

Traumatic Spinal Cord Infarction in a 16 Month Child Complicated by Suspected Child Abuse: A Case Report

Jonathan Burgess Cardwell*

Vidant Medical Center, Greenville, North Carolina 27834, USA

*Corresponding author: Jonathan Burgess Cardwell, MD, Vidant Medical Center, Greenville, North Carolina 27834, USA, Tel: 678-673-1260; E-mail: joncard12@gmail.com

Received date: December 01, 2017; Accepted date: December 14, 2017; Published date: December 20, 2017

Copyright: ©2017 Cardwell JB. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Citation: Cardwell JB (2017) Traumatic Spinal Cord Infarction in a 16 Month Child Complicated by Suspected Child Abuse: A Case Report. *Pediatr Emerg Care Med Open Access*. Vol. 2 No. 1:2.

Abstract

Spinal cord infarction is a well-known entity in adult medicine, but remains exceedingly rare in children quite more so when considered by causation of trauma. This is the case of a 16-month old Hispanic child who presented to the emergency department greater than 24 hours after an unwitnessed fall from a table found to have a traumatic spinal cord infarction. The father initially voiced concern of the child vomiting and "not acting right." The child had a previous history of non-accidental trauma and initial plan of care reflected a suspicion of head injury in the absence of obvious external injuries. Subsequent examination and history would yield that the child had paraplegia with MRI showing acute/subacute infarction of thoracic spinal cord without obvious extraneural pathology. The patient had no worsening or improvement in symptoms and at 7 months after presentation would remain a paraplegic. Investigation by child protective services determined father neglected to seek care for >24 hours after the accident. The child was removed from his home and the father imprisoned on charges of child abuse. This case highlights the importance of early recognition of injury, detailed and specific history taking, and completeness of exam in setting of suspected abuse.

Keywords: Traumatic spinal cord infarction; NAT; Spinal cord injury; SCIWORA

Introduction

Medicine is continuously evolving, and definitions change to adapt to emerging technology and advancements in every field. A relatively recent emergence in the way of neuroimaging by MRI has offered insight into spinal cord injuries with no apparent radiological abnormalities while skewing a once proper definition known as SCIWORA. While it remains rare, there are increasing case studies that show, perhaps under the same umbrella, presentations of traumatic spinal cord infarctions with delayed onset. Timing of onset is

an important key in recognition given a presumed benefit in initiating treatments directed at neurological recovery and salvage. However, as we will see in this case of a 16-month old patient who presented with traumatic spinal cord infarction, the implications in an unclear picture of not seeking care proved to have legal ramifications in a suspected case of child neglect.

Summary of Case/Presenting Concerns

A 16 Month old male was brought to the pediatric emergency department in the evening by his biological father, and two non-family care-givers. The father reported, through a Spanish interpreter, that on the day prior to his visit to the emergency department he was at his home with the child when he left the child alone for several minutes to retrieve papers from his automobile. When he returned the child was lying on the kitchen floor crying with his head and torso between his spread legs. There was cereal covering the table and the father presumed the patient climbed over the table to reach the cereal and then fell from the table landing in the position in which he was discovered. He noticed the chair had been pulled away from the table as well. The father initially states the child was very unstable when standing, noting his knees shook under his own weight. He states that the patient appeared to favor his left leg and he thought he may have injured the leg. He gave the child ibuprofen and Tylenol for his continued irritability sustained after the fall. During the night the child continued to cry and began to vomit. He states the patient had a history of vomiting with Tylenol, so he thought this was a similar reaction. The father gave the patient more medications, avoiding Tylenol, and called his employer for recommendations. The employer, however, did not answer his phone. Later in the day he successfully contacted his boss, who then took the father and the patient to the emergency department. The father denied a witnessed loss of consciousness but did feel as though the patient was febrile.

It was noted on arrival to the emergency department that the patient had a previous history of cocaine exposure in utero

as well as non-accidental trauma wherein he presented with subdural hematomas and retinal hemorrhages. Chart review would show that the child was removed from his home for 3 months and stayed with care providers who presented to hospital with father on this most recent visit. He would eventually be returned to his biological father and mother. The mother reportedly left the family one month prior, per the father, who states she became addicted to drugs and has no current role in the care of the patient.

