

Imaging of spinal injury in abusive head trauma: a retrospective study

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Abstract

Background Spinal imaging has been a neglected part of abusive head trauma (AHT) imaging. As most of the radiographs and CT spine are negative in AHT in infants, the cervical spine is assumed to be normal. There is increasing evidence in the role of injury to brainstem and cervical cord in the pathogenesis of AHT. In addition, in courts of law, there is fierce debate about AHT, its mimics and other disparate nontraumatic diagnoses explaining the neuroradiological and skeletal findings. However, this discussion ignores the evidence and significance of spinal injury. We sought to study the cervical spine in an AHT cohort to understand the true prevalence of spinal injuries in AHT and contrast it with cohorts of accidental and nontraumatic groups to give the clinicians a robust diagnostic tool in evaluating AHT.

Objective The purpose of this study is to compare the relative incidence of spinal ligamentous and soft-tissue abnormalities on spinal MRI among three groups of children ages <48 months: 1) those with AHT, 2) those with accidental trauma, and 3) those with nontraumatic conditions.

Materials and methods This comparative study included 183 children who underwent spine MRI: 67 with AHT, 46 with accidental trauma and a clinical suspicion of spinal injury, and 70 with nontraumatic conditions. Clinical and radiographic

findings were collected in all cases and were analyzed retrospectively to identify MRI evidence of traumatic spinal injuries. The incidence of spinal injuries among the three groups was compared. The incidence of spinal ligamentous injuries was calculated for those with and without radiographic evidence of hypoxic-ischemic encephalopathy. All comparisons were performed using Fisher exact test with $P < 0.05$ considered statistically significant.

Results Cervical spine ligamentous injuries (predominantly the nuchal, atlanto-occipital and atlanto-axial ligaments) were present in 78% of the AHT group, 46% of the accidental trauma group and 1% of the nontraumatic group; all of these differences were statistically significant. Among the AHT group, ligamentous injuries were statistically correlated with evidence of brain ischemia.

Conclusion Injury to the cervical spinal posterior ligamentous complex is common in AHT and even more prevalent than in clinically symptomatic traumatic cases. The high correlation between the radiographic findings of occipitocervical ligamentous injuries and hypoxic-ischemic brain injury is consistent with an interpretation that transient upper occipitocervical spinal cord injury in AHT leads to disordered breathing and results in hypoxic-ischemic encephalopathy. We recommend imaging the entire spine in AHT to properly identify and classify these injuries.

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Introduction

Abusive head trauma (AHT) is the most frequent cause of traumatic brain injury in infants with a mortality of 15–25% and significant neurological morbidity in the majority of

survivors [1]. Cranial injuries have been well described with subdural hemorrhage (SDH) and retinal hemorrhages in an average of 80% of cases; brain injuries such as focal or global hypodensities on CT, edema and loss of gray-white differentiation, superficial parenchymal contusions, and deep punctate hemorrhages in many on CT or MRI; and extracranial injuries such as rib and classic metaphyseal fractures [2].

Spinal injuries have rarely been reported in AHT [3–12]. However, with the proposal by Geddes [3] that the majority of axonal injuries (particularly those cranial to the cervicomedullary junction) are hypoxic-ischemic rather than traumatic in origin, the discovery that apnea or disordered breathing is common in cases of AHT [7], and the knowledge that infants are more susceptible to occipitocervical and upper cervical spine injuries [6], increasing attention has focused on identifying spinal injuries in AHT. The rarity with which spinal injuries have been identified in the past likely reflects the fact that they were rarely considered (and therefore not sought). In traditional autopsy protocols the brain is removed from the calvarium by cutting crudely across the cervicomedullary junction with a large scissors, which destroys the precise area of potential injury. A change in the autopsy protocol that led to removing the brain and spinal cord en bloc and an increasing focus on the spine and spinal cord have now allowed the identification of spinal injuries (most of them in the upper cervical spine) in up to 70% of fatal cases of AHT [4, 13]. Similarly, spinal injuries in cases of AHT have only rarely been reported on neuroimaging [3–12] because spinal imaging such as MRI is not routinely performed. In 2000, we began to routinely obtain cervical spine MRI scans in infants with AHT; in 2008, we expanded our protocol to include whole spine imaging. We have previously reported our findings on spinal subdural hemorrhages in this cohort [14].

In the present comparative study, we contrasted the findings on spinal neuroimaging (CT and/or MRI) among three groups of children ages younger than 48 months: 1) those with AHT, 2) those with accidental trauma and 3) those imaged for reasons other than trauma. The purpose of the study was to identify the incidence, distribution and radiological characteristics of bone, ligamentous and soft-tissue injuries of the spine in these three groups, contrast the abnormalities in cases of AHT with those in accidental trauma, and confirm these abnormalities as traumatic in nature by their absence among the control group with no identified trauma. Our aim was to better understand the mechanism of injury in AHT.

Materials and methods

After approval by the Human Subjects Protection Office, three groups of infants and young children <48 months of age were identified retrospectively. The first cohort included children

with AHT identified from an institutional AHT registry between 2000 and 2012 inclusive who had undergone a dedicated MRI of the spine (including a STIR [short tau inversion recovery] sequence). The second cohort included children with documented accidental trauma who were managed at our institution during the same time period and who had undergone a dedicated MRI of the spine (including a STIR sequence) because of a clinical concern for spine injury. The third cohort served as a control and included infants and children who underwent spinal MRI imaging (including a STIR sequence) between 2004 and 2012 inclusive for clinical indications other than trauma. These included masses of brain, spinal cord or neck, spinal cord malformation including concern for tethering of the cord, vascular malformations including concern for abnormalities of the vascular structures and PHACES syndrome (posterior fossa brain malformation, hemangioma, arterial anomalies, cardiac defect, eye abnormalities, sternal cleft/supraumbilical raphe), chiari, syrinx, structural abnormalities of head and neck, infection, neurological abnormalities of the extremities, torticollis, concern for neurofibromatosis type 1 (NF1), congenital kyphosis and syndromes such as Ellis van Creveld.

