

Case Report

Chiari I malformation with acute neurological deficit after craniocervical trauma: Case report, imaging, and anatomic considerations

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Abstract

Background: In patients with Chiari I malformation (CMI), the occurrence of acute neurologic deficit after craniocervical trauma is rare. However, the pathologic potential of exacerbating anatomic overcrowding of the posterior fossa has immense clinical consequences and prompt recognition is essential.

Case Description: This case study describes a 41-year-old male who sustained a single blow to the face, fell, and struck the occiput. On admission, neurological examination revealed a profound paraparesis, upper extremity diplegia, a C4 sensory level and apnea that required intubation. On arrival, computerized axial tomography of the head showed a small amount of contrecoup left frontal traumatic subarachnoid hemorrhage. Magnetic resonance imaging (MRI) performed 19 h after admission was negative except for the presence of a CMI. He acutely declined on post injury day 2, prompting emergent decompression of the posterior fossa where anatomic overcrowding was observed. At 19 weeks post injury, his motor function had significantly improved.

Conclusion: The constellation of severe neurologic deficit in patients with CMI after relatively minor craniocervical trauma has been previously described. In our patient, neurologic deficit disproportionate to the mechanism of injury was observed and likely in part attributed to the presence of a Chiari malformation. Unfortunately, MRI has not yet been able to clearly define the underlying pathoanatomy, help understand the mechanism of injury, and delineate when operative intervention is indicated in these patients. Here, we review similar cases from the literature, examine findings on MRI, and evaluate mechanisms of injury following craniocervical trauma in patients with CMI to help clarify these questions.

Key Words: Chiari I malformation, central cord syndrome, craniocervical trauma

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INTRODUCTION

Chiari I malformation (CMI) of the adult type is considered rare and its incidence is uncertain.^[1] However, an incidence of 0.77% is suggested after a review of 22,000 magnetic resonance images (MRI) selected from a normal population that showed 169 to have cerebellar tonsillar ectopia >5 mm.^[2] CMI represents a spectrum of disease defined as overcrowding of the posterior fossa of varying degrees.^[13] In Milhorat *et al.*,^[13] 91% of the 364 patients diagnosed with CMI showed at least 5 mm of tonsillar descent below the level of the foramen magnum. The implications of tonsillar herniation compromising space at and below the level of the cervicomedullary junction (CMJ) reach beyond the usual presenting neurological signs and symptoms which accompany this finding. Symptom onset may be spontaneous, but when precipitated by head and/or neck trauma can result in severe motor and sensory deficits. More typically, headache of varying quality and degree, often affected by physical activity and position may also occur without provocation. Symptoms may relate to lower cranial nerve palsies such as dysphagia and/or dysphonia. Neurological signs may include spasticity, nystagmus, impaired visual acuity, and papilledema.^[6,9,12,13]

It is difficult to diagnose CMI because of these variable signs and nonspecific symptoms. MRI usually confirms the diagnosis. A certain group of patients may present acutely following trauma or in a delayed fashion. In Milhorat's^[13] series, 23 of the 126 patients (18%) with CMI presented after experiencing a whiplash injury or a direct blow to the head. In Levy's^[11] 127 cases of diagnosed CMI, 8 (6%) were previously characterized as having "posttraumatic syndrome." Patients with CMI who present after trivial craniocervical trauma (CCT) may have mild or moderate symptoms. In contradistinction, these patients may also experience acute quadriparesis,^[4,19] central cord syndrome,^[7] cervicomedullary paralysis,^[4] or death.^[20,23] The extent of neurological deficit is often disproportionate to the severity of the traumatic event in patients with CMI.^[12] A reduction in subarachnoid space at the level of the CMJ in individuals with CMI predisposes tissue within the foramen magnum to injury.^[6,12,21] Certain cases describe minor CCT in individuals with CMI [Table 1]. Our patient had a CMI and his neurological examination after trauma was also inconsistent with the severity of the injury. This case provokes discussion regarding the necessity of surgical decompression, optimal timing and sequencing of MRI, and the underlying mechanisms of injury.

