

Spinal injuries in abusive head trauma: patterns and recommendations

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Abstract A growing body of scientific evidence suggests that there is an association between occult spinal injury and abusive head trauma (previously known as shaken baby syndrome). Consideration needs to be given to the nature of these injuries, the possible causal mechanisms and what investigations should be undertaken to delineate the full extent of spinal involvement in infants with suspected abusive head trauma. This association has the potential to influence our understanding of the biomechanics and subsequent neuropathology associated with abusive head trauma.

Keywords Abusive head trauma · Spine injury · Non-accidental injury · Child

Introduction

Patterns of intracranial injury in abusive head trauma have been well described and include extra-axial hemorrhage in the majority, hypoxic ischemic encephalopathy, cerebral edema in a significant proportion of cases [1] and focal parenchymal injury in a small number of cases. The overriding proposed mechanism of injury is shaking with or without impact. When acceleration-deceleration forces are applied to the head during shaking, it is reasonable to consider that the head would pivot around points in the cervical spine that may also be injured. It

is also plausible that forcible shaking may elicit injurious hyperextension or flexion forces to other regions of the spine, depending upon how and where the child is held during the incident. However, until recently, there has been less attention paid to the likelihood, significance or nature of spinal injury in cases of abusive head trauma.

The evidence base for spinal injury in physical abuse

In 2010, Kemp et al. [2] published a systematic review that described spinal injuries in physical child abuse. The methodology involved a thorough literature search of 20 electronic databases, websites, references and bibliographies (1950–2010) using selected keywords. The review included primary studies of inflicted spinal injury in children <18 years, alive at presentation, with a high surety of diagnosis of abuse and sufficient detail to analyze.

The systematic review aimed to identify high-quality comparative studies; however, the extent of the published literature was restricted to 19 studies that described a series of highly selected case reports of 25 children with abusive spinal injury. The reports fell into two groups. Younger infants, with a median age of 5 months, had cervical injury. The injuries described included musculoskeletal injury, with variable involvement of the spinal cord, and spinal cord injury without musculoskeletal involvement. There was an association between cervical spinal injury and abusive head trauma in 6 of the 12 reported cases. Clinical signs of cervical spinal injury were masked by respiratory symptoms and impaired levels of consciousness, attributed to the association with abusive head trauma. Twelve older children, with a median age of 13.5 months, presented with thoracolumbar lesions. Most lesions were fracture dislocations and presented with lumbar kyphosis, thoracolumbar swelling or focal neurology. One child had widespread subdural hemorrhages throughout the

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thoracolumbar spine, extending to the cervical region with associated abusive head trauma [3]. The systematic review was updated annually and by 2014, the number and quality of studies had improved. Eight further studies were identified; three comparative case series [4–6], three cross-sectional [7–9] and two case series [10, 11]; five of these recent studies [4–6, 10, 11] focused on spinal injury in association with abusive head trauma.

Spinal fractures in physical abuse

Spinal fractures are relatively uncommon in physical abuse, yet they have been reported, and when present are associated with a risk of abusive head trauma. The estimated prevalence of spinal fractures in children investigated for suspected child abuse varies from 0.3% to 2.7% (Table 1). In children with a positive skeletal survey the prevalence of spinal fractures was 0.8–9.7%.

Barber et al. [11] recorded abusive head trauma in 71% (10/14) of the children with abusive spinal fractures. Barber et al. [11] calculated a significantly greater risk for AHT in children with spinal fractures than those without (odds ratio 5; 95% confidence interval 1.6–16.4). Four other studies identified isolated [4, 5, 10] or no cases [12] of abusive head trauma with associated spinal fractures.

Spinal fractures in the ten cases of abusive head trauma described in the Barber et al. study [11] were predominantly vertebral body compression fractures to the thoracic or lumbar vertebrae (between T3–L2: 12 of 17 fractures were below T7); one child had a fracture dislocation. Five of the ten cases had multiple spinal fractures. While Kleinman et al. [8] (from the same center utilizing data from a different time period) did not present data regarding the association of abusive head trauma with spinal fractures, the study shows a similar fracture type and distribution of abusive thoracolumbar spinal fractures.

