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Review Article

Getting to Know the Many Faces and Facets of Chiari Malformation Type 1 in Association with Blunt Force Trauma

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Abstract

There are numerous factors to consider when examining and treating a patient with a history of blunt force trauma or motor vehicle accident. The following review lays a foundation in understanding the mechanisms of Chiari malformations, acquired and inherited; the focus of the review being Chiari malformation type 1 (acquired) in association with blunt force trauma. Also, a range of case reports of patients who sustained malformations are reviewed for key points in the differential diagnosis of Chiari malformations. In an emergency room or intensive care setting, the prompt recognition of a Chiari malformation in a patient can be life-saving.

Keywords: *Blunt Force; Brain Injury; Cerebellum; Cerebellar Tonsils; Chiari; Encephalocele; Hearing Loss; Visual Disturbances*

Abbreviations

A-P: Anterior-Posterior; CM: Chiari Malformation; CM-0: Chiari Malformation Type 0; CM-1: Chiari Malformation Type 1; ER: Emergency Room; MRI: Magnetic Resonance Imaging; MVA: Motor Vehicle Accident

Introduction

Chiari malformation (CM) is characterized as an acquired defect or heterogeneous congenital anomaly of the cerebellum, brainstem, and or cervico-occipital junction. Except for CM type-0, CM types 1–4 are regarded as pathological continua of hindbrain maldevelopments characterized by a downward herniation of the cerebellar tonsils [1–5]. In CM type 0 (CM-0), no herniation is evident but other Chiari-like pathophysiologies (i.e., a tight cisterna magna, arachnoid lesions, fourth ventricular veils and obstructions, and syringomyelia may exist [5–7]. In CM types 1–4, caudal herniation of the cerebellar tonsils (tonsillar ectopia) is present, with or without brainstem descent, hydrocephalus, syringomyelia, and osseous abnormalities at the craniovertebral junction [8,9]. Any or all of the CM types may be accompanied by osseous defects, such as atlas assimilation, atlantoaxial dislocation, hypoplastic posterior fossa, Klippel-Feil anomaly, platybasia, basilar invagination, or lacunar skull [10].

Defining CM-subset characteristics

CM-0 involves the obstruction of the CSF flow and persistence of Chiari-like symptoms in the absence of tonsillar herniation. According to Freeman *et al.* (2010), the fundamental definition of a CM-1 (tonsillar ectopia) is "a pathological caudal herniation of the cerebellar tonsil(s) into the foramen magnum that extends five millimeters or more below the basion-opisthion line with or without symptoms" [9]. CM types 1.5–4 are progressively differentiated from CM-1 by the downward displacement of additional brain tissue and or anomalies

[4]. For example, CM 1.5 and CM-2 are associated with myelomeningocele; the medulla, fourth ventricle, and cerebellar vermis are also downwardly displaced. CM-3 involves a small posterior fossa and displacement of the cerebellum and brainstem into a posterior encephalocele. CM-4 is characterized by cerebellum aplasia or hypoplasia [1,9].

Etiological factors

CM-1 and CM 1.5 malformations can develop congenitally due to overcrowding within the cranial vault, or as a result of acromegaly, CSF leaks, craniosynostosis, growth-hormone injections, hydrocephalus, head and neck injuries, Paget's disease, surgical procedures, space-occupying lesions, or tethered cord syndrome [1,5]. Acquired defects can be seen when cerebellar tonsillar herniation occurs secondary to their downward displacement by mechanical factors (i.e., superior to inferior directed force) [10,11]. Whereas, CM-2–4 originate only as congenital anomalies [3]. Congenital anomalies occur more frequently than acquired defects. CMs were once thought to be rare; however, with the growing use of magnetic resonance imaging (MRI), more cases of CM are being uncovered. CM-1 is the most frequently diagnosed form among all Chiari malformations [1].