CASE REPORT

History and examination

The patient is a 41-year-old male who was sucker punched in the face, fell, and struck his occiput

on a curb. A bystander initiated cardiopulmonary resuscitation. Glasgow Coma Score was reported to be 3 when emergency medical service arrived. He was intubated in the field. Blood pressure was 67/47 mm Hg. 0.4 mg of Atropine was given, which increased the blood pressure to 123/94 mm Hg. Upon arrival to the emergency department, the patient was found to be awake, alert, and attentive. Neurological examination showed a C4 sensory level with an upper extremity diplegia. The right lower extremity showed 3/5 strength in all muscle groups. The left lower extremity showed 1–2/5 strength in the plantar and dorsiflexors and absent strength proximally. Computerized axial tomography of the head showed left frontal traumatic subarachnoid hemorrhage. On admission, all other radiographic studies were normal. Sagittal MRI demonstrated 7 mm of tonsillar descent below the foramen magnum, consistent with CMI [Figure 1]. Axial MRI showed significant medullary compression, with the right tonsil causing more compression than the left [Figure 2]. No evidence of acute pathology was present. On post injury day 1, the patient acquired minimal intrinsic right-hand function and improved to 2/5 strength. On post injury day 2, right hand function was absent. Due to deterioration in neurologic status, a suboccipital craniectomy and partial C1 laminectomy with duraplasty were performed.

Surgery and operative findings

A second preoperative MRI study of the brain was performed the morning of surgery. A cine cerebrospinal fluid (CSF) flow study showed qualitatively decreased flow at the level of the foramen magnum. T2-weighted MRI demonstrated hyperintense signal in the pyramids at and below the foramen magnum and in the dorsal columns of the spinal cord [Figures 3 and 4]. At surgery,

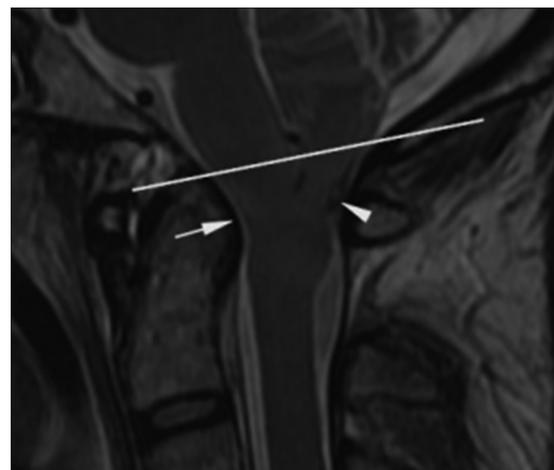


Figure 1: Preoperative. Nineteen hours after admission. Sagittal T2-weighted MR image (TR 3116.7 TE 102.0) of the CMJ shows 7 mm of tonsillar herniation (arrowhead). The subarachnoid space is absent and the CMJ is indented by the dens (arrow). There is no signal change suggesting acute injury. The reference line indicates the anatomical level of the axial image in figure 2

Table 1: The type of trauma is presented as head, cervical flexion/extension or a combination thereof. Radiographic imaging modalities were recorded as (CT) computed tomography or (MR) magnetic resonance. X-ray is recorded in the case where it was the only imaging study performed. Time contained in parentheses represents the interval between admission and initial imaging study. Clinical improvement was qualitatively assessed as none, minimal or significant. Surgical intervention was recorded as 1, suboccipital craniectomy or 2, cervical laminectomy

