremities). Eight of 11 (73%) had 1 or more rib fractures. Among those 8, rib fractures were the only fractures in 7 (88%), with the other child also having a femur fracture. Three of 11 children (27%) had only extremity fractures without rib fractures; these consisted of a tibial metaphyseal fracture, a clavicle fracture, and fractures of the ulna and radius. Twenty-one children (51%) had concomitant visceral injuries such as liver, splenic, or renal injuries. Twenty-seven children (66%) had facial bruising, and 20 (49%) had bruising to the rest of the body.

Of the 41 children with abusive head trauma, 27 (66%) had evidence of cerebral edema, and 23 (56%) had hypoxic-ischemic injury. Due to the physical condition of the brain specimens, determination of cerebral herniation could not be made in 1 case and determination of infarction could not be made in another. However, of the remaining children, 9 (23%) had herniations and 6 (15%) had cerebral infarctions. Cerebral edema, hypoxia-ischemia, infarction, and herniation were not specific to children who died of abusive head trauma; in fact, there was no statistically significant difference in the proportions of children with these findings by mechanism of death (Table 5).

Brainstem Injuries

Brainstem trauma was found in 16 (40%) of 40 children with abusive head trauma (1 specimen could not be examined due to poor condition). Fourteen of 16 (88%) also had primary cervical SCI.

Retinal Hemorrhages

Thirty of the 41 children (73%) with abusive head trauma had retinal hemorrhages, which were bilateral in 21 cases and unilateral in 9. Of the 11 children who died without evidence of CNS injuries, 7 had eyes available for neuropathological examination, none of which had retinal hemorrhages. There were 3 children who had both SDHs and retinal hemorrhaging but no external or other injuries.

TABLE 4: Association of primary cervical SCIs with cerebral edema, hypoxia-ischemia, infarction, and brainstem herniation among infants with abusive head trauma

Finding	Cervical SCI (29 infants)	No SCI (12 infants)	OR*	p Value
cerebral edema	22 (75.9%)	5 (41.7%)	4.4 (1.09–17.7)	0.036
brainstem herniation	8 (28.6%)	1 (8.33%)	4.4	0.160
hypoxia-ischemia	16 (55.2%)	7 (58.3%)	0.88 (0.24–3.31)	0.853
infarction	5 (17.9%)	1 (8.33%)	2.4	0.44

* Numbers in parentheses are 95% CIs.

TABLE 5: Association of cerebral edema, hypoxia-ischemia, infarction, and brainstem herniation lesions with MOD among 52 infant victims of homicide

Finding	Abusive Head Trauma (%)	Other MOD (%)	p Value
cerebral edema	27 (65.9)	7 (63.6)	0.89
brainstem herniation	9 (22.5)	1 (9.1)	0.321
hypoxia-ischemia	23 (56.1)	6 (54.6)	0.93
infarction	6 (15)	1 (9.1)	0.614

Although retinal hemorrhages were found in children who had evidence of isolated blunt abusive head trauma without any cervical cord injury, children who had both cervical cord injuries and evidence of blunt head trauma had the highest rate of retinal hemorrhages (92%; Table 6).

Discussion

In this study of homicide victims 2 years of age or younger, cervical SCIs were commonly found in those who died of abusive head trauma and occurred in those with and without visible evidence of impact injury. There have been several reports of spinal cord and neck injuries in children with abusive head trauma, but our case series is the largest and most complete analysis of these injuries reported to date.[16–18,20,23,33,38–42,44,45] Several studies of pediatric cervical cord injury of various causes have included abuse as a mechanism of trauma, but these accounted for 4% or less in most case series.[10,21,29,36,37] Currently, it seems that radiographic evaluation (primarily MR imaging) of the apparently intact neck has not been useful in identifying SCI. The presence of cervical SCI, hematomas, and nerve root damage has been documented postmortem in infants who have sustained abusive head trauma;[16–18,23,25,41] however, MR imaging failed to identify the cervical injuries among inpatients.[16]

Our findings corroborate those of others who report that the majority of infants with fatal abusive head trauma exhibit external evidence of blunt trauma[14] and that a high proportion have SDHs[10–14] and retinal hemorrhages.[14,35] A considerable number had concomitant skeletal injuries.[7,13,34]

Damage to the cervical spinal cord and roots has also been reported by others. Feldman et al.,[16] in a study of 5 victims of abusive head trauma who underwent autopsy, found that 1 had diffuse thin subdural blood (in continuity with thin cranial subdural blood) overlying the upper cervical cord, and 3 had subarachnoid blood overlying the cord (associated either with cranial subarachnoid blood or extensively distributed subarachnoid blood). Hadley et al.[23] found that 5 of 6 patients with abusive head trauma had epidural and/or SDHs of cervical spinal cord at the cervicomedullary junction and 4 of 6 patients had evidence of ventral spinal cord contusions at high cervical levels on postmortem examination. In their case series of 4 victims of abusive head trauma who underwent autopsy, Johnson and colleagues[25] found that 1 had spinal cord contusion and laceration, and another had a cervical SDH.