The clinical charts were reviewed by a pediatric neurosurgeon (M.S.D.) with 22 years of experience and pediatric neuroradiologist (A.K.C.) to identify demographic information, clinical presentation, radiographic findings and clinical outcomes. All cross-sectional images (including CT and MRI) of the brain and spine were independently analyzed by two experienced neuroradiologists (A.K.C. and T.Z.) with 7 and 6 years of experience, respectively. The images of the accidental and AHT cohorts were reviewed randomly with the readers blinded to the clinical history and diagnosis. After independent analysis, any interobserver differences were resolved by consensus. The spinal ligaments, membrane and paraspinal soft-tissue structures were assessed for evidence of injury. The presence of tears or hyperintensity on STIR sequence surrounding the ligaments or membranes was considered evidence of trauma (Figs. 1, 2, 3, 4 and 5). The images were also reviewed to identify hemorrhage within the epidural or subdural spaces; the character of the blood (nodular or smooth); the presence or absence of dural thickening, and any effacement of the subarachnoid space and/or mass effect on the cord (Figs. 6, 7 and 8). We identified blood as subdural in location if the epidural fat was normal in signal intensity and there was no displacement of dura; blood was considered to be epidural in location if there was displacement of the dura with nonvisualization of the epidural fat or stranding of the dural fat [14]. Hemorrhages were less hyperintense than fat on T1-weighted MRI sequences and were therefore readily distinguished; correlating the T1 findings with T2 and STIR imaging sequences also helped differentiate hemorrhage from fat within the spinal canal and eliminate false-positives. When both cranial and spinal SDH were

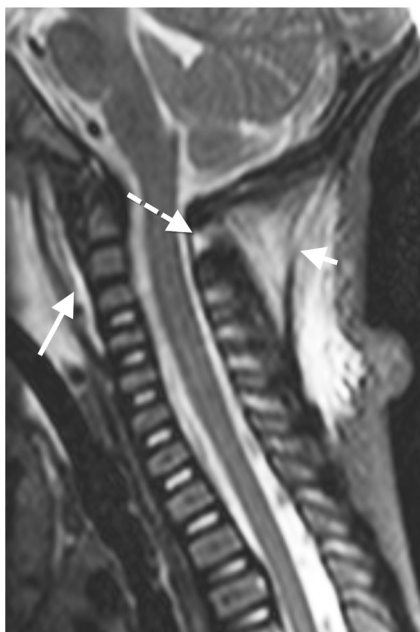


Fig. 1 Sagittal STIR image in an 8-month-old girl with AHT demonstrates injury to the nuchal ligament (*small arrow*) with associated hematoma and edema, injury to the atlanto-axial ligament (*dotted arrow*) and prevertebral edema (*large arrow*)

present, the similarity of the signal intensity in both locations was compared in an attempt to relate the two temporally. The contiguity of SDH in the posterior fossa and spine was also noted. Evidence of bone and spinal cord injury was also recorded. Finally, brain parenchymal injuries, particularly



Fig. 2 Sagittal STIR image in a 3-month-old girl with AHT demonstrates injury to interspinous (*arrow*) and nuchal ligaments



Fig. 3 Sagittal STIR image in a 15-month-old girl with accidental trauma demonstrates a partial tear with edema along the nuchal ligament. Injuries to the atlanto-axial (*white arrow*) and interspinous (*black arrow*) ligaments are also identified



Fig. 4 Sagittal STIR image demonstrates atlanto-axial ligament injury (*black arrow*) with spinal epidural hematoma (*white arrow*) in a female newborn with traumatic vacuum delivery with intracranial injury and hemorrhage

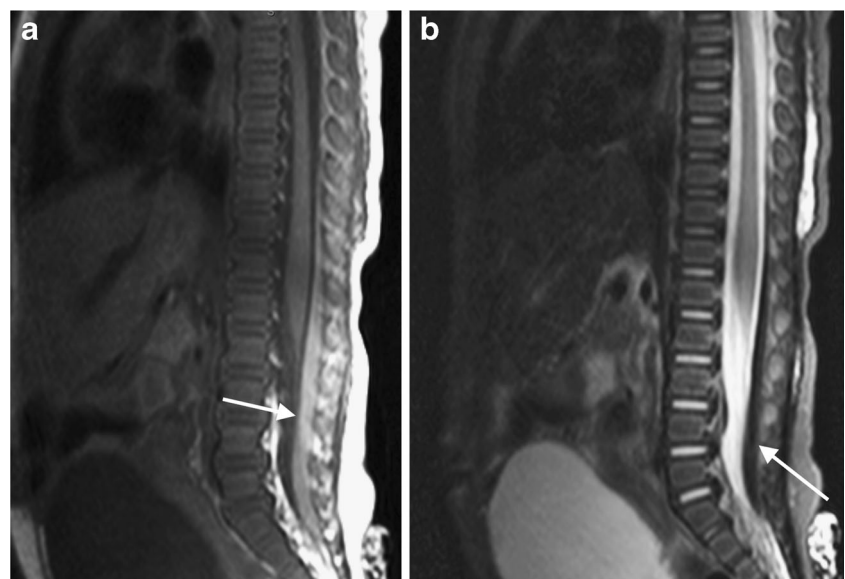
Fig. 5 Sagittal STIR image (a) in a 15-month-old boy involved in a motor vehicle crash demonstrates injury to the atlanto-axial ligament (*dotted arrow*), odontoid fracture (*small arrow*) with edema of the peridental ligament and prevertebral hematoma (*large arrow*). **b** Capsular injury with distraction of the atlanto-axial joint is also identified (*arrow*)



hypodensities, loss of gray-white differentiation, and ischemic changes on diffusion-weighted and ADC imaging were noted.

Statistical analyses were performed using SPSS software version 21 (IBM, Armonk NY, USA). Interobserver agreement was calculated using kappa statistics with κ values of 0–0.20, 0.21–0.40, 0.41–0.60, 0.61–0.80, 0.81–0.99 and 1.00 representing slight, fair, moderate, substantial, excellent and absolute agreement, respectively. Fisher exact test was used to compare the frequency of various spinal imaging findings among the three cohorts with a $P < 0.05$ considered statistically significant. Logistical regression analyses were performed to compare the variable findings in the three cohorts and between brain and spinal cord imaging findings.