Discussion

Blunt force or blunt trauma caused by blows, falls, and motor vehicle accidents (MVAs) are a common cause of traumatic brain, muscle, spine, and nerve injuries [12]. Direct contact is responsible for some injuries (i.e., the head or neck making contact with a stationary object); whereas, indirect contact is responsible for other types of injuries (i.e., rapid acceleration and deceleration of flexion-extension forces exerted on the head and brain, neck, and torso). Increasingly, medical case reports, literature reviews, quantitative and qualitative studies, and published case-law are reporting that CM-1 can be acquired de novo or awakened from a quiescent state as a result of such trauma. Also, a Chiari 1 malformation can be acquired or awakened as a result of lifting or iatrogenic injuries secondary to manipulative and surgical procedures (i.e., multiple lumbar punctures, placement of shunts, spinal manipulations, or vigorous forceps deliveries) [13,14]. A retrospective incidence and validity study performed by Wan *et al.* (2008) found that minor head and neck trauma could precede the onset of symptoms in previously asymptomatic patients [15]. Also, this study of eighty-five patients found, using specific inclusion criteria, that eleven subjects (12.9%) had a history of minor head or neck trauma before being diagnosed with CM-1. Finally, it was noted that three subjects (3.5%) had symptoms directly attributed to trauma, using strict inclusion criteria [15].

Clinical manifestations

Clinical manifestations of CM-1 include the following: acoustic disturbances (decreased acuity, pressure, hyperacusis, sensorineural hearing loss, and conductive hearing loss); breathing difficulties; cranial nerve dysfunction (persistent, lower); cerebellar dysfunction (ataxia, drop attacks); dysesthesia (extremities, trunk); developmental delays (in infants); extremity weakness (upper and lower); facial pain and pressure (intermittent); excessive drooling (infants); failure to gain weight or thrive (infants); headaches (continuous or intermittent, global or occipital, pulsating or throbbing type, debilitating) that worsen with strain, exertion, or postural change, valsalva maneuvers; irritability with being fed (infants); neck pain (limited extension ROM, muscle tension, throbbing); neck stiffness (infants); numbness (facial, upper extremities); nystagmus (downward or lateral gaze); oropharyngeal dysfunction (chronic cough, choking, difficulty swallowing, gagging); regurgitation; orthostatic disturbances (dizziness, disequilibrium, syncope); paresthesias (upper extremities); psychological disturbance (anxiety, depression): respiratory distress; scapulothoracic pain, sleep disturbances (apnea, insomnia); spinal curvature, truncal weakness, vertigo; visual disturbances (blurred vision, diplopia, decreased vision, flashing lights); vocal cord disturbances; vomiting; and weak cry (infants) [16–22]. Also reported are associations with traumatic brain injury, epilepsy, increased muscle tone, mental retardation, scapular winging, scoliosis, speech delay, and torticollis [12,20,22–24] (additional references omitted).

A review of specific trauma-related case studies

Numerous studies have been published regarding Chiari malformations (4250 per a search of PubMed using the term "Chiari malformation"). A significant number of studies noted that traumatic head, neck, and brain injuries can result in Chiari malformation initiation or aggravation. The studies further noted that these malformations frequently go unrecognized and untreated even after

the patient reports to a primary care provider, urgent care center, or emergency room (ER). The review of research showed that CM-1 malformations are frequently discovered de novo following blunt trauma. Such findings are described in the following studies.

Mehta *et al*. (2011)

A previously diagnosed and symptomatic 32-year-old female presented to an outpatient clinic twice within a six-week period—before an MVA and following the MVA. Initially, her symptoms were limited to intermittent bifrontal headaches. Per MRI findings, she was found to have tonsillar ectopia of 8 mm descent with normal CSF flow. Within a day of the MVA, she began experiencing an increase in muscle pain, neck stiffness, and frontal tussive headache. After presenting to the ER, one day post-MVA, she was treated for back pain. Over the next few weeks, she began experiencing an increasing in intensity occipital headache, burning sensation at the back of her head that radiated into her right arm, facial numbness, and right arm numbness with non-dermatomal distribution, increased pain with deep breathing, a sensation of nausea and dizziness, and decreased cervical range of motion. Repeat MRI six weeks after the initial visit demonstrated tonsillar ectopia of 12 mm descent. During decompression surgery, the tonsils were noted to be markedly distended and elongated with compression, and a partial web was found over the obex. A CSF leak was noted on CT myelogram, and a CT-guided epidural patch was placed at C6/C7, after which all symptoms resolved [25]. Key points include the following:

1. Patients with CMs can remain asymptomatic for years until a trigger (i.e., head or neck trauma) causes them to become symptomatic.
2. This case demonstrates the strong temporal relationship between trauma and radiographic progression of CM-1 malformations.
3. Traumatic injuries to the head and neck may cause a CSF leak resulting in spinal intrathecal hypertension and symptomatic progression.
4. A transition from asymptomatic to symptomatic CM-1 can be presupposed as a result of CSF leak.

Kim *et al*. (2010)

A previously undiagnosed and asymptomatic six-year-old female presented to the ER after sustaining a minor cervical injury following a fall. Her sister accidentally dropped her while giving her a "piggyback ride". MRI revealed cerebellar tonsillar herniation of 7 mm descent with mild ventriculomegaly and an Evans ratio of 0.33 (for ventricular size). Her obex measured 2 mm superior to the foramen magnum and her medulla oblongata anterior-posterior (AP) diameter measured 8 mm. The angulation of the odontoid process measured 75 degrees. A spinal MRI was not performed at that time. At the age of 8 years, her 2-year comparative MRI did not show any marked change, and she continued to remain asymptomatic. However, at the age of 13 years (seven years post-diagnosis), she began to complain of intermittent neck pain that occurred with exercise and became exacerbated with neck extension. A comparative MRI revealed the following changes: 1) increased tonsillar herniation of 9 mm descent and 2) a shift in her obex from 2 mm superior to the foramen magnum to 1 mm inferior. At the nine-year post-traumatic mark (age 15 years), significant changes were noted as follows: 1) tonsillar descent had progressed to 24 mm per mid-sagittal MRI (however, the herniations were asymmetrical as the right reached the C-2 level, whereas the left reached the C-1 level); 2) the obex had continued to shift and was now 8 mm inferior to the foramen magnum; 3) the medulla oblongata appeared flattened and elongated after its AP diameter changed to 6 mm; 4) the angulation of her odontoid process had progressed from 75 to 68 degrees (however her Evans ratio for ventriculomegaly remained unchanged). Cervical and thoracic MRIs taken at this time demonstrated syringomyelia that extended from of C-1 to T-10 [4]. Key points include the following:

1. The clinical appearance of Chiari malformations can progress over time.
2. Patients can experience a change in the level of signs and symptoms over time.
3. Patients with CM-1 can have minimal symptomatology and major diagnostic imaging features of the malformation, or vice versa.
4. CM-1 can be diagnosed with or without syringomyelia. A small group of patients falls into the subcategory of CM-1.5.
5. It is crucial to consider the differential diagnosis of Chiari 1.5 in that different treatment strategies are needed, as the incidence of unresolved syringomyelia after decompression surgery is nearly two-fold higher in CM-1.5 than CM-1, leading to repeat surgery.

Freeman *et al*. (2010)

This prospective study found that subjects with a history of an MVA associated with neck pain had a substantially higher frequency of clinically significant cerebellar tonsillar ectopia than non-traumatic neck pain subjects. The frequency of this finding in trauma patients was four times greater than in non-trauma patients when the subjects were evaluated with an upright MRI scanner [9].

Uzoigwe *et al*. (2009)

A previously asymptomatic and undiagnosed 27-year-old woman presented in an outpatient setting with neck pain and occipital headaches after experiencing a stationary rear-end car accident. Her symptoms became debilitating after experiencing a second stationary rear-end car accident within two months of the first. Initially, her symptoms were limited to restricted cervical movement in all directions and marked tenderness in the cervical and lower occipital regions. After the second MVA, she began experiencing pain and paresthesia in her left arm, neck pain, and occipital headaches that prevented her from sleeping. An MRI taken after the second accident revealed an inferior herniation of the cerebellar tonsils of 10 mm [26]. Key points include the following:

1. Neurological sequelae of CMs can include cranial nerve deficits and limb pain and weakness.
2. There is an overlap of symptoms between cervical acceleration-deceleration (flexion-extension) injuries and Chiari malformation. Commonly shared features include headache, neck pain, shoulder pain, arm numbness and weakness, and cranial nerve neuropathies leading to vertigo and diplopia.
3. Clinicians must consider both differentials when evaluating patients after head or neck trauma. It is vital to undertake a thorough clinical investigation to rule out such severe conditions as Chiari malformations.
4. The neurological picture of CMs can progress and result in tetraplegia or death.