Spinal subdural hemorrhage and abusive head trauma

In 2009, Koumellis et al. [10] described 18 infants with abusive head trauma who had a spinal MRI scan, eight of whom (44%) had spinal subdural hemorrhages, all of which were clinically occult; six cases were described as large spinal subdural hemorrhages extending from the sacral region to mid-thoracic or cervical region, and two were small. All eight were associated with intracranial subdural hemorrhages in the supratentorial and infratentorial compartments. The signal characteristics were the same as the posterior fossa subdural hemorrhage while supratentorial subdural hemorrhages were of mixed intensity. Two spinal subdural hemorrhages were cervical and continuous with the posterior fossa subdural hemorrhage. In one case, there was a small epidural hematoma in association with thoracic vertebral fractures.

A larger comparative study analyzed spinal imaging (CT, MRI or both) of 67 children younger than 2 years old who had cranial imaging for abusive head trauma, and 70 with accidental head trauma investigated for clinical concern regarding abdominal or spinal injury [4]. While the extent of the spine exposed to imaging and the imaging modality varied over the timescale of the project, the study identified spinal subdural hemorrhages in 46% (31/67) of abusive head trauma cases in comparison with 1/70 of the accidental cases. This child had severe posterior fossa injuries with depressed fracture and cerebellar contusions. Of the 70 accidental cases, 22 had small intracranial subdural hemorrhages, one of whom had the spinal subdural hemorrhage.

Of the 29 children with abusive head trauma who had cervical spinal imaging only, seven had cervical spinal subdural hemorrhages. The imaging did not extend to the rest of the spine and the extent of spinal subdural hemorrhage may have been underestimated, especially in the context that 63% (24) of the 38 who had thoracolumbar imaging had subdural hemorrhage (21 thoracolumbar only, 3 cervical and thoracolumbar). All children with spinal subdural hemorrhage had intracranial supratentorial and posterior fossa subdural hemorrhage. All except two cases were asymptomatic (symptoms included priapism and back pain). Spinal subdural hemorrhages were of the same attenuation as intracranial subdural hemorrhages and were mostly located in the posterior dura, although some were circumferential. In addition to the one child with lumbar spinal fracture, one had spinal cord injury and subdural hemorrhage of the spinal canal.

Spinal ligamentous abnormalities

Two recent studies have explored the relationship between cervical spinal ligamentous injury and abusive head trauma [5, 6]. Choudhary et al. [5] compared spinal ligamentous and soft-tissue injury among three groups of children younger than 4 years old. Cervical ligamentous injuries were identified in a significantly greater proportion ($P < 0.05$) of abusive head trauma cases (78% of 67 children) than children with accidental trauma who had MRI for suspected spinal injury (46% of 46) or in children who had spinal MRI for other nontraumatic clinical concerns (1% of 70). The ligamentous injuries were predominantly to the cervical spinal posterior ligamentous complex (the nuchal, atlanto-occipital and atlanto-axial ligaments) consistent with hyperflexion injury. The higher frequency of ligamentous injury among the abusive head trauma cases may support the fact that infants are subject to “more significant neck forces” from vigorous shaking as compared to single impact or flexion-hyperextension (whiplash) injury in children with accidental neck trauma. A significant and important association was identified between occipitocervical ligamentous injury and brain ischemia in the abusive head trauma and the accidental traumatic control group.