Fig. 6 Sagittal T1 (a) and T2 (b) images in a 2-month-old boy with AHT, who presented with cardiac arrest and who had skull fracture and intracranial SDH, demonstrate T1 hyperintense and T2 hypointense spinal SDH (*arrow*) visualized only within the thoracolumbar spinal canal



Results

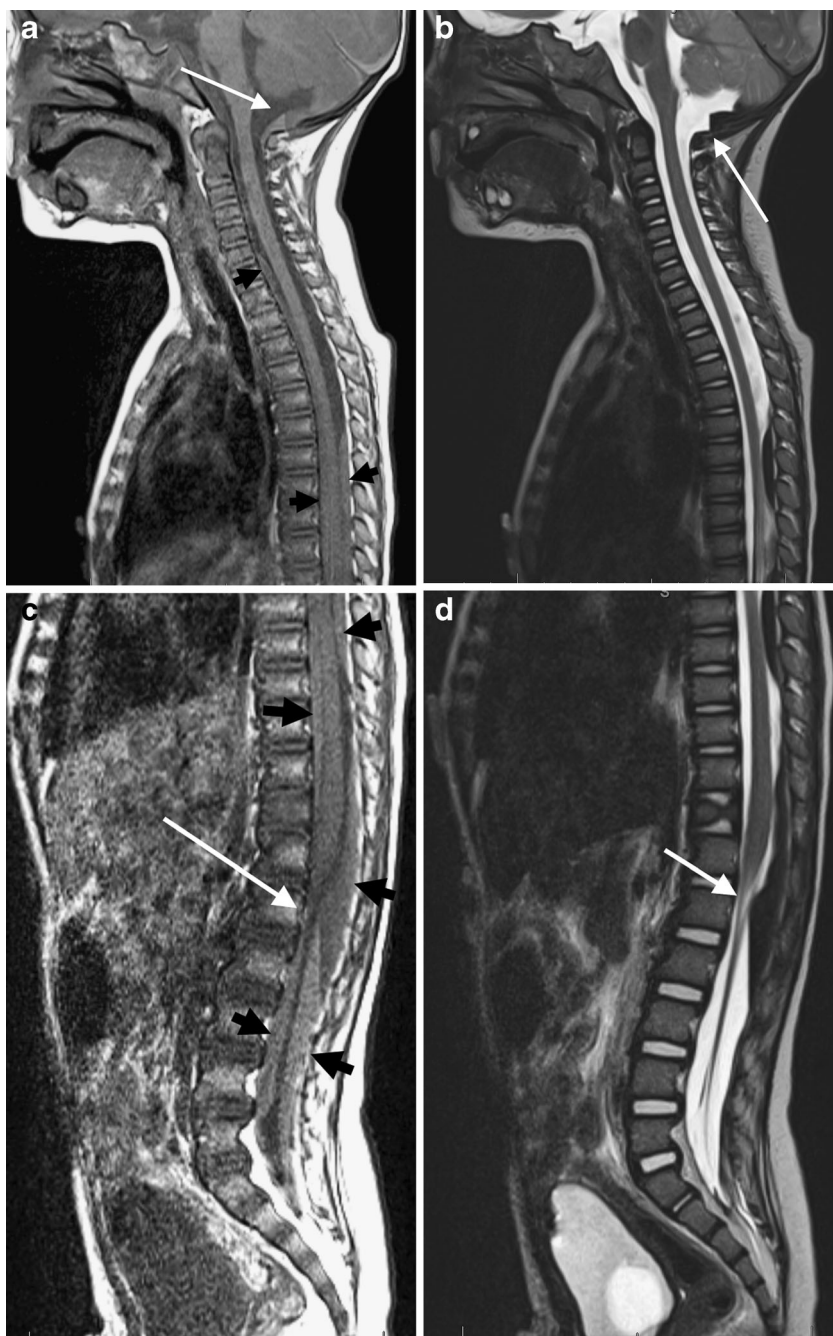
Interobserver agreement

There was excellent ($\kappa=0.95$) interobserver agreement for identifying the presence of spinal injuries among the AHT cohort and absolute agreement ($\kappa=1.00$) for identifying spinal injuries in the accidental trauma cohort.

AHT cohort

The AHT cohort included 67 children; 43 (64%) were boys and 24 (36%) were girls. The median age was 4 months.

Fig. 7 Sagittal T1 (**a, c**) and T2 (**b, d**) images in a 10-month-old boy with AHT, who had multiple metaphyseal and rib fractures, demonstrate posterior fossa SDH in continuity with spinal SDH. The spinal SDH extends along the entire spinal canal both anteriorly and posteriorly (*small black arrows on a and c*). The posterior fossa and spinal SDH are similar in signal intensity with layering T1 isointense/T2 hypointense and T1 hyperintense/T2 hyperintense blood. The *long white arrows* point to the layering effect of the SDH of different signal intensity



Cranial imaging in this group identified cranial SDH in 54 children (81%) and evidence of brain ischemia (focal or global hypodensities on CT, T2 fluid attenuated inversion recovery (FLAIR) hyperintensities and/or restricted diffusion on diffusion and apparent diffusion coefficient (ADC) sequences in 40 (60%). Cervical spine MRI was available in all 67 children. Thoracic and lumbar spine MRI was also available in 31 cases. The distribution of injuries is displayed in Table 1. Bone trauma was seen in only 4 cases (6%). Ligamentous injuries were much more common and were seen in 52 cases (78%) (Figs. 1, 2 and 3). Injuries to the nuchal ligament were

present in 52 (78%), to the interspinous ligament in 43 (64%), to the posterior atlanto-occipital ligament in 10 (15%), to the posterior atlanto-axial ligament (AA ligament) in 36 (54%), to the ligamentum flavum in 8/67 (12%), and to the atlanto-occipital and/or atlanto-axial joint capsule in 15 (22%). Prevertebral retropharyngeal edema was present in 9 (13%) and edema of the supradental space was present in 13 (19%).