Murano and Rella (2006)

A previously undiagnosed 36-year-old woman with a history of chronic headaches presented in the ER immediately following an MVA where she was struck on the driver's side of the vehicle she was driving, lost consciousness, and had no recall of the accident. Presenting symptoms included headache, hand pain, and abnormal reflexes. Later, she began to experience worsening headaches and vomiting. CT and MRI taken during this ER visit demonstrated hydrocephalus and a 20 mm Chiari malformation type 1 with no syrinx (the hand pain was related to a fracture per CT). The patient was referred to the neurology department, and a shunt was placed six weeks after the MVA [27]. Key points include the following:

1. Symptomatic patients may progress after major or minor physical trauma.
2. Is not always clear whether the trauma is initiating or aggravating the symptoms.
3. Patients who become symptomatic or whose symptoms progress after trauma should be monitored carefully for more than 24–48 hours to ensure there are no complications, especially concerning the respiratory or cardiac systems.

Suleyman *et al*. (1999)

A previously asymptomatic and undiagnosed 20-year-old man presented to the ER immediately following an MVA with a complaint of neck pain. Despite negative cervical spine and skull x-rays, he was admitted to the hospital for physical therapy. On day four, he went into respiratory failure while performing neck exercises, and was transferred to the intensive care unit. His neurological decline included unconsciousness, lack of motor or verbal responses, quadriplegia, lower cranial nerve dysfunction (isochoric and miotic pupils), and a very weak light reflex. Follow-up x-rays were negative. Cervical and thoracic spine MRIs taken on day seven revealed Chiari malformation with a hyperintense lesion in the medulla (subacute hemorrhage). Decompression surgery resulted in spontaneous breathing and improvement in motor function. Aspiration pneumonia and a second arrest subsequently occurred, followed by labored breathing and difficulty in swallowing. Comparative MRIs twenty days post-op revealed a narrowing of the cervical syrinx, an abnormal medullary cistern, and opening of the fourth ventricular outlet [28]. Key points include the following:

1. Chiari malformation can manifest as variations in otherwise healthy adults and adolescents.

2. Sudden onset of symptoms may occur due to rapid enlargement or formation of a cervicothoracic syringomyelia defect secondary to aggravation of a preexisting Chiari malformation.
3. A Chiari malformation with syringomyelia may cause a patient to be sensitive to hyperextension of the atlantooccipital junction.
4. It is imperative to include CM-1 as a differential diagnosis in patients presenting with neck pain, head pain, or cerebellar signs (after a closed-head injury, flexion-extension injury, or chiropractic manipulation), paraplegia, tetraplegia, lower cranial nerve dysfunction, or recurrent respiratory arrest.

Sakai *et al.* (1990)

A previously undiagnosed asymptomatic 55-year-old male was diagnosed with CM-1 approximately three months after being involved in an MVA. The initial symptom, manifested shortly after the accident, was limited to mild nuchal pain. Three weeks later, the patient began experiencing pain radiating from his neck into his right shoulder, arm, and hand, and blurred vision. His symptoms worsened to include gait instability and difficulty in swallowing. After being admitted to the hospital due to these symptoms, he was found to have horizontal nystagmus and a slight rotary component-accentuation that was greater in the right eye. Also, he was found to have a depressed gag reflex, mild hyperreflexia, hyperthymesia of the third and fourth fingers, and a mild sensation of pain and throbbing on the right side of his body. An MRI revealed chronic subdural hematoma in the right frontotemperoparietal region, and tonsillar herniation due to CM-1. The swallowing difficulties and neck pain worsened during his hospitalization. However, all symptoms improved after the evacuation of the hematoma and decompressive surgery [29]. Key points include the following:

1. Biomechanical factors are fundamental considerations in the onset of symptoms of CM-1 malformations.
2. A downward displacement of the cerebellar tonsils can be produced by the breakdown of the pressure equilibrium position between the supratentorial, infratentorial, and spinal spaces.
3. Trauma can cause a supratentorial acute or chronic subdural hematoma, which can provoke the herniation of tonsils and initiate or worsen any symptoms.