Clinical Findings

The initial evaluation by the emergency department staff was abbreviated and benign without acute findings to explain any evident pathology. The patient was awake and alert, crying softly with tear production. He was afebrile and norm cephalic without bulge of any fontanelle. There was no external injury or bruising to head, chest, abdomen or extremities. The spinal examination revealed no apparent tenderness, step-offs, bruising, or deformities. The patient had active movement of his upper extremities with normal tone and intact reflexes. Bilateral lower extremities were noted to be flaccid, areflexic and did not respond to pain. The bladder was palpable. Sensation was difficult to appreciate but thought to be between T8-T10.

Diagnostic Focus and Assessment

Given the history and presentation there was initial concern that this was another case of non-accidental trauma and a proclivity to pursue a cranial injury considered a suspected fall from a height and subsequent vomiting. The admitting team was called for early involvement regarding a sensitive issue. The patient was subsequently found to be paraplegic and further studies were considered. Further questioning of the father showed he had not seen the patient walk since the accident occurred as previously stated.

Temperature on admission was 100.6 with other vital signs within normal limits. There were no signs of shock. Computed tomography scan of brain without contrast showed not acute intracranial pathology. CTA of chest and abdomen showed no evidence of aortic injury or dissection. Abdominal ultrasound and chest x-ray were unremarkable. Complete blood count was within normal limits. Urinalysis showed trace leukocytes and 3+ ketones. Total body skeletal survey was negative for fractures, including the spine. Hypercoagulable studies were completely negative.

Magnetic Resonance Imaging of the spine showed: "Extensive acute/subacute infarct involving the thoracic spinal cord extending from the mid T6 level inferiorly with associated edema extending superiorly to the T3-T4 level. Extremely subtle signal abnormality within the right eccentric T7 vertebral body abutting the endplate may reflect osseous contusion/subtle compression deformity. No typical MRI findings of spinal vascular malformation."



Figure 1 Sagittal T2-weighted: Note infarct involving the thoracic spinal cord extending from the mid T6 level inferiorly with associated edema extending superiorly to the T3-T4 level.

Therapeutic Focus and Assessment

The patient was admitted to the pediatric ward where he was put on strict bed rest for six days with preference for remaining flat to ensure adequate spinal cord perfusion. Patient had PRAFO orthotics placed and a foley was placed with concern for a neurogenic bladder. An abdominal binder was placed for stability. Steroids or further bracings were noted used. Physical therapy began working with the patient early during his course of stay. It was not felt that the patient would have recovery of function given extent of infarction. Child protective services were notified. An investigation during the admission proved to yield no strong evidence to support a case of delay in seeking care for an injured child, although it was determined the child would go home with the caregiver allowing the father visitation rights given his high need for therapy and extensive care. The patient was discharged from the pediatric ward after several weeks and transferred to inpatient rehab. He was discharged to the caregivers' home where he was to continue routine therapy.

Follow-up and Outcomes

The patient continues to receive therapy three times weekly. He unfortunately remains a paraplegic but has shown progress in mobility with using arms to drag body distances during play. He lives in the home of the new caregiver and was recently officially adopted by the caregiver family. The case against his father was reopened with the initial concerns regarding father's delay of care for the child. The actual case findings and investigation were not available for this report, but he was eventually charged and imprisoned for suspected failure to seek medical attention for a child in a timely manner.

Discussion

Injury to the spinal cord without radiographic injury has been described as far back as the turn of the century in 1907 [1]. Specifically, the term SCIWORA (spinal cord injury without radiographic abnormality) was introduced by Pang et al. in 1982 [2]. They defined SCIWORA as signs of myelopathy resulting from trauma without evidence of ligamentous injury or fractures on plain x-ray films or tomographic studies. Noted exclusions were given for penetrating trauma, congenital spine anomalies, birth injuries and complications, and electrical shock [2,3]. With the advent of MRI technologies, the terminology previously described excludes abnormality found through this medium. Launay et al. performed a meta-analysis of all documented cases of SCIWORA presenting in the United States between 1980 and 2002 encompassing 392 cases. Their findings noted that in the 13 cases that reported initial MRI imaging results, extra neural or intraneural abnormalities were present in each one [1]. While this is a very low number of documented cases to reach any firm conclusions, it is likely most, if not all, cases would reflect structural abnormalities with the more sensitive MRI. Nonetheless, it should be noted that subtle fractures or dislocations in the young pediatric population can be missed because reflex muscle spasm may act to reduce the injury, resulting in a falsely normal radiograph [4].