Spinal SDH was present in 32 of 67 cases (48%) (Table 1) (Figs. 6, 7 and 8). Of 31 children with whole spine MRIs, 21 (67%) had a spinal SDH; among this subgroup, the cranial extent of the spinal SDH varied from C1 to the T10 vertebral

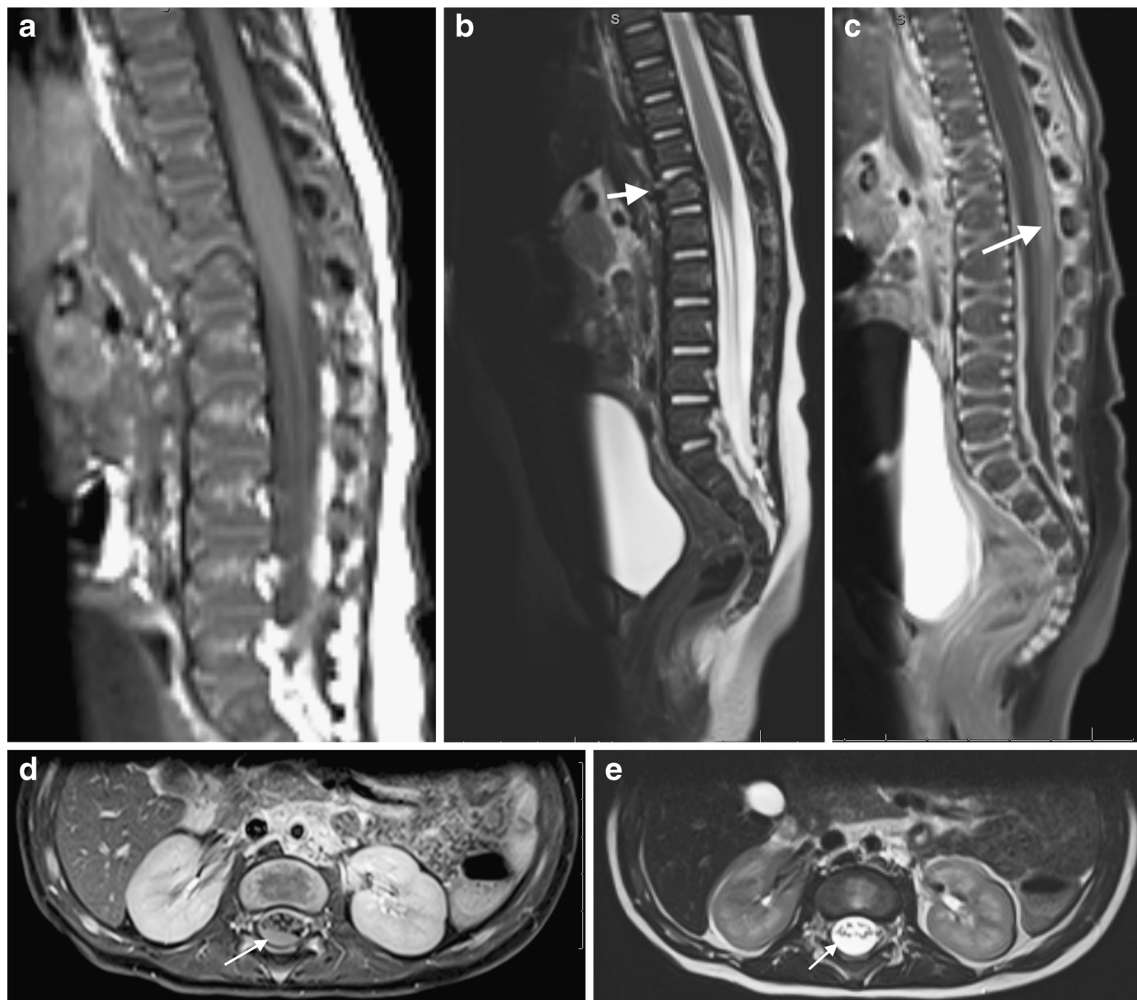


Fig. 8 A 4-month-old girl with multiple classic metaphyseal and rib fractures and a chronic intracranial SDH. Sagittal T1 (**a**) and T2 (**b**) images fail to demonstrate spinal SDH as the hemorrhage is T1 hypointense and T2 hyperintense and is similar to CSF. A three-column fracture of T12 is identified (**b**, *small arrow*). Postcontrast 3-D-VIBE FS (3-D gradient echo volumetric interpolated breath hold examination with

fat suppression) sagittal (**c**) (reconstructed from axial data) and axial (**d**) images, as well as an axial T2 (**e**) image, confirm spinal SDH with displacement of nerve roots anteriorly (*arrows*). The contrast was administered to obtain postcontrast MRV of the brain and subsequently the spine was also scanned postcontrast medium

Table 1 MRI findings of abusive head trauma and accidental trauma cohorts

	Abusive head trauma	Traumatic	Nontraumatic
Total cases	67	46 (43 with brain imaging)	70
Brain subdural	54/67 (81%)	20/43 (46%)	0
Brain ischemia	40/67 (54%)	20/43 (46%)	0
Spinal subdural	32/67(48%)	1/46 (2%)	0
Nuchal ligament injury	52/67 (78%)	20/46 (43%)	1/70 (1%)
Interspinous ligament injury	43/67 (64%)	15/46 (33%)	0
Posterior AO ligament injury	10/67 (15%)	4/46 (9%)	0
Posterior AA ligament injury	36/67 (54%)	17/46 (37%)	0
Capsule ligamentous injury	15/67(22%)	5/46 (11%) all bilateral	0
Ligamentum flavum injury	8/67 (12%)	2/46 (4%)	0
Prevertebral edema	9/67 (13%)	1/46 (2%)	0
Peridental ligamentous injury	13/67 (19%)	6/46 (13%)	0

AO atlanto-occipital, AA atlanto-axial

levels with 18/32 (56%) extending only as far as the thoracolumbar junction; the caudal extent in all cases was the terminus of the thecal sac. In 10 cases the spinal canal SDH was in continuity with posterior fossa SDH. With respect to the adjacent spinal cord, the spinal SDH was predominantly T1 hyperintense in 29/32 (91%), T1 isointense in two cases and hypointense in one case. In two cases, T2 imaging was not available for review. Among the remaining 30 cases, the spinal SDH was T2 hyperintense to spinal cord in 15/30 (50%) and hypointense to spinal cord in 15/30 (50%). All cases of thoracolumbar SDH also had posterior fossa SDH that, although not contiguous with the spinal SDH, had identical signal intensities as the spinal SDH (Fig. 7).

