

Postoperative pediatric cerebellar mutism syndrome: A literature review

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Introduction: Postoperative pediatric cerebellar mutism syndrome (POPCMS) is a significant complication observed after posterior fossa surgery for tumors, particularly in pediatric neurosurgery. This paper presents a literature review on the pathophysiology, diagnosis, treatment, and long-term outcomes of POPCMS.

Methods: A systematic review following PRISMA guidelines, searching for articles published in PubMed, Web of Science, and Lilacs in the last ten years. Inclusion criteria involved studies on human subjects under 18 years of age, focusing on the pathophysiology, diagnosis, treatment, and outcome of POPCMS.

Results: The review identified 34 studies on POPCMS for final inclusion. Preoperative risk factors commonly associated with cerebellar mutism included midline tumor location, diagnosis of medulloblastoma, younger age at diagnosis, preoperative language impairment, brainstem infiltration, and tumor histology. The SHH subgroup was found to reduce the risk of CMS. Diagnostic criteria for POPCMS included language impairment, emotional lability, hypotonia, oropharyngeal dysfunction, dysphagia, cerebellar motor syndrome, cerebellar cognitive affective syndrome, and brainstem dysfunction. Treatment approaches varied, with medications showing potential benefits. Rehabilitation options for speech and language deficits were limited. Cognitive remediation programs showed promise in improving academic achievement and attention. A significant proportion of patients experienced clinical improvement.

Conclusion: POPCMS remains a challenging complication following posterior fossa surgery. Further research is needed to develop standardized diagnostic criteria, effective treatments, and rehabilitation strategies to improve long-term outcomes for patients with POPCMS.

Keywords: mutism, speech, pediatric neurosurgery, posterior fossa, brain tumor, rehabilitation

INTRODUCTION

Posterior fossa lesions surgery pose significant challenges for neurosurgeons, especially in pediatric neurosurgery. Among the various complications commonly encountered in neurosurgical procedures, cerebellar mutism stands out as one of the most feared [1]. Recently, an international working group reached a consensus and introduced the term "postoperative pediatric cerebellar mutism syndrome" (POPCMS) to eliminate confusion about other disorders that share similar clinical features in children and adults. This term replaces previous designations such as posterior fossa syndrome, cerebellar mutism, and cerebellar cognitive affective syndrome [2]. This paper aims to present a recent literature review on the pathophysiology, diagnosis, treatment and Long-term outcomes of POPCMS

MATERIALS AND METHODS

Following the PRISMA guidelines [3], a search considering the articles published in PubMed, Web of Science and Lilacs in the last 10 years regarding POPCMS was undertaken using the following constructs: "cerebellar mutism" and "posterior fossa tumor". Studies on adults focused only on imaging and case series were excluded.

Inclusion criteria were limited to articles published within the last 10 years (from 2013 to 2023), involving human subjects under 18 years of age, and providing insights into the disease's pathophysiology, diagnosis, treatment, and outcome.



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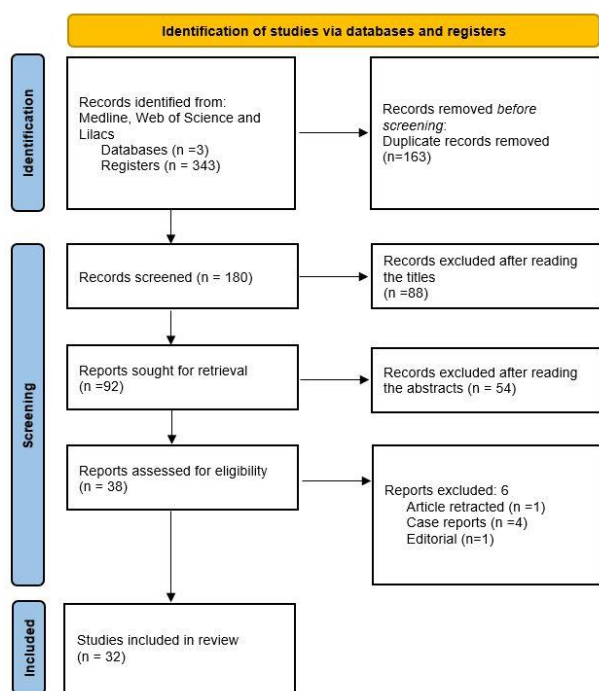


Figure 1 - PRISMA 2020 flow diagram for postoperative pediatric cerebellar mutism syndrome

RESULTS

Preoperative risk

Six review articles focused on the preoperative risk for cerebellar mutism and were included in our study.