Table 1 Spinal involvement in physical child abuse and abusive head trauma

	Investigation (number of children)	% children with spinal fractures when physical abuse suspected	% of children with spinal fracture when SS positive for any fracture	% children with spinal fracture in AHT	% children with AHT and spinal injury on neuroimaging or postmortem
Radiographical studies					
Barber et al. [11] <4 years	SS (751)	1.9% (14/751) had 22 spinal fractures	9.7% (14/145)	71% (10/14) with spinal fracture had AHT	
Karmazyn et al. [9] <2 years	SS (930)	0.3% (3/930) had 5 fractures NB initially 6 children had suspected fractures on SS, later excluded on CT/MR	0.8% (3/371)	Not stated	
Kleinman et al. [8] <2 years	SS (365)	2.7% (10/365) had 25 fractures	4.4% (10/225)	Not stated	
Lindberg et al. [7] <5 years	SS (2,049)	0.8% (22/2,049) had (at least) 38 spinal fractures	3.4% (16/471)	Not stated	
<2 years		0.8% (14/1750) had at least 21 spinal fractures	2.4% (10/417)		
Neuroimaging studies					
Choudhary et al. [4] <2 years	CT/MRI 67 AHT 70 Controls			1.5% (1/67) had lumbar spinal fracture	Cervical SDH in 24% (7/29) who had cervical imaging only 63% (24/38) of those who had thoracolumbar imaging had SDH (21 thoracolumbar SDH only, 3 cervical and thoracolumbar SDH)
Choudhary et al. [5] <48 months	MRI: 67 AHT, 46 accidental 70 nontraumatic conditions			4/67 (6%) bone trauma AHT 1/46 (2%) in accidental trauma	Cervical spinal ligamentous injuries were present in 78% (52/67) of the AHT group, 46% (21/46) of the accidental trauma group and 1% (1/70) of the nontraumatic group. Spinal SDH 48% (32/67) AHT 2% (1/46) accidental trauma but none in traumatic group
Kadom et al. [6] <3 years	MRI 74 suspected AHT				Cervical spinal injury in 27/74 (36%) investigated for suspected AHT (outcome: 26 accidental/38 likely AHT 10 unconfirmed)
Koumellis et al. [10] <1 year	MRI (18)			11.1% (2/18) thoracic fracture	44% (8/18) AHT cases with spinal MRI had spinal SDH 2/13 craniocervical EDH
Soto Ares et al. [13] <10 months	MRI (13)				
Postmortem findings					
Brennan et al. [14] <2 years	PM (41)			0/41	71% (29/41) cervical SCI, 24 had meningeal hemorrhages, 16 nerve root avulsion/dorsal root ganglion hemorrhage

Table 1 (continued)

	Investigation (number of children)	% children with spinal fractures when physical abuse suspected	% of children with spinal fracture when SS positive for any fracture	% children with spinal fracture in AHT	% children with AHT and spinal injury on neuroimaging or postmortem
Feldman et al. [12] <3 years	MRI (12) PM (5)			0/12	16/40 (40%) brainstem injuries (1 case could not be examined)
Geddes et al. [15, 16] <10 months	PM: (37) (Beta Amyloid precursor protein)				0/12 on MRI but 4/5 of same cases at PM had SDH/SAH 63.6% (11/37) at PM had localized axonal injury to the craniocervical junction or the cervical cord At PM 5/6 had cervicomedullary EDH or SDH 4 had proximal SCI
Hadley et al. [17] <15 months	CT/radiology (13) PM (6/13)				1 atlanto-occipital ligament disruption 1 SCI (contusion and laceration)
Johnson et al. [18] <2 years	PM (4)				1 cervical and thoracic SDH
Kemp et al. [19] <2 years	PM (14)				28.6% (4/14) postmortem cases cervical spine, extra-axial bleeding and brainstem injury
Shannon et al. [20] <28 months	PM: (14) (Beta amyloid precursor protein)				Axonal damage in cervical cord and nerve roots in 7/11 AHT cases (where examined)

AHT abusive head trauma, CT computerized tomography, EDH epidural hemorrhage, MRI magnetic resonance imaging, PM postmortem, SAH subarachnoid hemorrhage, SCI spinal cord injury, SDH subdural hemorrhage, SS skeletal survey

Kadom et al. [6] identified cervical spinal soft-tissue injuries in 36% of 74 children who were investigated for suspected abusive head trauma with cranial and cervical spinal MRI; most of these were ligamentous injuries. The authors identified significant associations between both posterior fossa hemorrhage and diffuse brain hypoxia with abusive head trauma. While cervical spinal injury was not significantly associated with abusive head trauma, 83% (10/12) of cases with both cervical injuries and diffuse hypoxic brain injury were classified as abusive head trauma.