While the definition continues to evolve for SCIWORA, it is also worth noting that it is a categorical term or description and less of a specific diagnosis. Of the different traumatic possibilities, three main ideas are thought to encompass most: longitudinal distraction, flexion/extension, and ischemia/infarction of spinal cord. The most latter takes special consideration in that it can present without trauma. Therefore, the qualifier of traumatic spinal cord infarction is a further designation and subset of SCIWORA [5].

SCIWORA remains a rare presentation in all settings but does show a predilection for children. In the United States, the reported incidence of all spinal cord injuries in the pediatric population is approximately 1300 new cases per year. The review by Launay et al. estimates SCIWORA in the pediatric population ranges from 19–34% of all spinal cord injuries with very few cases reported under the age of 1 [1,3]. It is often a devastating injury with a poor prognosis. They found that neurologic deficit or recovery after treatment was available for 109 patients of the 393 reviewed. Complete recovery occurred

in 36 patients (33%), partial recovery occurred in 16 patients (15%), no recovery occurred in 53 patients (49%), and four patients died (4%) [1]. According to Kriss et al., the only clinical prognostic factor in SCIWORA is the degree of neurologic injury at presentation. Severely injured spinal cords rarely improve significantly and children with mild deficits usually recover partially or fully.

The proposed mechanism of trauma or insult in SCIWORA is important in understanding why the pediatric population is the barer of most pathology. As previously mentioned there are 3-4 propositions on injury: hyperflexion/hyperextension, ischemia, and distraction. The most common noted etiologies include MVA, falls from a height, and direct blows to the thoracic area. While the trauma is presumably more severe in most cases, there have been multiple case reports of mild trauma that presented with SCIWORA. The specific mechanism of injury for the SCIWORA syndrome was available for 105 patients in the Launay review and was found to be flexion in 57 patients (54%); distraction in 22 patients (21%); extension in 19 patients (18%); and crush in seven patients (7%) [1]. Three were presumably a result of child abuse.

The infantile bony spinal column itself is elastic allowing for stretching up to 5 cm prior to rupture. The spinal cord, however, anchored inferiorly by the cauda equina and superiorly by the brachial plexus, is less pliable and distracts with only 5-6 millimeters of traction [4]. This allows deformation of the musculoskeletal structures beyond physiologic extremes, permitting direct cord trauma followed by a reduction of the bony spine to its approximate normal anatomical position [4]. Beyond the elasticity of the ligaments and joint capsules, children's facet joints also have a more horizontal orientation as well anterior wedging of the superior aspects of the vertebral bodies which collectively allows an increased range of motion in an AP plane [1,2,4].

This range of motion may not only directly injure the spinal cord but, as much, affect the blood supply resulting in ischemia, infarction and edema. The middle and lower thoracic regions of the cord have the poorest segmental blood supply [4]. This area receives blood supply from a single artery called the great anterior radiculomedullary artery or artery of Adamkiewicz (AKA). This artery typically originates on the left, variably from T9 to T12 intercostal arteries or less commonly anywhere from T5 to L2 [5]. As such, even a transient occlusion to this vessel can have devastating effects. While thrombus is a common cause of infarction in adults and children alike, a traumatic mechanism is more congruent with a direct impediment on or pressure-induced vasospasm of these vessels. In other cases of severe trauma, this is confounded by hypotension and poor perfusion secondary to blood loss, proximal injury to central essential vessels and/or impaired autoregulation by the central nervous system. The central cord appears to be more vulnerable to ischemia and injury, likely explained by its lack of collateral blood supply. Motor neurons in particular have been shown to be the most susceptible to anoxia, with paralysis being a hallmark of traumatic cord infarction. Presentations are often delayed from several minutes to several days with some reports of

initial post-injury wellness and deterioration on a later date without evidence or suspicion of recurring trauma.