Mampalam *et al.* (1988)

After sustaining a closed-head injury, the patient experienced transient upper extremity weakness, persistent lower cranial nerve dysfunction, and slowly resolving cerebellar signs. MRI demonstrated tonsillar ectopia. However, displacement of the brain stem or syringomyelia was not present [30]. Key points include the following:

1. CM-1 should be included in the differential diagnosis of patients presenting with upper extremity weakness, lower cranial nerve dysfunction, or cerebellar signs after experiencing blunt trauma.
2. Sudden death has been reported in persons with known and unknown Chiari malformations following blunt trauma.

Kara *et al.* (2016)

A previously asymptomatic and undiagnosed 20-year-old male, whose father had CM-1 and died unexpectedly at age 45, was transported to the ER after a heavy object fell on him. He had an open fracture of the tibia, and was tachycardic. All other vital signs were normal. He was conscious, alert, and oriented. A craniocervical CT revealed cerebellar tonsillar herniation of 6.5 mm descent below the foramen magnum and a cystic lesion consistent with a syrinx in the lower cervical and thoracic spine, medulla spinalis. CT revealed a fracture of the twelfth thoracic vertebra, segmental fracture in the left femoral diaphysis, and segmental fracture in the ramus, superior and inferior to the pelvis, bilaterally. The patient was actively bleeding from the tibial injury, and was kept hemodynamically stable with erythrocyte suspension. During surgery, he was placed under general anesthesia. However, he never regained consciousness. Cerebral edema was noted, brain-stem reflexes could not be detected (despite antiedema treatment), vital EEG activity was absent, SPECT and Doppler USG showed no cerebral flow, spontaneous ventilation was not detected, and the apnea test was positive. Thus, the patient was declared brain dead, and cessation of cardiac function soon followed [31].

Zhang *et al.* (2013)

A de novo finding of CM-1 during an autopsy was reported following the death of a 17-year-old who fell off of a motorbike during vehicular acceleration. Although she was previously asymptomatic and healthy, the autopsy revealed an extremely large, cerebellar, left-sided tonsillar herniation, multiple unexplained cavities in the cerebral hemispheres, loss and migration of the Purkinje cells, and capillary congestion of the herniated tonsil. The cause of death was considered to be cardiopulmonary dysfunction and arrest secondary to compression of the medulla and cervical cord, induced by the positional insult and head trauma [32].

Wolf *et al.* (1998)

De novo findings of CM-1 during autopsies were reported in a deceased 71-year-old and 22-year-old, following two unrelated events of minor head trauma [33].

James (1995)

A de novo finding of CM-1 and syringomyelia during an autopsy were reported following the death of a 25-year-old man after sustaining a blow to the face and collapsing. No gross or microscopic evidence of fresh brain or spinal cord injury was found [34].

Tomaszeck *et al.* (1984)

A de novo finding of CM-1 during an autopsy was reported following the death of a (previously asymptomatic) 3-year-old child, two days after falling down the stairs and sustaining what appeared to have been at the time, a mild head injury [35].

Conclusion

Chiari malformation can be acquired or congenital, being an anomaly of the cerebellum, brainstem, and or cervico-occipital junction. In CM types 1–4, caudal herniation of the cerebellar tonsils (tonsillar ectopia) is present and may be accompanied by osseous defects. CM-1 and CM 1.5 malformations can develop congenitally due to overcrowding within the cranial vault due to various factors. Acquired defects involve cerebellar tonsillar herniation.