Traumatic cohort

The traumatic cohort included 46 children who underwent spinal neuroimaging because of a clinical concern for spinal injury resulting from trauma, most commonly from motor vehicle injuries. There were an equal number of boys and girls. The median age was 15 months. MRI of the brain was obtained in 43 children (93%). Cervical spine MRIs were obtained in all children in this group. One child (2%) had a bone injury (Fig. 3). Ligamentous abnormalities were identified in 21 cases (46%) (Figs. 3, 4 and 5). Injury to the nuchal ligament was identified in 20 (43%), to the interspinous ligament in 15 (33%) and to the posterior atlanto-axial ligament in 17 (37%). Spinal subdural hemorrhage was present in only 1 child (2%) (Table 1).

Nontraumatic cohort

The cohort being imaged for reasons other than trauma included 74 children. Five children in this group had abnormal soft-tissue signal in the suboccipital region (Table 2). Four of the 74 children were excluded from further analysis because there was an alternate explanation of suboccipital soft-tissue

signal abnormality as described below. Of the analyzed 70 cases in this cohort, the median age was 14 months with 33 (47%) boys and 37 (53%) girls. Among the five cases with suboccipital soft-tissue signal abnormality, the first case was a newborn with T2 hyperintensity in the suboccipital region associated with arthrogryposis and significant contracture deformities as well as intraventricular hemorrhage. The second case had a posterior neck hemangioma. The third case had a venous malformation in the soft-tissue of the neck with hemorrhage. The fourth case had OEIS complex (Omphalocele-exstrophy-imperforate anus-spinal defects) with diffuse body wall edema that included the neck. All four of these cases were excluded from further analysis. The remaining case had a 20-min generalized tonic-clonic seizure associated with fever and septicemia, and a transient post-ictal (Todd's) paralysis of the left arm. The soft-tissue structures in the suboccipital region demonstrated T2 hyperintensity. This child was included in the nontraumatic cohort for further analysis. Apart from this case, there were no other cases with evidence of bone, soft-tissue or ligamentous injuries involving the cervical spine.

Statistical analysis

The findings among the three cohorts are summarized in Table 1. The children in the AHT cohort were younger and more frequently male compared with the traumatic and nontraumatic cohorts. On spinal imaging, bone fractures were found only among the two traumatic cohorts. Ligamentous injuries were significantly more frequent among the two traumatic cohorts compared with the nontraumatic cohort. Ligamentous injuries were more frequent among the AHT cohort (78%) compared with either the accidentally injured (46%) or the nonaccidental (1%) cohorts ($P<0.05$). Among the AHT group, both isolated interspinous ligament injury as well as combinations of interspinous with nuchal and/or atlanto-axial ligamentous injuries were statistically associated with evidence of brain ischemia (Table 2). Individually, atlanto-axial and interspinous ligament injuries were somewhat predictive of brain ischemia (49% and 71% probability with odds ratios of 3 and 8, respectively), the combination of interspinous and posterior atlanto-axial ligament injury was most highly predictive of brain ischemia (88% probability with odds ratio of 24) (Tables 2 and 3). In the traumatic cohort, atlanto-axial ligamentous injury was also significantly correlated with brain ischemia ($P<0.05$).

The overall incidence of spinal SDH among the AHT cohort was 48% compared with only 2% among the accidentally injured group ($P<0.001$). The incidence of spinal SDH in the AHT group was even higher (66%) among those cases in which the thoracolumbar spine was also imaged. The presence of combined nuchal, interspinous and posterior atlanto-axial ligament injury predicted a 60% probability of spinal SDH (Tables 4 and 5).

Table 2 Predicted probabilities for ischemia

Predictor	Predicted probability of ischemia (95% confidence interval)
None	0.237 (0.085, 0.510)
Nuchal ligament	0.179 (0.042, 0.519)
Interspinous ligament	0.710 (0.269, 0.942)
Posterior AA ligament	0.489 (0.144, 0.846)
Nuchal + interspinous ligaments	0.633 (0.374, 0.833)
Nuchal + posterior AA ligaments	0.403 (0.141, 0.736)
Interspinous + posterior AA ligaments	0.883 (0.423, 0.987)
Nuchal + interspinous + posterior AA ligaments	0.842 (0.678, 0.931)

AA atlanto-axial

Table 3 Odds ratios for ischemia

Comparison	Odds ratio (95% confidence interval)	P-value
Nuchal ligament vs. none	0.7 (0.1, 4.9)	0.72
Interspinous ligament vs. none	7.9 (1.7, 36.1)	0.01
Posterior AA ligament vs. none	3.1 (0.8, 11.5)	0.09
(Nuchal + interspinous ligaments) vs. none	5.6 (1.1, 27.4)	0.04
(Nuchal + posterior AA ligaments) vs. none	2.2 (0.3, 13.7)	0.41
(Interspinous + posterior AA ligaments) vs. none	24.4 (3.1, 194.4)	0.003
(Nuchal + interspinous + posterior AA ligaments) vs. none	17.2 (3.6, 81.6)	<0.001

AA atlanto-axial

Discussion

Cervical spinal injuries historically have been considered a rare event in association with both accidental and abusive infant trauma. In one study of 905 infants with head trauma, only 2 (0.002%) had an associated cervical spine injury, both of these resulting from AHT [15]. In another study of 95,964 infants <3 years of age with head trauma, only 1.5% had associated spinal injuries, with a C2 fracture being the most common injury type [16]. Among 8,992 children with AHT reported by Kemp [17] as part of a meta-analysis of studies published prior to 2010, only 25 (0.003%) had an associated spinal injury, 23 of whom were <2 years of age. Twelve of the cases had isolated cervical spine involvement; the median age of this subgroup was 5 months and all presented with impaired consciousness and respiratory distress. In contrast, among 12 children with isolated thoracolumbar spine injuries, with a median age of 13.5 months, all presented with focal neurological deficits or a visible spine deformity. Among 16 children for whom MRI scans were obtained, spinal cord injuries