Author, yr	Age (yrs), Sex	Type of Trauma	Neurological Exam, Signs and Symptoms	Cranial Nerve Palsy	Respiratory Insufficiency	Imaging	Follow-up Period	Clinical Improvement	Surgical Intervention
Ajis <i>et al.</i> , 2004	37, F	Combination	Balance disturbance, Romberg sign, loss of consciousness	N	N	CT (unk) MR (unk)	6 hr	Significant	N
Bondurant <i>et al.</i> , 1993	2.25, M	Flexion/extension	Quadriplegia, bilateral Babinski signs, fecal incontinence, upper extremity areflexia	N	Y	CT (2 d) MR (2 d)	30 mo	Significant	N
Bunc <i>et al.</i> , 2001	35, F	Flexion/extension	Progressive neck pain, headache, upper and lower extremity paraesthesia, gait ataxia	Y (IX, X)	N	CT (5 mo) MR (5 mo)	1 yr	Significant	Y (1,2)
Couldwell <i>et al.</i> , 1998	11, M	Head	Loss of consciousness, hyperreflexia, bilateral Babinski signs	N	Y	CT (admission) MR (14 hr)	21 d	Significant	N
Erlich <i>et al.</i> , 1989	2.75, F	Flexion/extension	Neck pain, upper extremity diplegia and areflexia, bilateral Babinski signs	N	N	CT (admission) MR (2.5 d)	5 mo	Significant	N
Mampalam, <i>et al.</i> , 1988	13, F	Combination	Full cardiopulmonary arrest, aphonia, localized pain in all extremities, gag and palate areflexia	Y (IX, X, XI)	Y	CT (admission) MR (12 d)	35 d	Significant	N
Riviello <i>et al.</i> , 1990	2, F	Flexion/extension	Gait ataxia, progressive quadriparesis to quadriplegia	N	Y	CT (12 hr) MR (unk)	2 yrs	Significant	N
Tomaszek <i>et al.</i> , 1984	3, M	Combination	Vomiting, cardiopulmonary arrest and death	N	Y	X-ray (12 hr)	16-24 hr	Deceased	N
Vleck <i>et al.</i> , 1987	2.5, F	Flexion/extension	Paraparesis, bilateral Babinski signs, knee and ankle hyperreflexive	N	N	CT myelogram (1 d)	2 mo	Significant	Y (1,2)

Contd...

Table 1: Contd...

Author, yr	Age (yrs), Sex	Type of Trauma	Neurological Exam, Signs and Symptoms	Cranial Nerve Palsy	Respiratory Insufficiency	Imaging	Follow-up Period	Clinical Improvement	Surgical Intervention
Wan <i>et al.</i> , 2008	17, F	Flexion/extension	Progressive and intensifying chronic neck pain, nausea, severe headache	N	N	MR (8 mo)	37 mo	Significant	Y (1)
Woodward, Adler, 2018	41, M	Head	Paraparesis, upper extremity diplegia, a C4 sensory level and apnea	N	Y	CT (admission)	3 yrs	Significant	Y (1)

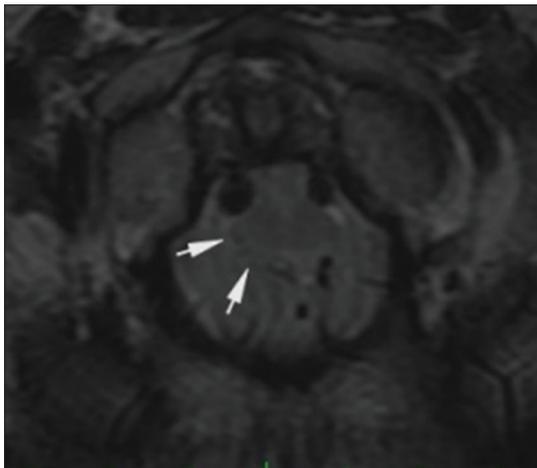


Figure 2: Preoperative. Nineteen hours after admission. Axial T2-weighted MR image (TR 5150.0 TE 101.0) shows dorsolateral compression of the medulla (arrows) without evidence of surrounding CSF. There is no signal change identifying acute injury

the tonsils were found to be herniated to the level of the arch of C1 and adherent to the obex. The arachnoid was not opacified. Arachnoid adhesions were lysed. The right tonsil descended 4 mm more than the left. No tonsillar or cerebellar contusion was identified grossly. Subarachnoid hemorrhage was not present. The tonsils were not coagulated.