Postmortem studies

While the results from systematic reviews described above did not include postmortem studies, there are neuropathology studies that lend further detailed evidence to the coexistence of spinal injury with abusive head trauma.

As early as 1988, Hadley et al. [17] reported spinal findings at postmortem in five of six children who died of abusive head trauma; had cervicomedullary junction injury consisting of extradural hemorrhage and/or subdural hemorrhage, and four had proximal spinal cord contusions. All had cerebral subdural hemorrhages, contusions, swelling and herniation. In 1997, Feldman et al. [12] described cervical spinal extra-axial hemorrhages at postmortem in four of five children who died from abusive head trauma. One child had a, thin upper cervical subdural hemorrhage continuous with a cranial subdural hemorrhage. Three had cervical subarachnoid bleeding associated with cranial or extensive spinal subarachnoid hemorrhage. Extra-axial bleeds were associated with both presumed impact and non-impact head injury. Only one postmortem case had hypoxic neuronal changes in the cervical spinal cord at a microscopic level. None of these subdural hemorrhages was confirmed on antemortem MRI. Further cases of cervical spinal cord damage and extra-axial bleeding are reported [18, 19].

Brennan et al. [14] undertook a full neuropathological postmortem examination of the neck in 41 fatal cases of abusive head trauma in children younger than 2 years of age. The authors concluded that cervical spinal cord injury was common but not a universal finding. Twenty-nine (71%) had primary cervical cord injuries, 21 of whom had parenchymal injuries (contusions, lacerations or transections), 24 had meningeal hemorrhages and 16 had nerve root avulsion/dorsal root ganglion hemorrhage. Sixteen (40%) of the cases had brainstem injuries. Ligamentous or muscular neck injuries were uncommon and were present in a minority of these cases, 6/29 children (21%). No child had a spinal fracture. There was no significant association between primary cervical spinal cord injury hypoxia-ischemia, infarction or herniation but there was with cerebral edema ($P=0.036$). There was a trend toward cervical spinal injury in children with no apparent

evidence of blunt trauma to the head ($P=0.088$). All six children with abusive head trauma and no evidence of an impact to the head, had primary cervical spinal cord injury vs. 23/35 (65.7%) of those with impact injuries. The authors were unable to analyze the relationship between pathological findings with antemortem symptomatology.

Rutty et al. [21] describe two cases of abusive head trauma with cervical spinal nerve root injury and spinal subdural hemorrhage throughout the spinal cord, but no extradural hemorrhage. These cases are presented alongside findings of small fresh spinal epidural hemorrhage, predominantly in the cervical region, in six children (<2 years of age) where there was no history of trauma. These extradural findings were deemed to be artifactual. The authors caution against the over-interpretation of spinal epidural hemorrhage as supportive evidence for a shaking mechanism in abusive head trauma in the absence of supportive evidence of antemortem trauma or associated features of intracranial injury.

Two studies used the expression of β -amyloid precursor protein utilizing the immunoperoxidase technique to identify traumatic axonal damage. Geddes et al. [15, 16] presented data on 37 children 9 months of age or younger who died from abusive head trauma and 14 controls. In 11/37 cases of abusive head trauma, there were cervical epidural hemorrhages and focal traumatic axonal damage to the corticospinal tracts in the brainstem and the spinal nerve roots, but not in the controls. The authors proposed a strong association between prolonged apnea, hypoxic ischemic brain damage and concluded that the craniocervical junction was vulnerable to damage in children with abusive head trauma. Similar findings had been reported previously by Shannon et al. [20], who identified widespread hypoxic ischemic cerebral damage and axonal injury in the nerve roots and the high cervical spinal cord in 7/11 children with abusive head trauma. This association was not evident in the control group of children with hypoxic ischemic encephalopathy. Both studies confirm the overriding finding of hypoxic ischemic brain damage rather than diffuse traumatic axonal injury in the brain itself.