Blunt force or blunt trauma can result in Chiari malformations or can provoke a preexisting Chiari malformation. The symptoms and clinical manifestations of CM-1 are numerous and involve various systems, separately or concomitantly. Thus, the differential diagnosis is vital. Key points to remember in patients presenting with a history of blunt force trauma or acceleration-deceleration injuries are as follows:

- Patients with CMs can remain asymptomatic for years until a trigger (i.e., head or neck trauma) causes them to become symptomatic, i.e., head or neck trauma.
- There is a strong temporal relationship between trauma and radiographic progression of CM-1 malformations.
- Traumatic injuries to the head and neck may create a CSF leak resulting in spinal intrathecal hypertension and symptomatic progression.
- A transition from asymptomatic to symptomatic CM-1 can be presupposed as a result of CSF leak.
- The clinical appearance of Chiari malformations can progress over time.
- Patients can experience a change in the level of signs and symptoms over time.
- Patients with CM-1 can have minimal symptomatology and major diagnostic imaging features of the malformation, or vice versa.
- CM-1 can be diagnosed with or without syringomyelia. A small group of patients falls into the subcategory of CM-1.5.
- It is crucial to consider the differential diagnosis of Chiari 1.5 due to different treatment strategies as the incidence of unresolved syringomyelia after decompression surgery is nearly two-fold higher in CM-1.5 than CM-1, leading to repeat surgery.
- Neurological sequelae of CMs can include cranial nerve deficits and limb pain and weakness.

- There is an overlap of symptoms between cervical acceleration-deceleration (flexion-extension) injuries and Chiari malformation. Common shared features include headache, neck pain, shoulder pain, arm numbness and weakness, and cranial nerve neuropathies leading to vertigo and diplopia.
- Clinicians must consider both differentials when evaluating patients after head or neck trauma. It is vital to undertake a thorough clinical investigation to rule out such severe conditions as Chiari malformations.
- The neurological picture of CMs can progress and result in tetraplegia or death.
- Symptomatic patients may progress after major or minor physical trauma.
- It is not always clear whether the trauma is initiating or aggravating the symptoms.
- Patients who become symptomatic or whose symptoms progress after trauma should be monitored carefully for more than 24–48 hours to ensure there are no complications, especially concerning respiratory or cardiac systems.
- Chiari malformation can manifest as variations in otherwise healthy adults and adolescents.
- Sudden onset of symptoms may occur due to rapid enlargement or formation of a cervicothoracic syringomyelia defect secondary to aggravation of a possibly preexisting Chiari malformation.
- A Chiari malformation with syringomyelia may cause a patient to be sensitive to hyperextension of the atlantooccipital junction.
- It is imperative to include CM-1 as a differential diagnosis in patients presenting with neck pain, head pain, or cerebellar signs (after a closed-head injury, flexion-extension injury, or chiropractic manipulation), paraplegia, tetraplegia, lower cranial nerve dysfunction, or recurrent respiratory arrest.
- Biomechanical factors are fundamental considerations in the onset of symptoms of CM-1 malformations.
- A downward displacement of the cerebellar tonsils can be produced by the breakdown of the pressure equilibrium position between the supratentorial, infratentorial, and spinal spaces.
- Trauma can cause a supratentorial acute or chronic subdural hematoma, which can provoke the herniation of tonsils and initiate or worsen any symptoms.
- CM-1 should be included in the differential diagnosis of patients presenting with upper extremity weakness, lower cranial nerve dysfunction, or cerebellar signs after experiencing blunt trauma.
- Sudden death has been reported in persons with known and unknown Chiari malformations following blunt trauma.

Chiari malformations have many faces and facets to consider in presentation, especially in association with blunt force trauma and motor vehicle injuries. Enhanced awareness of these presentations and astute differential diagnosis are vital in treating patients presenting with such histories and symptoms, and in many cases may save their lives.

Conflict of Interest Statement

The authors declare that this paper was written in the absence of any commercial or financial relationship that could be construed as a potential conflict of interest.

