

Case Report

Cerebellar mutism following head trauma: A case report and literature review

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ABSTRACT

Background: Cerebellar mutism (CM) is defined as the lack of speech production, despite an intact state of consciousness and cognitive function, that happens secondary to a cerebellar insult. To the best of our knowledge, only five cases have thus far been described in the English literature. In this paper, we report the sixth incidence overall, which is also the first case of a CM associated with penetrating head injury. The relevant literature is reviewed and analyzed, our current knowledge of the neuroanatomical and functional relations is summarized, and potential future research endeavors are indicated.

Case Description: An 8-year-old girl was transferred to our hospital having had fallen on a rod that penetrated her neck behind the ear. An urgent computed tomography scan of the head revealed a right cerebellar contusion with surrounding edema. Three days later, she became mute but was still obeying commands. Repeat imaging showed a resolving cerebellar contusion with increased edema and mass effect. By day 9, she had uttered a few words. At 1-month follow-up, the child had regained normal speech.

Conclusion: Posttraumatic CM is a rare and probably underreported condition with only six documented cases to date. Although it may well be on the same spectrum as postoperative CM, further understanding of the exact mechanism, clinical course, and prognosis of this entity is bound to significantly improve the recovery and quality of life of head trauma patients.

Keywords: Cerebellar mutism, Head trauma, Penetrating traumatic brain injuries

INTRODUCTION

Cerebellar mutism (CM) is defined as the lack of speech production, despite an intact state of consciousness and cognitive function, that happens secondary to a cerebellar insult. Conventionally, the extension to this definition is “the absence of evidence for supra or nuclear cranial nerve or long tract injury.” However, with our current limited understanding of the anatomical substrate for the condition, this extension remains subject to alterations.^[9] CM is most commonly a complication of posterior cranial fossa surgery, with an average reported incidence of 2–40% – in the pediatric age group.^[14] It has also been uncommonly described in conjunction with other etiologies, including infection^[11] and vascular events.^[5] CM following

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traumatic brain injury is an exceedingly rare occurrence. To the best of our knowledge, only five cases have thus far been described in the literature.^[4,6,8,9,15] In this paper, we report the sixth incidence overall. Interestingly, all previous cases were reported in association with a closed head injury. This is the first case of a CM associated with penetrating cranial trauma. The relevant literature is reviewed and analyzed, our current knowledge of the neuroanatomical and functional relations is summarized, and potential future research endeavors are indicated.

CASE DESCRIPTION

An 8-year-old girl was transferred to our hospital having sustained a penetrating head and neck trauma. According to her parents, she was climbing on a side of a water tank outside when she fell and landed on a steel rod. In the emergency department, the patient was conscious, oriented, and screaming. She had a right-sided retromandibular penetrating wound.

Her other findings included right-sided lower motor neuron facial nerve palsy, neck spasm (torticollis), and right-sided cerebellar signs, namely, horizontal nystagmus, impaired finger to nose test, and dysdiadokokinesia were elicited.

A computed tomography (CT) scan of the head revealed a skull base fracture through the floor of the posterior cranial fossa in association with right cerebellar contusion and edema. A cervical CT scan showed evidence of right parotid gland injury with surrounding hematoma (images not available).

Three days after admission, she became mute but was still obeying commands. Repeat imaging showed a resolving cerebellar contusion with increased edema and mass effect [Figures 1 and 2].

On the 7th admission day, she was ambulatory but with an ataxic gait and still could not speak. By day 9, she had spoken a few words and her neurological examination started to improve with a stationary facial exam. At her 1-month follow-up visit, the child had regained normal speech. Her facial palsy, which had persisted, was deemed secondary to direct injury to the extraforaminal segment of the facial nerve.

DISCUSSION

We reported a case of a child who was temporarily mute after penetrating trauma to the posterior fossa. The mental status and cognition of the patient remained intact throughout. Typical CM is characterized by an intact cranial nerve function. In this case, the patient had an injury to the parotid gland resulting in a coexisting CN VII palsy.