Postoperative course

On postoperative day 2, right intrinsic hand strength improved to 1/5. Antigravity strength was present in all muscle groups of the right lower extremity. The left lower extremity remained plegic. The C4 sensory level was unchanged. A tracheostomy was performed on postoperative day 7 for persistent apnea. Postoperative cine MRI showed restoration of CSF flow at the level of the foramen magnum. Three weeks post injury, T2-weighted hemorrhagic gradient echo sequence MRI (TR 867, TE 35, and FA 20) showed low-intensity signal in the ventral medulla, bilaterally along the midline and in the dorsal columns. A postoperative pseudomeningocele was identified and aspirated

percutaneously without recurrence. Neurological examination remained unchanged by the time of discharge 3 weeks later. Nineteen weeks post injury, the patient showed significant improvement. Antigravity strength was present in all muscle groups. Sensation to pinprick returned to normal in all extremities. The patient ambulated with assistance and was being actively weaned from the ventilator but remained in a rehabilitation institute.

DISCUSSION

Surgical management

The decision to operate on a patient with CMI who becomes symptomatic after CCT is not clear-cut. A review of the literature showed 10 such patients without syringomyelia, hydrocephalus, or other craniospinal abnormality [Table 1]. These individuals became symptomatic to varying degrees after CCT. The traumatic event was regarded by the authors as trivial,^[4,5,19] minor,^[20,21,23] or disproportionate to the severity of the injury.^[12] Follow-up ranged from 6 h to 37 months. All three patients who underwent suboccipital decompression, with or without laminectomy, improved neurologically.^[5,21,23] Two of the three patients had a normal preoperative neurological examination and experienced resolution of symptoms with decompression.^[21,23] The third patient was a child who had severe central cord syndrome but ambulated freely 2 months after surgery.^[21] Those patients who did not undergo neurosurgical intervention improved, except for one who expired within 24 h of the injury.^[20] It remains difficult to establish strict criteria for surgical intervention in this type of patient because of the paucity of cases reported. The decision to operate on our patient was made within 48 h of admission. It was based on the severity of the patient's deficit, as well as a deterioration of strength in the right hand from 1/5 to 0/5.

The majority of patients in this small series improved both with and without surgery. However, the enhanced

risk of neurological deficit after trivial CCT is worth attention and is an important criterion to consider the decision to perform prophylactic decompression in certain patients with CMI. Selected surgical series document rates of improvement in patients with CMI who have not experienced trauma between 80 and 90% and a 10–21% rate of relapse or persistence of symptoms.^[10,17] Thus, there is inconclusive evidence to speak definitively for or against surgical intervention in the symptomatic CMI patient. However, the presence of CMI increases the risk of injury to neural tissue within the foramen magnum and is a substantial factor regarding decompressive surgery.^[6,12,13] The neurological deficits sustained in the cases cited are inconsistent with the severity of the craniocervical insult and may require a prolonged hospital and rehabilitative course.^[12] In addition, respiratory arrest and sudden or near sudden death have been known to occur in both children and adults with CMI, with and without trauma.^[8,16,18] Although symptomatic patients with CMI who have decompressive surgery may not benefit permanently, surgery may still play a protective role against incurring severe neurologic injury following CCT.