References

1. Cociasu I., *et al*. "Chiari malformation type I - case report and review of literature". *Romanian Journal of Neurology/ Revista Romana de Neurologie* (2016): 138-142. https://www.researchgate.net/publication/312497135_Chiari_malformation_type_I_-_case_report_and_review_of_literature

2. Urbizu A., *et al*. "Chiari Malformation Type I: A Case-Control Association Study of 58 Developmental Genes". *PLoS ONE* 8.2 (2013): e57241. https://www.ncbi.nlm.nih.gov/pubmed/23437350

3. National Institute of Neurological Disorders and Stroke (2013).

4. Kim IK., *et al*. "Chiari 1.5 Malformation: An Advanced Form of Chiari I Malformation". *Journal of Korean Neurosurgical Society* 48.4 (2010): 375-379. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2982921/

5. Milhorat TH., *et al*. "Chiari I Malformation Redefined: Clinical and Radiographic Findings for 364 Symptomatic Patients". *Neurosurgery* 44 (1999): 1005-1017. https://www.ncbi.nlm.nih.gov/pubmed/10232534

6. Kyoshima K., *et al*. "Syringomyelia without hindbrain herniation: tight cisterna magna. Report of four cases and a review of the literature". *Journal of Neurosurgery* 96 (2002): 239-249. https://www.ncbi.nlm.nih.gov/pubmed/12450289

7. Tubbs RS., *et al*. "Analysis of the posterior fossa in children with the Chiari 0 malformation". *Neurosurgery* 48 (2001): 1050-1055. https://www.ncbi.nlm.nih.gov/pubmed/11334271

8. Iskandar BI., *et al*. "The resolution of syringomyelia without hindbrain herniation after posterior fossa decompression". *Journal of Neurosurgery* 89 (1998): 212-216. https://www.ncbi.nlm.nih.gov/pubmed/9688115

9. Freeman MD., *et al*. "A case control study of cerebellar tonsillar ectopia (Chiari) and head/neck trauma (whiplash)". *Brain Injury* 24.7-8 (2010): 988-994. https://www.ncbi.nlm.nih.gov/pubmed/20545453

10. Graham A., *et al*. "An unusual cause of neck pain: Acquired Chiari malformation leading to brainstem herniation and death". *Journal of Emergency Medicine* 43.6 (2012): 1000-1003. https://www.ncbi.nlm.nih.gov/pubmed/21215551

11. Sakaki T., *et al*. "Is Chiari I Malformation in the Aged Initiated by Mechanical Factors?" *Neurologia Medico-Chirurgica* (2006).

12. Jones PM., *et al*. "Traumatic Brain Injury – Missed Diagnosis: A Case Report". *MOJ Clinical and Medical Case Reports* 4.4 (2016): 00101. https://medcraveonline.com/MOJCR/MOJCR-04-00101.php

13. Late v. "United States, Civil No. 1:13-CV-0756, Dist. Court, MD Pennsylvania. Wyatt v. Hydraulics. Court of Appeals of North Carolina 768 S.E.2d 150 (N.C. Ct. App. 2014) (2015).

14. Payner TD., *et al*. "Acquired Chiari malformations: incidence, diagnosis, and management". *Neurosurgery* 34.3 (1994): 429-434. https://www.ncbi.nlm.nih.gov/pubmed/8190217

15. Wan MJ., *et al*. "Conversion to symptomatic Chiari I malformation after minor head or neck trauma". *Neurosurgery* 63.4 (2008):748-753. https://www.ncbi.nlm.nih.gov/pubmed/18981886

16. Benner K. "Chiari Malformation". Master of Science in Nursing (MSN) Student Scholarship (2017): 247.

17. Fischbein R., *et al*. "Patient-reported Chiari malformation type I symptomsand diagnostic experiences: a report from the national Conquer Chiari Patient Registry database". *Neurological Sciences* 36.9 (2015): 1617-1624. https://www.ncbi.nlm.nih.gov/pubmed/25972139

18. Robertson EK and Stanley C. "Type 1 Chiari malformation". *Journal of Orthopaedic and Sports Physical Therapy* 42.5 (2012): 483.