Table 4 Predicted probabilities for spine subdural

Predictor	Predicted probability of spine subdural (95% confidence interval)
None	0.267 (0.104, 0.533)
Nuchal ligament	0.373 (0.134, 0.696)
Interspinous ligament	0.349 (0.091, 0.742)
Posterior AA ligament	0.385 (0.117, 0.748)
Nuchal + interspinous ligaments	0.467 (0.243, 0.705)
Nuchal + posterior AA ligaments	0.505 (0.216, 0.792)
Interspinous + posterior AA ligaments	0.479 (0.124, 0.857)
Nuchal + interspinous + posterior AA ligaments	0.600 (0.428, 0.751)

AA atlanto-axial

Table 5 Odds ratios for spine subdural

Comparison	Odds ratio (95% confidence interval)	P-value
Nuchal ligament vs. none	1.6 (0.3, 8.7)	0.57
Interspinous ligament vs. none	1.5 (0.4, 5.6)	0.57
Posterior AA ligament vs. none	1.7 (0.5, 5.4)	0.35
(Nuchal + interspinous ligaments) vs. none	2.4 (0.5, 10.7)	0.25
(Nuchal + posterior AA ligaments) vs. none	2.8 (0.5, 15.3)	0.23
(Interspinous + posterior AA ligaments) vs. none	2.5 (0.5, 12.9)	0.27
(Nuchal + interspinous + posterior AA ligaments) vs. none	4.1 (1.0, 16.3)	0.04

AA atlanto-axial

were observed in 12, ligamentous or disc injuries in 4, soft-tissue injuries in 4 and fracture dislocation in 2. Violent shaking was reported in 8 of the 25 children, of whom 4 (50%) had cervical spine injuries [17].

Cervical spine trauma, in general, and ligamentous injuries and spinal SDH, in particular, may be significantly underreported in the literature for a number of reasons. First, infants are difficult to assess clinically; formal strength testing, as one would do for an older child or adult, is clearly not possible in an infant and modest neurological deficits may be missed [18]. Second, infants are much more susceptible than older children or adults to ligamentous injuries that would not necessarily be apparent on radiography or CT [19]. Some of these ligamentous injuries may not result in spinal instability but nonetheless serve as an important marker for trauma. Third, MRI scans – the only imaging modality that can reliably demonstrate ligamentous injuries and soft-tissue hemorrhages – are infrequently performed on infants with trauma unless clinical circumstances warrant such. Fourth, with respect to fatal cases, forensic autopsy protocols in use until recently have separated the brain from the spinal cord by lifting the brain within the calvarium, sectioning the cranial nerves along the ventral brainstem, and, in the final act, cutting the lower medulla from the spinal cord with a pair of curved Mayo scissors at the cervicomedullary junction. Unfortunately, the cervicomedullary junction and upper cervical spinal cord are precisely the regions most vulnerable in infant trauma. Since 2004, forensic autopsy protocols have included techniques for en bloc removal of the brain and cervical spine via a posterior approach that preserves this area [13]. Not surprisingly, more recent publications are increasingly demonstrating traumatic injuries to the cervicomedullary junction, upper spinal cord and upper cervical nerve roots in fatal cases of AHT [3–12]. Brennan and colleagues recently reported that 70% of infant homicide victims have concomitant cervical spinal pathology [4].

A number of anatomical features in infants predispose them to cervical (and particularly upper cervical) soft-tissue and ligamentous neck injuries. These include a large head size relative to the neck, poor muscle tone and head control, horizontally oriented facet joints, poorly developed uncovertebral and intervertebral joints, shorter spinous and transverse processes, and a more cranial fulcrum of movement for the infant neck (C2/3) compared to the older child (C5/6) [17].

The infant neck is stabilized by three sets of important ligaments. Posteriorly, the occipitocervical complex, from occiput to C2, is stabilized by the atlanto-occipital ligament (between occiput and atlas) and the atlanto-axial ligament (between atlas and axis). In addition, the midline is bolstered by the nuchal ligament (ligamentum nuchae), a thick midline ligament that takes origin at the posterior occipital protuberance and extends to the posterior arch of C1 and the spinous processes of C2 to C7 inclusive. The lamellar segment of the nuchal ligament is deepest at C1 and most superficial at C6/7 [20]. The nuchal ligament is in part responsible for maintaining the lordotic alignment of the cervical spine and stabilizing the head during flexion and cervical spine rotation; it is most commonly injured during flexion movements of the neck [20, 21]. Posterolaterally, the capsular ligaments of the occipital condyles and atlanto-axial joints provide further stabilization. Myodural bridges – fibrous bridges that connect the suboccipital musculature including, in particular, the rectus capitis posterior minimi, and the nuchal ligament with the cervical dura and atlanto-occipital and atlanto-axial ligamentous complex – may also be involved in cervical spine stabilization [22–30].

Immediately anterior to the spinal canal and spinal cord the occiput-atlas-axis complex is supported by a strong ligamentous complex that includes the tectorial membrane, posterior longitudinal ligament, cruciform ligament, apical and alar ligaments of the dens, and the accessory atlanto-axial ligaments. Finally, the anterior longitudinal ligament connects the axis and atlas along the anterior vertebral surfaces of these two vertebrae; the anterior longitudinal ligament becomes confluent with the anterior atlanto-occipital membrane to connect the anterior arch of the atlas to the anterior clivus.

Our study found that infants with both AHT and accidental injuries most commonly sustained predominantly posterior ligamentous complex injuries – nuchal, atlanto-occipital and atlanto-axial ligaments – without anterior ligamentous injuries and without bony injury. This favors a primary flexion mechanism of neck injury. The higher frequency of ligamentous injury among the AHT cohort compared with the accidental trauma cohort suggests both that infants with AHT are subjected to more significant neck forces than those with accidental neck injuries (who more frequently sustain impact injuries) and these neck injuries may represent the result of violent, forceful shaking (with or without impact).