CM is characterized by a specific onset and chronology. It is usually diagnosed following a period of latency (1–6 days

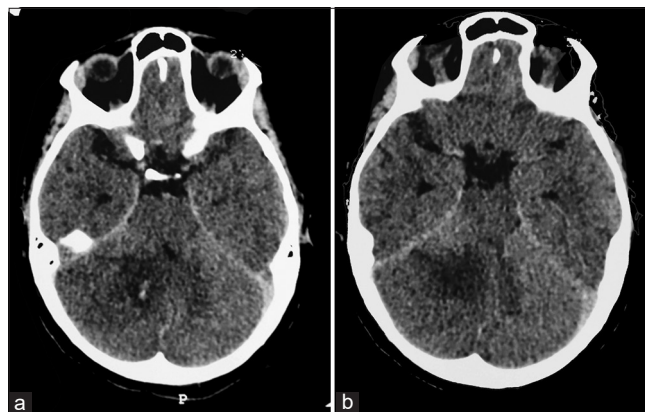


Figure 1: (a and b) Noncontrast computed tomography scan of the brain showing right cerebellar resolving contusions with edema that causing mass effect.

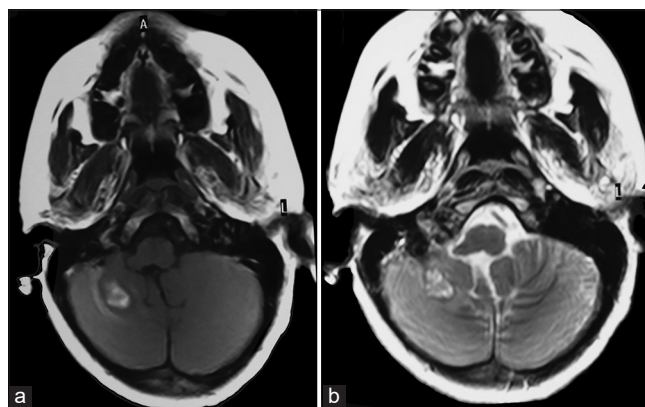


Figure 2: (a and b) Magnetic resonance imaging T1- and T2-weighted images, respectively, showing right cerebellar contusion without midline injury.

after the initial insult), with an average duration of 1 day–4 months. It follows a variable recovery path after that before the subsequent gradual return of verbalization. In its typical form, CM is a temporary condition. However, the resumption of baseline speech function follows a variable course, with residual neurological deficits such as persistent dysarthria, ataxia, and behavioral changes being infrequently documented.^[7] In our case, mutism began on the 4th day and continued for 5 days, and then gradually began to improve until the full recovery after 1 month. Notably, our patient demonstrated signs of cerebellar syndrome, with a concordance with the onset and resolution of her mutism.

As for the neuropathological and anatomical coordinates of CM, a number of studies have been published, with most data coming from postsurgical cases. A wide range of theories has thus been put in place and most of them focus on specific anatomical areas. For example, the involvement of median and paramedian structures,^[3] splitting of the

vermis,^[2] bilateral cerebellar injury,^[12] and transient neuron dysfunction of the A9 to A10 dopaminergic cells in the mesencephalon have all been proposed as potential mechanisms.^[1]

More recently, the involvement of particular tracts regardless of the specific anatomical areas *per se* has been identified as the most plausible hypothesis. Specifically, a bilateral disruption to the dentate-thalamocortical tract by ischemia and/or edema and the resultant cerebello-cerebral diaschisis has been cited by multiple authorities as the underlying mechanism.^[7,13] The latter explanation is consistent with the literature findings, where the heterogeneous anatomical location of the injuries was apparent. In our case, the involvement of the right cerebellar hemisphere fits into the conclusions of functional studies that tell us that the right cerebellar hemisphere plays a role in language production.^[10,14] Furthermore, the fact that the CM coincided with the resolution of contusion and onset of cerebral edema points to the latter as the cause of a bilateral disruption of the circuit between the cerebrum, the dentate nucleus, and the thalamus. However, to better understand the underlying neuroanatomical basis of CM, more tractography studies are needed.