Risk factors most commonly associated with development of CMS include midline tumor location, diagnosis of medulloblastoma and specific tumor subtype, younger age at diagnosis, and preoperative language impairment [4].

Left-handedness, and disruption of the connection between the right cerebellum and left frontal cortex have been associated with the occurrence of cerebellar mutism syndrome. Brain stem infiltration and tumor histology (medulloblastoma) also appear to be frequently associated with the development of cerebellar mutism syndrome [5]. Other authors believe that brainstem involvement is the most important, including preoperative tumor invasion or postoperative edema [6].

In a recent metanalysis, statistically significant risk factors identified from univariate analysis were brainstem invasion (OR 4.28, 95% CI 2.23–8.23; $p < 0.0001$), fourth ventricle invasion (OR 12.84, 95% CI 4.29–38.44; $p < 0.00001$), superior cerebellar peduncle invasion (OR 6.77, 95% CI 2.35–19.48; $p = 0.0004$), diagnosis of medulloblastoma (OR 3.26, 95% CI 1.93–5.52; $p < 0.0001$), medulloblastoma > 50 mm (OR 8.85, 95% CI 1.30–60.16; $p = 0.03$), left-handedness (OR 6.57, 95% CI 1.25–34.44; $p = 0.03$), and a vermis incision (OR 5.44, 95% CI 2.09–14.16; $p =$

0.0005). On the other hand, a tumor located in the cerebellar hemisphere (OR 0.23, 95% CI 0.06–0.92; $p = 0.04$), cerebellar hemisphere compression (OR 0.23, 95% CI 0.11–0.45; $p < 0.0001$), and intraoperative imaging (OR 0.36, 95% CI 0.18–0.72; $p = 0.004$) reduced the risk of pCMS [7].

It is interesting to note that several studies have recently identified the SHH subgroup as a significant factor in reducing the risk of post-operative CMS [8].

In a multivariable analysis, WNT type, Group 3 and Group 4 tumors were found to be independently associated with higher risk compared with sonic hedgehog tumors [9].

Preoperative language impairment has also been implicated. More precisely, 28.5% of children suffering from preoperative language impairment and developed cerebellar mutism postoperatively [10].

Transvermian surgical approach compared with telovelar was not found as a risk factor. This discrepancy can be partially explained by two factors: first, the telovelar approach requires greater technical skill, and is therefore practiced by experienced surgeons; and second, the transversian approach is preferred in the presence of large tumors, a well-established risk factor, introducing an additional bias. In fact, in another prospective study, surgery in a low-volume surgery center were found to increase the likelihood of CMS [5, 8, 10].

Walker et al. introduced a score model to predict the risk and included six relevant factors. These included primary tumor location, as it was specified by MRI, bilateral middle cerebellar peduncle involvement (invasion and/or compression), dentate nucleus invasion, and age at surgery > 12.4 years [10].

Sex, meningitis, and preoperative hydrocephalus are generally considered as insignificant risk factors [6].

Physiopathology

In this revision, twelve articles focused on the physiopathology of cerebellar mutism and were included in our study.

The cerebellum is classically involved in maintaining balance and posture and coordinating voluntary movements. However, there is recent converging evidence of the cerebellum's role in cognition and its implication in psychopathology as verbal working memory and articulatory subvocal rehearsal [11].

Structural and functional MRI data confirms a basic dichotomy in the human cerebellum, with sensorimotor functions in the anterior cerebellum and cognitive, emotional, and limbic functions in the posterior cerebellum. For both functional areas, broad connectivity to supratentorial structures via the cerebellar peduncles was

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confirmed. The widespread connectivity and topography of the human cerebellum with cerebral areas have been implicated in non-motor functions [10, 12].

Injury to the bilateral dentatothalamocortical tract appears to be critical for the development of mutism [13]. Most cases of mutism are due to injury to cerebellar tracts and cerebellar-cerebral circuits, involving particularly distinct points of the dentate-thalamus-cortical and dentato-rubro-thalamus-cortical. Advanced neuroimaging techniques, such as tractography and perfusion studies, have contributed to demonstrating changes in these pathways in patients with pediatric cerebellar mutism [14].