There was also a higher incidence of spinal SDH among the AHT cohort compared with the accidental trauma cohort. One possibility is that repetitive violent shaking (rather than an isolated flexion or extension movements as might be seen in accidental trauma) results in more frequent and severe cervical ligamentous injuries. We also speculate that myodural bridges may serve as a previously unrecognized conduit to transmit injury forces from the cervical musculature and ligaments to the underlying dura and spinal cord. Traction on the myodural bridges, by pulling the dura away from the underlying arachnoid, might open up the cervical spinal subdural space and allow posterior fossa subdural blood to migrate caudally into the spinal subdural space. Traction on intradural nerve roots and dentate ligaments could also transmit these forces to the spinal cord and brainstem. The role of the myodural bridges in spinal cord injuries requires further study.

We have previously reported on spinal SDH among infants with AHT in which more than 60% of the spinal SDH were thoracolumbar in origin and without any associated thoracolumbar ligamentous injury. This led us to postulate that spinal SDH was the result of caudal migration of blood from the posterior fossa SDH to the most dependent region of the spine rather than the consequence of direct spinal trauma [14]. This proposal is strengthened by the fact there is no venous plexus in the spinal subdural space [31]. The results of the present study support this conclusion, at least for the thoracolumbar SDH. All of the infants with spinal SDH had a coexisting posterior fossa SDH of similar MRI intensity. Among those with thoracolumbar SDH there were no thoracolumbar bone or ligamentous injuries found at the level of the SDH. This leads us to conclude that the posterior fossa SDH likely extends caudally into a gravity-dependent site. Among the children with cervical SDH, the posterior fossa and cervical SDH were contiguous leading to a similar conclusion although the presence, in some, of adjacent cervical ligamentous injuries does not allow us to exclude direct cervical trauma as another potential mechanism. In fact, our study found that although injury to individual cervical spine ligaments was not predictive of spinal SDH, the combination of nuchal, interspinous and atlanto-axial ligamentous damage was significantly correlated with spinal SDH.

One of the most important findings from this study is that, among both the AHT and accidentally injured cohorts, ligamentous injuries to the occipitocervical complex were significantly associated with cranial MRI findings of brain hypoxic-ischemic injury. These findings are consistent with the conclusions of others that the injuries to the brain in AHT are primarily hypoxic-ischemic in origin [3] and the result of disordered breathing or apnea [7]. Alternatively, this association could simply reflect the presence of greater overall injuries among the group having both cervical spine and hypoxic-ischemic brain injuries. An intriguing finding of this study is that direct imaging evidence of spinal cord injury was lacking,

in contrast to the findings of other studies [4]. One possibility is that these children have sustained a reversible neurapraxic type of injury that is not visible using standard MRI sequences. We hypothesize that injuries to the posterior occipitocervical ligaments may serve as a proxy to spinal cord and/or brainstem injury without visible evidence of spinal cord damage on routine MRI. Diffusion tensor imaging may demonstrate lesser degrees of axonal injury to a better degree and deserves further study.

Another interesting finding is that the only child among the nontraumatic cohort with cervical spine abnormalities similar to those in the traumatic cohorts had a 20-min tonic-clonic seizure. There remains the possibility that during the prolonged clonic phase of this child's seizure, there could have been violent shaking movements that caused traumatic spinal soft-tissue injury. We are undertaking a retrospective study of MRI among children with extensive or prolonged seizures to evaluate this possibility.

Our study results support previous findings and recommendations regarding spinal imaging in children with suspected AHT, with more than three-quarters of these children having visible MRI abnormalities that would be missed if spinal imaging is omitted [14]. Some have argued that the costs of additional imaging studies are prohibitive and that the abnormalities are not of practical significance as they do not change treatment plans. However, we have encountered two symptomatic spinal SDH, one found on imaging performed for evaluation of priapism and another for evaluation of delayed paraparesis. Although these did not lead to surgical treatment of the SDH, they did lead to greater vigilance for evidence of progression in these two children. Moreover, like skeletal surveys, most of which demonstrate bony injuries that do not require treatment, the identification of cervical ligamentous injuries is of extreme medicolegal importance in supporting trauma as the underlying etiology for the child's injuries. The finding of a much higher frequency of upper cervical ligamentous injury among the AHT cohort compared with the symptomatic accidentally injured cohort supports repetitive, violent infant shaking as a potentially significant injury mechanism in the pathogenesis of AHT. Finally, we want to emphasize that although cervical spine ligamentous injuries are common among the AHT cohort, this should not be interpreted to mean, as some biomechanical engineers have suggested, that the absence of spine or spinal cord injury excludes AHT as a possibility, or shaking as a potential injury mechanism.

There are a number of limitations to this study. First, it is a retrospective study with a small sample size. Moreover, the traumatic cohort was selected on the basis of clinical concern for cervical spine injuries although it is likely that an accidental trauma cohort that included all children with accidental trauma would have made the intergroup differences even greater. Second, although these findings were almost

universally absent in the nontraumatic cohort, we have no pathological confirmation that the findings in this study are directly trauma related. However, the fact that these findings were present among both the AHT and accidental trauma cohorts but in only one of the children from the nontraumatic cohort and that in the AHT groups the injuries were often associated with concomitant traumatic intracranial and extracranial injuries strongly supports the traumatic nature of the spinal injuries. Third, the median age of AHT cohort was 4 months as compared to 15 and 14 months in the traumatic and nontraumatic cohorts, respectively. This may reflect either nature of injury at different age groups vs. presentation at our institute. Finally, it is possible that we have missed some injuries that were too subtle to identify. In particular, spinal SDH was predominantly T1 hyperintense, but we may have missed other isointense or hypointense SDH (Fig. 5). This raises the possibility that the true incidence of spinal subdural hemorrhage may be higher in AHT. Nonetheless, these limitations do not significantly change the conclusions of the study.

Conclusion

Cervical spinal ligamentous injuries are much more common than bony injuries in AHT, therefore radiographs and CT will be insufficient. We describe a complex of injuries that frequently involves the nuchal, interspinous and posterior occipital-atlantal-axial ligaments on MRI in cases of AHT. That this signature pattern among the AHT cohort more closely mirrors the accidental trauma cohort than the nontraumatic cohort supports that these findings are traumatic in nature. Although our study did not directly identify spinal cord injury, the strong correlation between ligamentous injury to the occipitocervical junction and MRI findings of hypoxic-ischemic brain injury supports the conclusion that transient upper cervical spinal cord and/or upper cervical root injuries result in transient apnea or disordered breathing that leads ultimately to hypoxic-ischemic encephalopathy. We recommend imaging the entire spine in AHT to properly identify these associated injuries. STIR sequence was found to be most useful for detecting soft-tissue injury and sagittal T1 was found most useful for detecting spinal subdural. Axial T1 was useful for detecting small spinal subdural hematoma. Further study of spinal and brainstem injuries using diffusion tensor imaging, detailed studies of the suboccipital musculature and ligaments during autopsy, and the role of myodural bridges in producing dural traction may help us to further understand the pathophysiology of brain and spine injury in AHT.