While postmortem cases represent the most serious cases of abusive head trauma and findings cannot automatically be extrapolated to nonfatal cases, the level of detail provided from histopathology gives rise to hypotheses around mechanisms of injury.

Proposed mechanisms of spinal injury in abusive head trauma

The emerging evidence surrounding the association of spinal injury in a proportion of cases of abusive head trauma has the potential to inform our understanding of the mechanisms that contribute to the neuropathology of the condition.

Much of the published literature concentrates on cervical spinal injury. There are anatomical characteristics of the infant neck that increase the likelihood of cervical flexion, extension and rotational injury in association with abusive head trauma. These include neck instability as a result of low muscle tone, horizontally oriented facet joints, incompletely formed joints and vertebral structures, laxity of spinous ligaments and the shape of the infant vertebral body [22]. The increased proportion of head to body ratio in an infant makes the infant more prone to cervical injury. While an association between cervical spinal fractures and abusive head trauma has been shown, the prevalence of spinal skeletal fractures in abusive head trauma appears to be relatively uncommon (Table 1). It is possible that the increased flexibility of the infant spinal column is greater than that of the spinal cord itself and while this may exert a protective effect on the skeletal structure during flexion-hyperextension injury, it may increase the vulnerability of the spinal cord and extra-axial structures. Barnes et al. [23] propose that during infant cervical trauma, the vertebrae have the capacity to sublux and impact the spinal cord and return to their position. In support of this theory, several studies [4, 5, 12, 14–17, 20, 21] have identified injury to the cervical spinal cord, ligaments and extra-axial structures with minimal spinal skeletal injury in abusive head trauma. Shannon et al. [20] and Geddes et al. [15, 16] propose that the axonal damage in the corticospinal tracts supports the hypothesis of stretching injury to the spinal cord during flexion-hyperextension trauma to the neck.

While the vulnerability of the cervical spine to traumatic injury in flexion, extension and rotational injury to the neck can be conceptualized, the links to intracranial neuropathology are less established. It has been hypothesized that cervical spinal injury impairs respiratory centers and elicits apnea and, thus, cerebral hypoxia. Choudhary et al. [5] showed that posterior cervical ligamentous injury was strongly predictive of brain ischemia (positive predictive value: 84–88%) and proposes that this further supports the theory that cervical trauma contributes to apnea, breathing disruption and brain ischemia. The findings of Kadom et al. [6] would also support this hypothesis. While studies are increasingly showing an unequivocal coexistence of abusive head trauma, hypoxic ischemia and apnea [16–19] or cervical spinal injury [5, 6], hypoxic brain injury is present both with and without evidence of cervical spinal cord damage [16]. In a Geddes et al. study [16], 25 of the 28 cases with documented apnea had “global hypoxic ischemia” but only eight of these cases had evidence of neck injury. Brennan et al. [14] identified cerebral hypoxia in 55% (16/29) of cases with abusive head trauma and spinal cord injury and 58% (7/12) cases without spinal cord injury. Choudhary et al. [5] and Kadom et al. [6] also describe cases of abusive head trauma with hypoxic brain injury but no evidence of cervical spinal injury. While transient spinal cord injury to the upper cervical spinal cord that cannot be

visualized on MRI or at postmortem may contribute to apnea and hypoxic ischemia during shaking damage, it is unlikely to be the only pathogenesis. Hypoxic ischemic injury to the brain may also have its route in secondary brain damage from compromised cerebral perfusion as part of the cascade reaction [24].

While most publications concentrate on the cervical spinal cord, recent attention has turned to the rest of the spinal cord, with the more widespread use of more sensitive MRI technology. Extensive thoracolumbar spinal subdural hemorrhages have now been described by a number of authors in fatal [21] and nonfatal cases [4, 5, 10]. Two possible mechanisms have been proposed, namely tracking of intracranial subdural hemorrhage into the spinal compartment [4, 10] or injury to the vessels around the spinal cord.

