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Case report

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An Unusual Postoperative Outcome in Posterior Fossa Surgery: Cerebellar Mutism Syndrome Case Report

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Abstract

Background: Cerebellar mutism is an unusual complication of posterior fossa surgery, particularly within children, whose diagnosis in the postoperative period is delicate. ***Case Report:*** A 4-year-old child underwent surgery for a 4th ventricle medulloblastoma. Postoperatively, he developed total mutism with a lack of contact but appropriate reactions to external stimuli. Hypoglossal nerve damage led to persistent swallowing difficulties. Brain MRI showed no compressive elements or residual tumor. After one month, he gradually regained contact, initially with his mother and others. By the second month, he began speaking with dysarthria. Speech therapy led to significant improvement. ***Conclusion:*** This case report highlights the importance of early diagnosis of cerebellar mutism to ensure optimal management and improve prognosis. Its exact pathophysiology is still poorly specified in the literature. Postoperative rehabilitation is the cornerstone of treatment, knowing that certain medications have been tested but without great efficiency.

Keywords: ***Cerebral mutism, posterior fossa surgery, diagnosis.***

Introduction

Cerebellar mutism syndrome (CMS) is a rare complication of posterior fossa surgery, particularly in children. It was first described in 1917 [1] with the victims of the First World War. Since then, numerous articles have attempted to establish the pathophysiology of this syndrome, which remains ambiguous to this day.

We report the case of a child operated on for a tumor of the posterior cerebral fossa, who presented with CMS post-operatively with good evolution.

Case Reports

A 4-year-old boy with no medical history and good psychomotor development was admitted to the neurological intensive care unit for postoperative management of the 4th ventricle tumor resection. The patient had undergone ventriculocisternostomy 17 days before surgery, in response to signs of intracranial hypertension secondary to the cancer. The surgical procedure was uneventful, besides a tachycardia at 130 linked to hypovolemia secondary to blood loss estimated at 600 cc. So, he received 2 packed red blood cells and 3 fresh frozen plasma at the end of the procedure. A total tumor removal was achieved. The extemporaneous examination revealed a medulloblastoma.

The postoperative course was marked by persistent swallowing difficulties secondary to hypoglossal nerve damage, necessitating a tracheotomy on day 5.

On day 6, the clinical examination revealed total mutism, with no contact, even with the mother. However, his reactions to external stimuli were appropriate but without command execution. Given this, a cerebral CT scan was performed, showing no compressive element in the posterior cerebral fossa, tumor residue, or supratentorial lesion. The metabolic workup and cerebrospinal fluid (CSF) analysis were normal. Over time, calcium levels increased from 84 mg/L on day 6 to 89 mg/L on day 10, while sodium levels remained stable (144 mmol/L to 145 mmol/L), and phosphorus levels slightly decreased (28 mg/L to 26 mg/L). cerebrospinal fluid (CSF) analysis was negative with white blood cells 3 elements/mm³, red blood cells 500 elements/mm³, and cultures remaining negative.

After respiratory weaning, the child was transferred to the neurosurgery department.

One month later, the child presented a progressive resumption of contact, initially with the mother and then with his environment, with order execution. At the end of the 2nd month, he began to speak, but with marked dysarthria. A follow-up brain MRI revealed no lesions or residues in the posterior cerebral fossa (Figure 2). The patient received several speech therapy sessions, with good progress. Then, he was referred to the Pediatric Hematology and Oncology Center for further treatment.

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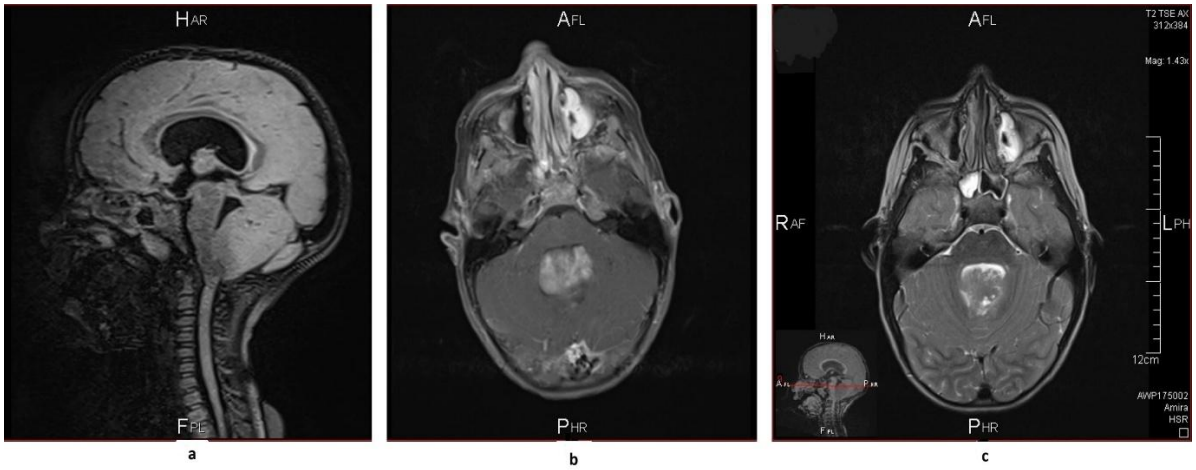