Conflicts of interest None

References

- Bradford R, Choudhary AK, Dias MS (2013) Serial neuroimaging in infants with abusive head trauma: timing abusive injuries. *J Neurosurg Pediatr* 12:110–119
- Dias MS, Backstrom J, Falk M (1998) Serial radiography in the infant shaken impact syndrome. *Pediatr Neurosurg* 29:77–85
- Geddes JF, Hackshaw AK, Vowles GH et al (2001) Neuropathology of inflicted head injury in children. I. Patterns of brain damage. *Brain* 124:1290–1298
- Brennan LK, Rubin D, Christian CW et al (2009) Neck injuries in young pediatric homicide victims. *J Neurosurg Pediatr* 3:232–239
- Geddes JF, Vowles GH, Hackshaw AK et al (2001) Neuropathology of inflicted head injury in children. II. Microscopic brain injury in infants. *Brain* 124:1299–1306
- Hadley MN, Sonntag VK, Rekatte HL et al (1989) The infant whiplash-shake injury syndrome: a clinical and pathological study. *Neurosurgery* 24:536–540
- Johnson DL, Boal D, Baule R (1995) Role of apnea in nonaccidental head injury. *Pediatr Neurosurg* 23:305–310
- Kemp AM, Stoodley N, Copley C et al (2003) Apnoea and brain swelling in non-accidental head injury. *Arch Dis Child* 88:472–476
- Shannon P, Becker L (2001) Mechanisms of brain injury in infantile child abuse. *Lancet* 358:686–687
- Shannon P, Smith CR, Deck J et al (1998) Axonal injury and the neuropathology of shaken baby syndrome. *Acta Neuropathol* 95:625–631
- Matshes EW, Evans RM, Pinckard JK et al (2011) Shaken infants dies of neck trauma, not of brain trauma. *Acad For Path* 1:82–91
- Bandak FA (2005) Shaken baby syndrome: a biomechanics analysis of injury mechanisms. *Forensic Sci Int* 151:71–79
- Judkins AR, Hood IG, Mirchandani HG et al (2004) Technical communication: rationale and technique for examination of nervous system in suspected infant victims of abuse. *Am J Forensic Med Pathol* 25:29–32
- Choudhary AK, Bradford RK, Dias MS et al (2012) Spinal subdural hemorrhage in abusive head trauma: a retrospective study. *Radiology* 262:216–223
- Katz JS, Oluigbo CO, Wilkinson CC et al (2010) Prevalence of cervical spine injury in infants with head trauma. *J Neurosurg Pediatr* 5:470–473
- Polk-Williams A, Carr BG, Blinman TA et al (2008) Cervical spine injury in young children: a National Trauma Data Bank review. *J Pediatr Surg* 43:1718–1721
- Kemp AM, Joshi AH, Mann M et al (2010) What are the clinical and radiological characteristics of spinal injuries from physical abuse: a systematic review. *Arch Dis Child* 95:355–360
- Mulcahey MJ, Gaughan J, Betz RR et al (2007) The International Standards for Neurological Classification of Spinal Cord Injury: reliability of data when applied to children and youths. *Spinal Cord* 45:452–459
- Anderson RC, Kan P, Vanaman M et al (2010) Utility of a cervical spine clearance protocol after trauma in children between 0 and 3 years of age. *J Neurosurg Pediatr* 5:292–296
- Kadri PA, Al-Mefty O (2007) Anatomy of the nuchal ligament and its surgical applications. *Neurosurgery* 61:301–304, discussion 4
- Takeshita K, Peterson ET, Bylski-Austrow D et al (2004) The nuchal ligament restrains cervical spine flexion. *Spine (Phila Pa 1976)* 29:E388–E393
- Kahkeshani K, Ward PJ (2012) Connection between the spinal dura mater and suboccipital musculature: evidence for the myodural bridge and a route for its dissection—a review. *Clin Anat* 25:415–422
- Nash L, Nicholson H, Lee AS et al (2005) Configuration of the connective tissue in the posterior atlanto-occipital interspace: a sheet plastination and confocal microscopy study. *Spine (Phila Pa 1976)* 30:1359–1366
- Dean NA, Mitchell BS (2002) Anatomic relation between the nuchal ligament (ligamentum nuchae) and the spinal dura mater in the craniocervical region. *Clin Anat* 15:182–185
- Zumpano MP, Hartwell S, Jagos CS (2006) Soft tissue connection between rectus capitus posterior minor and the posterior atlanto-occipital membrane: a cadaveric study. *Clin Anat* 19:522–527
- Hack GD, Koritzer RT, Robinson WL et al (1995) Anatomic relation between the rectus capitis posterior minor muscle and the dura mater. *Spine (Phila Pa 1976)* 20:2484–2486
- Johnson GM, Zhang M, Jones DG (2000) The fine connective tissue architecture of the human ligamentum nuchae. *Spine (Phila Pa 1976)* 25:5–9
- Humphreys BK, Kenin S, Hubbard BB et al (2003) Investigation of connective tissue attachments to the cervical spinal dura mater. *Clin Anat* 16:152–159
- Mitchell BS, Humphreys BK, O'Sullivan E (1998) Attachments of the ligamentum nuchae to cervical posterior spinal dura and the lateral part of the occipital bone. *J Manip Physiol Ther* 21:145–148
- Pontell ME, Scali F, Marshall E et al (2013) The obliquus capitis inferior myodural bridge. *Clin Anat* 26:450–454
- Nicholas DS, Weller RO (1988) The fine anatomy of the human spinal meninges. A light and scanning electron microscopy study. *J Neurosurg* 69:276–282