Tracking of blood from intracranial subdural bleeding has some support from the fact that the spinal dura is continuous with the intracranial cerebral dura and a proportion of the reported spinal subdural hemorrhages have been shown to be continuous with cranial subdural hemorrhages in the posterior fossa [5, 10]. In addition, the spinal subdural hemorrhages reported had a similar attenuation to the intracranial subdural hemorrhages [4, 5, 10]. Migration of intracranial traumatic subdural hemorrhage to the lumbar spine has been described in young adults [25, 26]. The pooling of blood in the thoracolumbar region is explained by the fact that this is the most dependent point in a recumbent infant due to the spinal curvature at this point. Having identified a significantly higher proportion of spinal subdural hemorrhages in infants with abusive head trauma. Choudhary et al. [5] propose the following theory: During vigorous shaking, myodural bridges may transmit forces from the cervical muscle and ligaments to the dura and pull it away from the arachnoid, which may open up the posterior subdural space and allow migration of blood from a posterior cranial subdural hemorrhage into the spinal subdural space. The authors offer the following to support this theory: In children with cervical subdural hemorrhage, the posterior fossa and cervical subdural hemorrhages were continuous and all children with thoracolumbar subdural hemorrhages had a coexisting posterior fossa subdural hemorrhage of similar MRI intensity and no coexisting thoracolumbar ligamentous or bony injury. The authors further identified that injury to the cervical spinal posterior ligamentous complex was significantly associated with spinal subdural hemorrhage.

When considering the second theory that the bleeding arises from coexistent localized spinal trauma and bleeding within the dura or subdural space, consideration must be given to the vascular supply to the spinal dura and its structure. There are a few studies that describe the histological structure of the infant intracranial dura [27–29]. The spinal dura is similar but less vascular and has little innervation. The dura is a fibrous structure that ensheathes the spinal cord and extends beyond the dorsal nerve routes. The dura is composed

of an outermost loosely arranged fibroelastic layer, a middle fibrous portion that is vascularized and an innermost cellular layer of dural border cells [30]. There are no subdural bridging veins in the spine. Arteries and veins travel from the extradural space to the intradural space via the nerve root sheaths [3]. In hyperflexion of the spine, it is proposed that the spinal dura will be displaced [30]. Gruber and Rozzelle [3] propose that the thoracic spine is a relatively rigid structure and that in shaking, the thoracolumbar junction is susceptible to flexion-extension injury if the infant is grasped around the thorax. The authors hypothesize that hyperflexion exerts sufficient stress to rupture blood vessels within the spinal nerve sheaths along the spinal nerves and nerve roots causing subdural bleeding. In the case study described at neurosurgery [3], there was evidence of active bleeding at the site of the thoracolumbar subdural hemorrhage from a radicular vein with an associated breach of the arachnoid membrane.

Further studies are needed to explore the proposed mechanism of injury. The studies reviewed here explored neuroimaging, predominantly conducted within 3 days of presentation to hospital [4, 10], and thus the temporal relationship of spinal subdural hemorrhage to intracranial injury is unknown. Further animal studies have the potential to use immunohistochemistry techniques to identify the points of maximal stress during shaking as has been done by Finnie et al. [31]. The authors analyzed neonatal lambs that had been shaken and compared the immediate early gene *c-Fos* throughout the brain and spinal cord. *c-Fos* is used to identify cells that become activated and phenotypically altered in response external stimuli such as trauma. The greatest *c-Fos* expression was in meningeothelial cells of the cranial cervical spinal cord and, to a lesser degree, in hemispheric, cerebellar and brainstem meninges in the lambs that were shaken but not in the controls. The authors postulated that the “most prominent *c-Fos* expression was in meningo-epithelial cells of the pia-arachnoid in the rostral cervical spinal cord at the craniocervical junction, the site of maximal stress during shaking.” The animal experimentation raises interesting theories regarding the areas of trauma within the brain and spinal cord during shaking.