Figure 1: Preoperative brain MRI showing tumor in 4th ventricle in FLAIR (a), T1 with gadolinium injection (b) and T2 (c) sequences

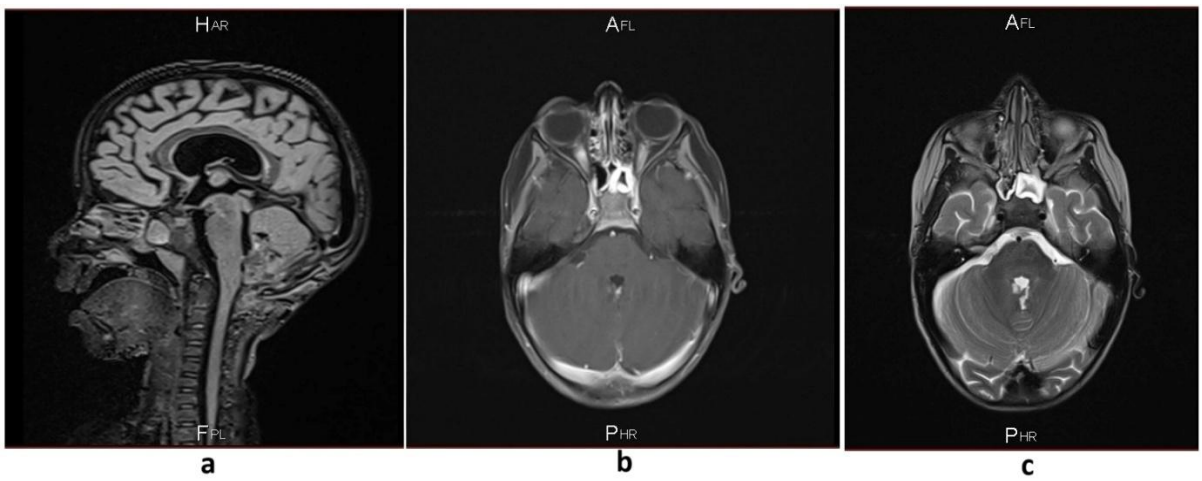

Figure 2: Postoperative brain MRI showing no residual tumor in Flair (a), T1 with gadolinium injection (b) and T2 (c) sequences

Discussion

Several definitions and terms tried to define and describe this Cerebellar mutism syndrome (CMS). Examples include posterior fossa syndrome, cerebellar mutism syndrome, cerebellar cognitive-affective syndrome, and transient cerebellar mutism [2]. In 2016, after the work of "The Posterior Fossa Society" group, a consensus was published to define this clinical entity [3]. Thus, postoperative cerebellar mutism syndrome is characterized by late-onset mutism or reduced speech associated with neurobehavioral disorders and occurs after posterior fossa surgery in children. It causes hypotonia, oropharyngeal dysfunction, or cranial nerve damage. It occurs generally between the first and sixth day postoperatively, with a gradual recovery from 1 to 6 months [2].

The incidence varies from 11 - 29% in children, but less than 1% in adults [4]. This may be explained by cerebral immaturity in the pediatric population, with incomplete myelination of axons linking the cerebellum to the thalamus and higher centers [1]. A second factor that may explain this difference is that posterior cerebral fossa tumors are much more frequent in children, and consequently the incidence becomes higher.

Several pathophysiological theories have attempted to explain the mechanisms responsible for this syndrome, but none has yet been validated [5]. Dentothalamic bundle damage theory is mostly accepted by experts [5]. It is responsible for disrupting the transmission of nerve impulses from the cerebellum to the cerebral cortex, leading to its activity reduction. This bundle damage could be caused by cerebro-cerebellar diaschisis. It is a sudden functional inhibition of a part of the brain located at a distance from a cranial lesion that needs to have direct or indirect anatomical or physiological links with the injured area. This has been demonstrated by several studies [5,6] that showed hypoperfusion of the frontal cortex on brain MRI in patients with cerebellar mutism, which regressed after recovering a basic neurological state. Other mechanisms also exist, such as post-operative edema, altered cerebellar perfusion linked to vasospasm [3], or damage to the vermis.

Data on therapeutic management is poor and is limited to some case reports. Several medications have been tested [6]: bromocriptine, fluoxetine, haloperidol, risperidone, delorazepam and zolpidem. Their benefits generally appear within the first 24 hours, but full recovery takes several months, making it difficult to assess the real contribution of these medications to recovery [6].

Post-operative rehabilitation is pivotal to managing CMS and helps improve the patient's functional prognosis. It must address three main areas: speech, cognitive behavior, and movement coordination. Collaboration between several professionals - the physiotherapist, the speech therapist, the neurologist, the psychologist, and the neuropsychiatrist - is essential to ensure comprehensive, optimal care.

Conclusion

CMS is a poorly understood complication of posterior fossa surgery. Early recognition and proper management are essential to improve patient outcomes. Some neurocognitive and motor sequelae may persist after the resolution of the mutism. Hence, further research is needed to understand better the underlying physiopathology and to develop strategies to prevent CMS.

Declaration

Consent of Publication

The author obtained written informed consent from the patient's family to submit this manuscript for publication.

Source of funding

None

Conflicts of Interests

The authors report no conflict of interest.

Availability of Supporting Data

Available on corresponding author upon responsible request

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None

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