Magnetic resonance imaging

MRI is the best radiographic modality for determining spinal cord and brainstem injury. However, within the first 12 to 24 h, signal change may be difficult to detect or distinguish from artifact, especially at the level of the skull base. In the cases reviewed, T2-weighted MRI demonstrated hyperintense signal in the spinal cord and brainstem. This finding was characterized as edema,^[7] cervicomedullary swelling,^[4] or cord contusion,^[7] and was identified within 48 h of injury. No additional follow-up MRI were obtained in these cases. We found that MRI 19 h after admission did not identify the cause of our patient's neurological deficit. However, 2 days post injury, hyperintense signal became visible and was thought to be most consistent with edema at the level of the CMJ [Figures 3 and 4]. Three weeks post injury, pathologic findings were best demonstrated using axial T2-weighted hemorrhagic gradient echo sequence MRI (TR 867, TE 35, and FA 20) [Figures 5 and 6]. This signal change was hypointense and most consistent with early deposition of hemosiderin in areas of initial contusion. These hypointense foci correspond to previously observed areas of hyperintense signal 15 days earlier in the pyramids and bilaterally within the central and dorsal portions of the caudal medulla [Figures 3 and 4]. Changing the MR relaxation and excitation times can facilitate visualization and differentiation of edema and hemosiderin depending on the time at which imaging is performed following initial injury (compare Figures 3 and 4). Furthermore, T1-weighted imaging may also help differentiate subacute hemorrhage from hemosiderin deposition in the setting of trauma 2 to 3 weeks post injury.^[24]

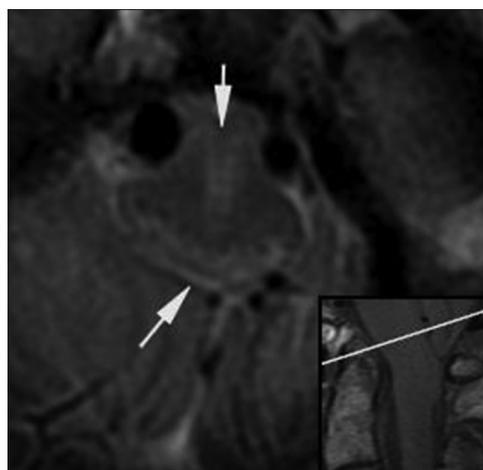


Figure 3: Preoperative. Sixty-one hours post injury. Axial T2-weighted MR image (TR 7200.0 TE 119.6) at the level of the foramen magnum shows hyperintense signal in the medulla in a paramedian location (short arrow) and in the dorsal columns (long arrow). The medulla is compressed by the cerebellar tonsils. The reference line of the inset sagittal image corresponds to the anatomic level of the axial image

Mechanisms of injury

Herniation of the cerebellar tonsils minimizes space below the foramen magnum and increases the risk of injury to the brainstem and spinal cord at the CMJ. In our patient, preoperative axial MRI show asymmetric dorsolateral compression of the brainstem from the cerebellar tonsils. The dens is angulated and protuberant posteriorly and compromises the spinal canal most at this level. There is no evidence of CSF at the level of the CMJ, which is indented ventrally by the dens. The medulla is effaced, shifted anteriorly to the left, and contiguous with the posterior margin of the odontoid process [Figures 1 and 2]. Together, these findings render the CMJ more vulnerable to compressive forces and mechanical injury.

In this patient, we suggest that flexion and/or extension of the atlantooccipital junction created a direct and forceful movement of the medulla and brainstem into the posterior aspect of the dens. Here, the spinal canal is made most narrow by the tonsils dorsally and the odontoid process ventrally. Sensory deficit appears to be less common and was not present in any of the cases reviewed. The C4 sensory level in our patient was a function of dorsal column involvement, albeit relatively distant from the posterior margin of the odontoid process and the direct point of trauma. Signal change in the dorsal columns was observed, implicating involvement of the cuneate and gracilis nuclei and fasciculi.

