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CEREBELLAR MUTISM FOLLOWING AN INTERNAL HEAD INJURY: REPORT OF A RARE CASE FROM SOUTH INDIA

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ABSTRACT

The muteness in an otherwise cognitively normal individual following cerebellar lesion is known as cerebellar mutism. Over 400 cases have been documented since 1985 but almost all occur as a result of posterior fossa surgeries rather than closed head trauma. The principal cause of cerebellar mutism is due to bilateral interruption of the dentato–thalamo–cortical pathway (DTC). This is a case report of a 3 year old male child who suffered from a closed head injury involving the right lobe of cerebellum. He later developed mutism and recovered spontaneously. Due to its atypical presentation, we reviewed articles to conclude on a pure clinical diagnosis and throw light into a probable variant form of cerebellar mutism in children.

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INTRODUCTION

Cerebellar mutism has been studied since 1985.[1] Mutism followed by posterior fossa surgery in pediatric age group was described in detail by Rekate et al.[2] It is also quoted by Yonemasu.[3] Over 400 cases has been documented since then but almost all as a result of posterior fossa surgeries rather than closed head trauma. Cerebellar parenchymal insults due to local perfusion defects, neurotransmitter release dysregulation, edema, axonal injury and cerebral perfusion defects will also give rise to clinical mutism. Hence it need not occur post operatively alone.[1-5] It is a collective opinion by many authors that the principle cause of cerebellar mutism is due to bilateral interruption of the dentato–thalamo–cortical pathway (DTC).

This is a case report of a 3 year old male child who sustained contusion in the right lobe of cerebellum and presented with features of mutism.

CASE REPORT:

3 year old male child presented to the ER (emergency room) in a semiconscious state following a fall from 10 feet height at home. Time lapse between the incident and the medical attention was noted to be 4 hours. Patient was irritable and spoke irrelevantly. No external wounds were present and there was no other gross neurological deficits on primary evaluation.

Further evaluation revealed stable vitals with a GCS of E3V4M5 (12/15). A plain Computerized Tomography of the brain was taken and shifted to the neurosurgical intensive care unit for further management. CT brain revealed contusion involving the right cerebellum with no apparent cranial fractures (Fig.1and 2). No other haemorrhage or parenchymal injury was noted.

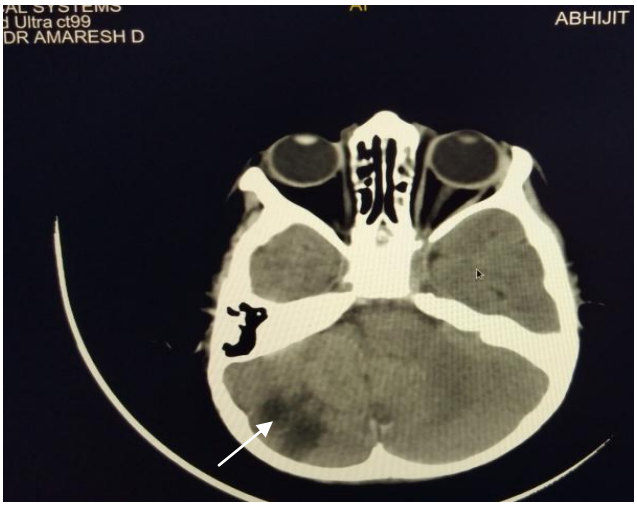

Fig 1: DAY-1
Right cerebellar lobe showing features of contusion. No active parenchymal bleeds or bony fractures noted.

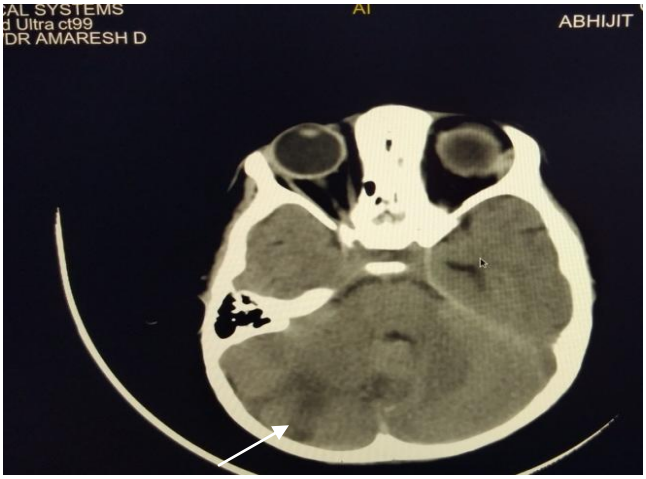

Fig2: DAY- 3
Repeat CT brain contusion reolved spontaneously.