Investigating cases of AHT to exclude spinal injury

The American College of Radiology [32] and British radiology standards [33] for a skeletal survey include anterior-posterior and lateral spinal views in a child younger than 2 years of age with suspected physical abuse and older children if skeletal injury is suspected. The necessity of both views to optimize chances of identifying all spinal fractures is confirmed by Barber et al. [11]. The content of the repeat skeletal survey 10–14 days later is more heavily debated. Sonik et al. [34] in their study of 22 children did not identify

any spinal fractures in follow-up skeletal surveys and together with Harlan et al. [35], who reviewed 101 children who underwent a full repeat SS, concluded that the spine could be omitted from follow-up. However, the low prevalence figures for spinal fractures in physical abuse would suggest that the studies were underpowered to make such recommendations. Some argue [9, 34, 35] that once abuse has been confirmed on initial investigations, the identification of further injury has little relevance to the child protection outcome. They further postulate that the low prevalence of spinal fractures in physical abuse and the risks and costs from excess radiation mitigate against repeating spinal radiation in a follow-up skeletal survey. However, arguments to support spinal imaging in a second skeletal survey would include the fact that since spinal injury has the potential for significant clinical morbidity, it is important to confirm equivocal spinal findings on the first skeletal survey and that the identification of the full extent of injury can contribute to discussions around the mechanisms of injury in legal proceedings [8]. Should spinal fractures be identified on skeletal survey then a spinal MRI should be done to exclude associated soft-tissue, ligamentous, spinal cord or extra-axial injury. As spinal neuroimaging utilizing MRI has the potential to identify spinal fractures or confirm equivocal findings, and is likely to be done in the course of clinical assessment for abusive head trauma, this may negate the need for spinal imaging in repeat skeletal surveys. Henry et al. [36] evaluated the sensitivity and specificity of cervical MRI in children (mean age: 9 years) and found that MRI had a high sensitivity for excluding bony abnormalities while CT had a low sensitivity for soft-tissue or ligamentous injuries, thus supporting the use of MRI in the investigation of pediatric cervical spinal injury. The prevalence of spinal subdural hemorrhage and ligamentous injury in children with abusive head trauma would support the recommendation that all children with suspected abusive head trauma should have a cervical spinal MRI early in the course of their clinical assessment. In children with abusive head trauma, whose level of consciousness is compromised, it is particularly important to stabilize the neck and include spinal imaging at the time of initial investigations.

Conclusion

There is clear evidence of spinal involvement in a proportion of cases of abusive head trauma. It is plausible that with the greater use of spinal MRI, further cases will be identified. The greater involvement of the cervical spine among children with abusive head trauma arguably supports flexion-hyperextension injury during shaking as a mechanism for abusive head trauma. Involvement of the upper cervical spinal cord may well induce apnea in a population of infants who have a relatively underdeveloped respiratory control center.

However, the injurious effects to the cervical cord are unlikely to be the only biomechanical trigger for the intracranial pathology seen. The biomechanics of shaking is inherently complex and rotational/hyperextension/flexion forces at different intracranial regions are likely to elicit injurious effects to the intracranial structures. Occult injuries at the thoracolumbar region are proving to be more common in abusive head trauma than previously recognized, and mechanisms for these injuries are proposed. Further comparative studies of the neuroimaging of the spine in abusive head traumas and accidental head injuries would assist in determining the specificity or sensitivity of spinal injury in abusive head trauma. Serial MRI imaging would outline the evolution and location of spinal subdural hemorrhage over time and contribute to a greater understanding of injury mechanisms.

Irrespective of our lack of knowledge of the full biomechanics of these injuries, it is important to undertake a series of thorough neuroradiological investigations to exclude occult spinal injury in any child with suspected abusive head trauma to optimize clinical management and medicolegal proceedings. MRI of the spinal column should be considered in any child with suspected abuse head trauma.

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