The first case of CM was reported in 1985 by ReKate *et al.*^[12] who described six cases of children with transient muteness following posterior fossa craniotomy for tumor removal. Since then, several similar cases have been identified. In this review, we focused on CM following head trauma, an extremely rare entity, of which only five cases have been documented, none of which was associated with a penetrating injury. The first case of a CM following head trauma was reported in 1990 by Yokota *et al.*^[15] who described the case of a 6-year-old boy who became mute after a road traffic accident (RTA). The authors attributed the CM in

this case to an injury to the cerebellar vermis or left cerebellar hemisphere. Two cases were then documented in 1997; Koh *et al.*^[9] reported a case of cerebellar muteness following an RTA with the injury located at the left cerebellar hemisphere (small contusion) and left cerebellar peduncle (small focal hemorrhage). In this particular case, a latency period of 44 h was recorded and the patient resumed normal speech after 25 days. Ersahin *et al.*^[4] described a case of cerebellar muteness in a 2.5-year-old boy who had fallen from a height. CT imaging revealed a hematoma in the right paravermian region and this patient regained normal speech function in 2 months. In 2005, Fujisawa *et al.*^[6] reported a case of CM following the evacuation of an acute subdural hematoma of the posterior fossa in a 7-year-old male who was involved in an RTA. The patient spoke normally after 39 days. Kariyattil *et al.*^[8] have recently reported a similar case. This was a 6-year-old boy with contusions of the cerebellar vermis and left cerebellar hemisphere following an RTA [Table 1].

Although it is difficult to draw conclusions from such a small number of cases, a number of observations may be made here: first, the scarcity of cases and the unknown time of onset in most instances reflect the possible lack of reporting of such cases, partly due to the intubated, sedated status of trauma patients. The second observation is that six cases have so far been reported in the pediatric age group reflecting the age distribution of surgical CM. Third, there is a lack of evidence in long-term follow-up studies, with none of the patients undergoing any formal neurocognitive testing. Forth, the indistinct anatomical location of cerebellar injuries solidifies the current school of thought on the neuroanatomical coordinates of CM. Further anatomical, functional, and clinical studies are needed to better understand the basis and prognosis of CM.

Table 1: A review of the literature on cases with CM following trauma.

Authors	Year	No. of cases	Age	Gender	Trauma mechanism	Location	Time to onset	Time to resolution
Yokota <i>et al.</i> ^[15]	1990	1	6 years	M	RTA	Injury to cerebellar vermis or left cerebellar hemisphere	NS	Unknown
Ersahin <i>et al.</i> ^[4]	1997	1	2.5 years	M	FFH	Hematoma in the right paravermian region.	NS	2 months
Koh <i>et al.</i> ^[9]	1997	1	Pediatric (age not specified)	N/A	RTA	Left cerebellar hemisphere (small contusion) and left cerebellar peduncle (small focal hemorrhage)	44 h	25 days
Fujisawa <i>et al.</i> ^[6]	2005	1	7 years	M	RTA	ASDH in posterior fossa	NS	39 days
Kariyattil <i>et al.</i> ^[8]	2014	1	6 years	M	RTA	Contusion of the cerebellar vermis and left cerebellar hemisphere	NS	Unknown

RTA: Road traffic accident, CM: Cerebellar mutism

CONCLUSION

We have reported the first case of a CM following a penetrating head injury. Posttraumatic CM is a rare and probably underreported condition with only six documented cases to date. Although it may well be on the same spectrum as postoperative CM, further understanding of the exact mechanism, clinical course, and prognosis of this entity is bound to significantly improve the recovery and quality of life of head trauma patients.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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