Patient was treated conservatively with analgesics, fluids and anti-edema measures that was adjusted to the body weight as per pediatric dosage. On the second day of admission, patient was unable to communicate verbally. He did not show any signs of comprehension to stimuli except for blinking and looking at mother. This was continued till day 4 when he spontaneously regained speech and carried out normal interactions with the family members. Repeat CT brain (Fig.2) showed decrease in contusion and patient was discharged on day 6 with a GCS of E4V5M6 (15/15). During the stay in the hospital, all blood parameters like haemogram, serum electrolytes etc. were within normal limits. Patient was called for review on 10th day post injury and was found to be very active and responsive with fluent speech. He did not have any features of ataxia or dysarthria.

Due to the lack of resources and financial constraints from the patient's side, a detailed speech analysis and higher brain imaging like MRI (Magnetic resonance imaging) could not be carried out.

DISCUSSION:

The muteness in an otherwise cognitively normal individual following cerebellar lesion is known as cerebellar mutism. It has a delayed onset (mean time=1.7 days) upto 4 months (mean duration= 7-8 weeks). Cerebellar mutism often seen among the patients/children who underwent posterior fossa surgery.[1] The sustained mutism tends to recover spontaneously over a short period of time, without any active intervention. [2] Manipulation of the site results in the damage of dentate-thalamo-cortical pathway as mentioned earlier. Involvement of right cerebellar hemisphere in trauma is very important as this part of the brain is responsible for language and cognitive tasks, whereas the left half is responsible for spatial and executive tasks. [1] It is due to the involvement of nerve fibers travelling via the symmetrically located dentate nuclei in the paravermal region at the VIth and VIIth lobules in cerebellum. Via pons, primary and supplementary motor area (SMA) of cerebrum afferent fibers reach this nuclei along with afferents from the lateral hemispheres of the cerebellum .The efferent fibers from here go up to the superior and middle peduncle and the brainstem, change track to the opposite ventro-lateral nucleus of the thalamus (VL thalamus) to end in the motor and prefrontal cortex. Dentate nuclei are also interconnected directly at the Mollaret's triangle in the midline. Hence the initiation of voluntary movements and higher brain functions are the result of this system. It is emphasized in

cerebellar mutism that injury to bilateral dentate nuclei is symptomatic.[4-6]

This case had the clinical features of cerebellar mutism following cerebellar contusion after an internal head injury involving right cerebellum. It is a rare occurrence in the absence of surgical intervention involving the structures in posterior fossa. Other associated clinical features include ataxia, hypotonia, cranial nerve palsies, hemiparesis and emotional lability.[3] This case did not have any residual neurological deficits. Formal speech and language tests such as Diagnostic Instrument Apraxia of Speech (DIAS), etc. are indicated in the follow up period to ascertain the cognition and higher mental functions.[4] This detailed speech analysis using various scoring systems, interventional monitoring and brain imaging other than CT could not be done due to lack of trained personnels. It plays a major role in motor, cognitive, and social–behavioral development, possibly via modulatory effects on the developing cerebral cortex.[7] A breech in connection between the right cerebellumand left frontal cortex results in poor verbal comprehension in children diagnosed with cerebellar mutism.[8] Proper evaluation is mandatory for further management. Mode of injury could be following post operative, post radiation-damaging white matter or trauma that may lead to hypoxic insult of the cerebellar tissue, dysregulation of neurotransmitter release, diffuse axonal injury,etc. [9] Often these children, in the post trauma phase develop behavioral abnormalities and memory impairment which may surface as poor scholastic performance. This is attributed to the early age of cerebellar injury. [7] It is stated that children diagnosed with autism do show defects in the cerebellar pathways.[10]

Aphasia with gradual restoration of verbal response and quick recovery within 10days lead to the probable clinical diagnosis of cerebellar mutism in this case.

CONCLUSION:

Cerebellar mutism is classically seen following posterior fossa surgeries and various other causes were previously described. Patients may present with mutism even in the absence of a surgical intervention. It may be due to the damage to cerebellar parenchyma following trauma, vascular injury or infection.[2] This case report is suggestive of mutism in a 3 year old boy with internal head injury following a fall. He recovered spontaneously without any neurological deficits. Early recovery could be attributed to less involvement/ damage to the cerebellum and visiting the hospital soon after the injury.

In the absence of adequate literature for cerebellar mutism following trauma, this study has been built on the strong clinical suspicion and available monitoring tools. There need to be extensive research on the atypical etiologies for mutism is children.

Conflict of Interest Statement-

There is no conflict of interest.

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