19. Worth DR and Milanese S. "Chiari Malformation Type I, presenting as scapulothoracic pain: A case report". *Manual Therapy* 17 (2012): 172-174. https://www.ncbi.nlm.nih.gov/pubmed/22245421

20. Makela JP. "Arnold-Chiari malformation type I in military conscripts: symptoms and effects on service fitness". *Military Medicine* 171.2 (2006): 174-176. https://academic.oup.com/milmed/article/171/2/174/4577880

21. Ajis A and Dharmarajah R. "Prolonged recovery of balance after a minor head injury as the only presenting feature of Chiari 1 malformation". *British Journal of Neurosurgery* 18.4 (2004): 398-400. https://www.ncbi.nlm.nih.gov/pubmed/15702845

22. Green JA. "Update of Chiari malformation: clinical manifestations, diagnosis, and treatments". *Pediatric Nursing Journal* 29 (2003): 331-335. https://www.ncbi.nlm.nih.gov/pubmed/12956559

23. Chandran V., *et al*. "Scapular winging in a patient with Arnold–Chiari malformation type 1 and syringomyelia". *BMJ Case Reports* (2014): 2014203571. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3975549

24. Tubbs RS and Oakes. "Winged scapula as the presenting symptom of Chiari I malformation and syringomyelia". *Child's Nervous System* 23 (2007): 255. https://www.ncbi.nlm.nih.gov/pubmed/17058083

25. Mehta AI., *et al*. "Radiographic progression of a Chiari I malformation after minor head trauma: Final increment of obstruction to create pathophysiology". *Journal of Surgical Radiology* 2.3 (2011): 290-293. https://scholars.duke.edu/display/pub774398

26. Uzoigwe CE., *et al*. "Simple whiplash?" *The Journal of bone and joint surgery British* 91.8 (2009): 1103-1104. https://www.ncbi.nlm.nih.gov/pubmed/19651845

27. Murano T and Rella J. "Incidental finding of Chiari I malformation with progression of symptoms after head trauma: Case report". *The Journal of Emergency Medicine* 30.3 (2006): 295-298. https://www.ncbi.nlm.nih.gov/pubmed/16677981

28. Süleyman B., *et al*. "Presentation of Chiari malformation after craniocervical trauma: A case report". *Turkish Neurosurgery* 3.9 (1999): 3-4.

29. Sakai. "Radiographic progression of a Chiari I malformation after minor head trauma: Final increment of obstruction to create pathophysiology". *Journal of Surgical Radiology* 2.3 (2009): 290-293.

30. Mampalam, T.J., *et al*. "Presentation of type I Chiari malformation after head trauma". *Neurosurgery* 23.6 (1988): 760-762. https://www.ncbi.nlm.nih.gov/pubmed/3216976

31. Kara., et al. "Unexpected Brain Death in a 20-year-old Man with Chiari Type I Malformation after Trauma: Case Report and Review of Literature". American Journal of Medical Case Reports 4.4 (2016): 140-143. https://www.researchgate.net/publication/301699731_Unexpected_Brain_Death_in_a_20-year-old_Man_with_Chiari_Type_I_Malformation_after_Trauma_Case_Report_and_Review_of_Literature

32. Zhang J., *et al*. "Sudden Unexpected Death due to Chiari Type I Malformation in a Road Accident Case". *Journal of Forensic Sciences* 58 (2013): 540-543. https://www.ncbi.nlm.nih.gov/pubmed/23278920

33. Wolf D., *et al*. "Death Following Minor Head Trauma in Two Adult Individuals with the Chiari I Deformity". *Journal of Forensic Sciences* 43.6 (1998): 1241-1243. https://www.ncbi.nlm.nih.gov/pubmed/9846405

34. James DS. "Significance of chronic tonsillar herniation in sudden death". *Forensic Science International* 75 (1995): 217-223. https://www.ncbi.nlm.nih.gov/pubmed/8586346

35. Tomaszek DE., *et al*. "Sudden death in a child with an occult hindbrain malformation". *Annals of Emergency Medicine* 13 (1984): 136-138. https://www.ncbi.nlm.nih.gov/pubmed/6691617

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