Additionally, Geddes et al.[17,18] and Shannon et al.[41] reported significant rates of cervical cord injury based on expression of β-amyloid precursor protein utilizing the immunoperoxidase technique in infants who sustained fatal abusive head trauma. Geddes and associates[17,18] found that 11 of 37 infants had epidural cervical hemorrhages and focal axonal damage involving the brainstem and spinal nerve roots. The findings of Shannon and colleagues[41] were even more striking, as 7 of 11 infants with no evidence of impact injury exhibited axonal injury of the cervical spinal cord. This was particularly prominent at the root entry zone.

One investigator has suggested that in fatal abusive head trauma in infants, cervical cord injury would be a universal finding because cord injury occurs at lower distraction forces than does primary cerebral injury.[1] Other investigators, however, have identified errors in these mathematical calculations.[32] While we have found that cervical cord injury is common, it is not universal. It was identified in the 6 children who had no visible evidence of

TABLE 6: Retinal pathology in infants with abusive head trauma and cervical SCIs*

Type of Injury	Retinal Pathology			
	None (%)	Minimal RH (%)	Bilat RH (%)	Retinal Detachment (%)†
BFT w/o cervical SCI (15 infants)	9 (60)	2 (13.3)	4 (26.6)	1/4 (25)
BFT & nerve root/meningeal trauma (15 infants)‡	3 (20)	1 (6.6)	11 (73)	5/11 (45)
cervical SCI (15 infants)	2 (13.3)	4 (26.6)	9 (60)	3/13 (23)
cervical SCI & nerve root trauma (2 infants)	0 (0)	0 (0)	2 (100)	1/2 (50)
cervical SCI & BFT (12 infants)	1 (8.3)	2 (16.6)	9 (75)	4/9 (44)
no cervical SCI or root trauma (16 infants)	11 (62)	0 (0)	5 (38)	1/5 (20)

* RH = retinal hemorrhage.

† The denominator represents cases in which retinal detachment was specifically noted and could be separated from artificial detachment secondary to processing of the specimen.

‡ No isolated nerve root/meningeal injury was present in the absence of blunt force trauma.

blunt impact trauma. Given results by Feldman et al.[16] that among 12 children with abusive head injury, no cervical cord injury was detected by MR imaging, these results may not be generalizable to nonfatally injured children. It may be that cervical cord injury is a marker for more severe injury.

There is currently some controversy regarding relationship of soft-tissue injuries and spinal cord trauma. Our data indicate that although cervical cord injury is common, adjacent soft-tissue injury occurs less frequently. Moreover, there was no evidence of fracture or dislocation. Absence of bone injury may perhaps be explained by the relative laxity and flexibility of spinal ligaments and musculature in the infant neck, which may be able to withstand more flexion and extension rotational forces than the spinal cord itself.

It has been postulated that cervical spinal/root injury initiates apnea, hypoxic-ischemic injury, and the subsequent death of these infant victims. Although this may be correct, there is evidence that infants with other types of neural and nonneural trauma also exhibit secondary CNS abnormalities such as hypoxic-ischemic lesions and edema. These may occur consequent to cerebral perfusion failure or biochemical abnormalities resulting from traumatic head injuries such as increased oxidative stress, which can mediate several cellular changes, any or all of which may lead to neuronal injury.[4,6,46]

The exact relationship between these commonly seen cervical injuries and clinical symptoms remains unclear. Unfortunately, we did not have adequate clinical data to correlate with our pathological findings, but it is possible that cervical SCIs may contribute significantly to apnea and other clinical correlates of abusive injuries. It is also possible that some infants have less severe SCIs that cause transient dysfunction and result in apnea or hypoventilation, but that are not visible on the postmortem examination or do not cause visible changes on MR images in survivors.

The present study is not without limitations. Our sample population is small, which limits our subgroup analysis. Clinical histories in these patients were spotty and often unavailable, so we were unable to perform any analysis of injuries in relation to the presenting complaint. Only 1 pediatric forensic neuropathologist performed the postmortem examinations and described the findings, and although this neuropathologist is very experienced, there was no second reviewer to support her findings objectively. Lastly, this study is generalizable to fatally injured children only, and the significance of these findings to the presentation and diagnosis of neck injuries in nonfatally abused children is unclear.

Future directions include correlating clinical and radiographic findings to pathological findings, as well as further exploration of the relationship and pathway between cervical SCI and death in a larger population.

Conclusions

Abusive head trauma is the most common mechanism of death among infant homicide victims. Victims exhibit a high frequency of SDH, retinal hemorrhages, and skeletal injuries. Cervical SCIs are also common, but not universal. In the present study, parenchymal and/or root injuries usually occurred without evidence of muscular or ligamentous damage, or bone dislocation or fracture. Moreover, associated brainstem injury was not always seen. Although there was a significant association of primary cervical SCI with cerebral edema, there was no direct relationship with brainstem herniation, hypoxia-ischemia, or infarction. This finding suggests that cervical spinal trauma is only 1 factor in the pathogenesis of these lesions. Future study may help determine whether SCI plays a major role in the common findings of apnea and hypoxic-ischemic brain injury in infants who have sustained abusive head trauma.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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