Several other mechanisms for traumatic brainstem and spinal cord injury in patients with CMI have been proposed but none are agreed upon. It is postulated that with blunt head trauma, a transient increase in intracranial pressure can promote further herniation of cerebellar tissue.^[13,20] Tonsillar impaction within the

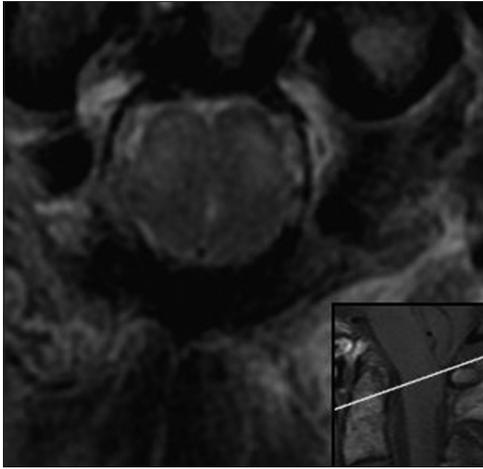


Figure 4: Preoperative. Sixty-one hours post injury. Axial T2-weighted MR image (TR 7200.0 TE 119.6) at the level of the dens shows hyperintense signal in the anterolateral aspect of the rostral cervical spinal cord. The medulla is compressed by the cerebellar tonsils. The reference line of the inset sagittal image corresponds to the anatomic level of the axial image

foramen magnum may prevent the normal transmission and dissipation of increased intracranial pressure to the spinal subarachnoid space.^[20] In the case reported by Tomasek,^[20] increased intracranial volume secondary to edema or hemorrhage caused acute medullary compression and death from tonsillar herniation. Couldwell *et al.*^[6] speaks of medullary compromise in blunt head trauma with motion of the “neuraxis relative to the skull” or from “hyperextension of the atlantooccipital junction.” It is plausible that with traumatic flexion and/or extension of the neck, cerebellar tissue at and below the level of the foramen magnum may exacerbate mechanical compression of the brainstem at the CMJ. Sudden increases in compression may result in ischemia and contusion. It has also been suggested by Wolf^[23] that compression of the tonsils against the posterior free edge of the foramen magnum causes dysfunction of the medullary cardiorespiratory centers, resulting in cardiopulmonary arrest. In children, it has been suggested that “compression and traction forces and vascular compromise” are intensified by tonsillar herniation in flexion and/or extension injury.^[4] When whiplash and blunt head injury occur together, one or more of these mechanisms may be responsible for causing neurological deficit or symptom onset.

In some of the cases reviewed, motor deficit was a common denominator, suggesting pyramidal tract involvement. In the case presented by Erlich,^[7] T2-weighted MRI demonstrated increased signal at the CMJ in a child who rolled off a low-lying bed. The location of signal change was diffuse within the rostral cervical spinal cord and medulla. Whether this increased signal represents edema, contusion, or a combination thereof remains unclear. Similarly, in other cases reviewed, increased signal does not clearly

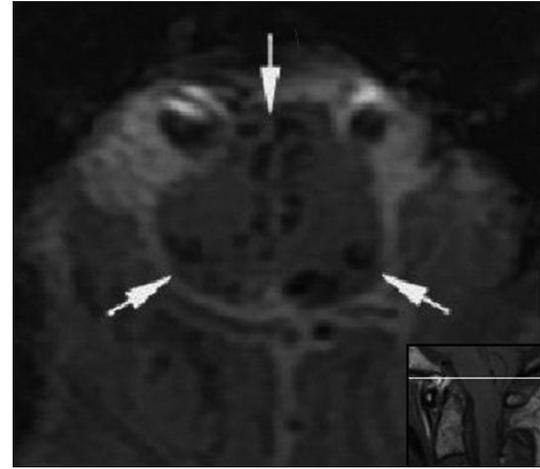


Figure 5: Postoperative. Twenty-one days post injury. Axial T2*-weighted hemorrhagic gradient echo sequence MR image (TR 867.0, TE 35.0 and FA 20) shows hypointense signal in the medulla consistent with the deposition of hemosiderin at the level of the foramen magnum in a paramedian and dorsal location (arrows). The medulla is decompressed with presence of a subarachnoid space. The reference line of the inset sagittal image corresponds to the anatomic level of the axial image