The area of treatment and management is controversial and comes with less proven techniques that a care team must take into account in weighing benefits and risks. As such, this is not an exhaustive literature search for effectiveness or by any means the complete scope of possible therapy. As with any case, a high level of suspicion and caution, especially in the pediatric population, for spinal cord injury in the setting of severe trauma, including sports related injuries, falls from a height, MVA, and child abuse must exist. Patients must be treated in order of resuscitations with advanced life and trauma support in mind for stability, especially with many presenting with comorbidities and hemodynamic instability. Suboptimal perfusion pressure to a traumatized cord (already compromised by impaired autoregulation of blood flow) will cause further ischemic damage [5,6]. Beyond this, beginning in the prehospital setting, immobilization is a constant agreed upon standard of care. At the very least, the patient should remain flat. Some studies would support bracing of injured portion of spine for possible instability even in light of negative imaging, as was previously mentioned for fear of occult instability leading to recurrence or progression of injury. However, in some studies these recurrent episodes of injury recovered completely, and bracing did not provide additional benefits in preventing these episodes [2]. Nonetheless, repeat imaging is routinely performed given the possibility of repeat injury and further delineation of injury. Steroids are often given in support of worsening edema surrounding the cord. This potentially has some benefit in outcome if given within 6 hours, although this idea has been inconclusive and is certainly less proven in delayed presentation while coming with a multitude of risks including infection and bleeding. Finally, surgery may be an option depending on the presenting pathology but more likely is less indicated in the evidence of mild structural irregularity [7]. Again, the initial presentation of the patient is most closely associated with long term prognosis. Therapeutic rehabilitation is routinely started early in the acute phase of presentation and approached with a collective team of professionals who help maintain range of motion as well as adjustment and functionality of the new condition with regard to independence in daily living, recreational activities, and future employment.

While this pathology is less often seen in pediatric emergency rooms, it is certainly one that cannot be missed. It is a devastating injury that can change the course of a child's life. As such, practitioners should be diligent and steadfast in their early recognition and management as there is at least a limited amount of information that shows a benefit to early intervention. In the case of this 16-month old child, the initial appreciation of his injuries was not completely known. Spinal

precautions were not instilled immediately, and the accurate history was not provided as the father told a less than accurate story [8]. While this is no excuse for the miss, it does bring to light several key points to bear in mind. The first of these is to have the creative liberty to explore the possibility of such an injury in the first place given a traumatic mechanism in a young child. Secondly, in cases of suspected abuse or neglect, a complete and thorough examination are absolutely essential in exploring every considerable insult. Finally, clear documentation of all injuries, history and timeline of events is imperative in the case of legal consequences. While the father was arrested in this case, it would not be outside the realm of imagination to conjure a scenario where the timing of paralysis is less clear. The father did admit that the child had not moved his legs since the accident and that he had waited more than 24 hours to bring him to the hospital. This was likely the smoking gun in this case, but understanding the possibility of a delayed presentation in paralysis could have made this a more arduous burden of guilt [9].

References

1. Launay F, Leet AI, Sponseller PD (2005) Pediatric spinal cord injury without radiographic abnormality: A meta-analysis. *Clinical Orthopaedics and Related Research* 433: 166-170.
2. Pang D, Pollack IF (1989) Spinal cord injury without radiographic abnormality in children-the SCIWORA syndrome. *J Trauma* 29: 654-664.
3. Bansal KR, Chandanwale AS (2016) Spinal Cord Injury without Radiological Abnormality in an 8 Months Old Female Child: A Case Report. *Journal of Orthopaedic Case Reports* 6: 8-10.
4. Kriss VM, Kriss TC (1996) SCIWORA (spinal cord injury without radiographic abnormality) in infants and children. *Clinical Pediatrics* 35: 119-124.
5. Robles LA (2007) Traumatic spinal cord infarction in a child: Case report and review of literature. *Surgical Neurology* 67: 529-534.
6. Choi JU, Hoffman HJ, Hendrick EB, Humphreys RP, Keith WS (1986) Traumatic infarction of the spinal cord in children. *J Neurosurg* 65: 608-610.
7. Bosch PP, Vogt MT, Wogt WT (2002) Pediatric spinal cord injury without radiological abnormality (SCIWORA): the absence of occult instability and lack of indication for bracing. *Spine (Phila Pa 1976)* 27: 2788-2800.
8. Brown RL, Brunn MA, Garcia VF (2001) Cervical spine injuries in children: review of 103 patients treated conservatively at level 1 pediatric trauma centre. *Journal of Pediatric Surgery* 36: 1107.
9. Ahmann PA, Smith SA, Schwartz JF, Clark DB (1975) Spinal cord infarction due to minor trauma in children. *Neurology* 25: 301-301.