define the underlying pathology. Erlich *et al.*^[7] inferred that the pyramidal decussation was damaged with arm fibers more affected than those of the lower extremities. This deficit results from the presumed more rostral location of the crossing arm fibers and their increased proximity to the compressive forces applied during craniocervical injury.^[7] Conversely, the lower extremity fibers of the pyramidal decussation were thought to be relatively caudal in location and more distant from the compressive forces, possibly conferring a degree of protection.^[16] This explanation has been generally accepted for injuries at the CMJ that cause central cord syndrome or Bell's^[3] cruciate paralysis. However, evidence exists to challenge this assumption. After studying two species of monkey, Pappas *et al.*^[16] reported no difference in decussating fore and hind-limb fibers and that these fibers intermingle. Additionally, this group also discovered significant differences in fiber location. However, the anatomical relevance of these findings to humans remains unclear.

There is no definitive explanation for cruciate paralysis. Those explanations that have been proposed include: (1) Somatotopic organization of the pyramidal decussation;^[12,15] (2) Rostral and medially located arm fibers with greater susceptibility to injury than the caudal and laterally located leg fibers; (3) Differential longitudinal termination of corticospinal fibers with the cell bodies of the arm located proximal and the leg distal to the compressive force;^[16] and (4) A well-developed anterior corticospinal tract subserving arm function that is more proximate to compressive forces than those subserving leg function. The exact organization and location of the fibers of the pyramidal decussation in

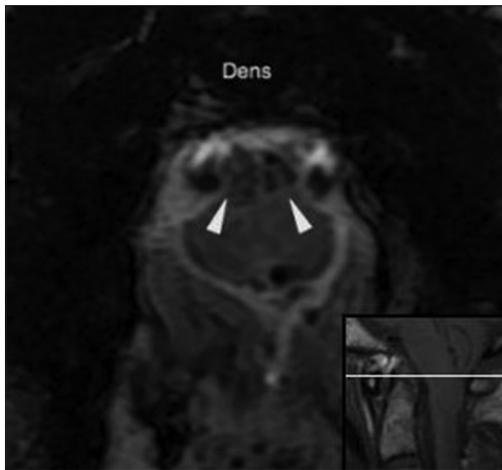


Figure 6: Postoperative. Twenty-one days post injury. Axial T2*-weighted hemorrhagic gradient echo sequence MR image (TR 867.0, TE 35.0 and FA 20) at the level of the dens shows hypointense signal within the ventral cervical spinal cord consistent with the deposition of hemosiderin (arrowheads). The reference line of the inset sagittal image corresponds to the anatomic level of the axial image

humans remains elusive.^[17] This patient demonstrated a variation of central cord syndrome. His neurological examination also raises questions regarding the exact location and extent of the decussating and nondecussating arm and leg corticospinal fibers. However, a somatotopic organization seems likely, given the well-defined pattern of cruciate paralysis that can result with injury to the rostral cervical spinal cord and medulla.

CONCLUSIONS

Minor CCT in the CMI patient may cause severe neurological deficit. However, there exists a paucity of reported cases, and the efficacy of suboccipital decompression for these patients remains unclear. Trauma in the cases reviewed, although felt to be minor, often resulted in disproportionate neurologic compromise. We feel that neurologic dysfunction following trauma in those with CMI without syringomyelia or hydrocephalus is multifactorial and determined by the following variables: (1) Age of the patient; (2) Type and severity of trauma, (3) Extent of tonsillar herniation, (4) Dens morphology and angle of retroflexion, and (5) Anatomic variation among individuals in location, proportion, and differential susceptibility to injury of decussating arm and leg corticospinal fibers.

Detailed imaging of the CMJ remains difficult and fiber tract anatomy is not easily obtained. Differentiating edema from contusion in the acute phase post injury is also challenging, but likely to have significant prognostic implications. Advancement of parenchymal MRI techniques about the skull base will allow for better identification and stratification of injury and inform prognosis to correlate neurological deficits with underlying pathoanatomy.

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Conflicts of interest

There are no conflicts of interest.

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