Even though there seems to be convincing evidence for bilateral frontal perfusion deficits in POPCMS patients compared to non-POPCMS patients [15], the included article does not fully support the hypothesis of cerebello-cerebral diaschisis as a pathophysiological explanation of the syndrome for not including preoperative exams

Mutism itself is inherently related to supratentorial dysfunction, mediated by crossed cerebello-cerebral diaschisis. Some reports correlate behavioral disturbances in the pediatric population with cerebellar mutism with frontal cortex dysfunction [10]. Some authors have proposed that cerebellar mutism is actually a supratentorial disorder, caused by hypoactivity in the cerebral hemispheres due to disruption of its connections [16]. Agrammatical language is also present and has been associated with lesions that involve the right cerebellar hemisphere as well as the midline posterior vermis [17].

Fronto-cerebellar tractography in children with cerebellar mutism have demonstrated reduced volumes in the superior cerebellar peduncles and midline cerebellum in the group with mutism compared with controls. Language difficulties following cerebellar damage in pediatric populations include the anomia, difficulties with semantic and pragmatic language, and agrammatism [17].

Regarding dysarthria, its anatomic substrate has been proposed to be damage of the dentate and interposed nuclei. The posterolateral hemispheric region, along with the adjacent compartments of the dentate nuclei are considered to contribute to the linguistic process [4, 10].

Two pathophysiological models have been proposed. The first one describes mutism itself is mediated primarily via supratentorial dysfunction caused by crossed cerebello-cerebral diaschisis. The second states that clinical symptoms following mutism are mediated primarily by direct surgical injury of cerebellar and brainstem structures [13].

Some authors have proposed that thermal injury resulting from the use of the Cavitron Ultrasonic Aspirator (CUSA) is an important mechanism in the pathogenesis of CMS. The vulnerability of Purkinje cells to thermal injury and

the shared imaging features of heat stroke and PFS support the possibility of an underlying pathological component that is common to both conditions [18].

There is an interesting similarity with supplementary motor area syndrome and some authors have linked these conditions. Both syndromes include disturbances in speech and motor function. The causes of the syndromes are unknown; however, surgical damage to the dentato-thalamo-cortical pathway (DTCP) has been associated with CMS. The similarities of the two syndromes could be traced back to their mutual connection through the DTCP and their membership to a cerebro-cerebellar circuit [19].

Another entity frequently associated to CMS is hypertrophic olivary degeneration (HOD). It is a rare neuronal degeneration condition resulting from injury to the dentato-rubro-olivary pathway (DROP), also referred to as the Guillain-Mollaret triangle. HOD is a rare complication related after PF tumors surgery and symptoms may be misdiagnosed with pediatric cerebellar mutism syndrome. Children with HOD usually do not develop palatal tremor but ataxia is common. However, HOD and CMS are different pathological entities. HOD results from an injury to the dentato-rubro-olivary pathway while CMS to the dentato-thalamo-cortical pathway [20].

Diagnosis

Nine review articles focused on diagnosing cerebellar mutism and were included in our study.

Diagnostic criteria are not strict and universally adopted. These include language impairment and emotional lability, as proposed by the international Board of the Posterior Fossa Society in their consensus statement [4, 10]. Additional common features including hypotonia, oropharyngeal dysfunction and dysphagia. It is also frequently accompanied by the cerebellar motor syndrome, the cerebellar cognitive affective syndrome, and brainstem dysfunction including long tract signs and cranial neuropathies [21–23].

The incidence of CMS has been reported in 8% to 32% of children, and significant discrepancy is related to the high variability of definitions of CMS used by different authors[24]. A clear and accepted universal definition would help improve reporting, as would the application of agreed outcome measures [25].

A Survey of Experts in the clinical care of children with posterior fossa tumors to identify trends and discrepancies in diagnosing posterior fossa syndrome and created a set of “working” diagnostic criteria for POPCMS [26] (Table 1). A scale for diagnosis and scoring has been proposed, including the duration and severity of cardinal symptoms [27].

Table 1 - Proposed Diagnostic Criteria for POPCMS

Criterion A	Acquired cerebellar injury (e.g., postsurgical or stroke-related), with symptoms in Criteria B, and C or D, emerging within two weeks of injury
Criterion B	Presence of one of the following speech and/or language deficits: 1 Mutism (inability to speak) or 2 Significant impairment in language as indicated by one or more of the following: reduced phrase length (speech limited to single words or two- to three-word phrases), agrammatism, atypical speech rate/rhythm (slowed, gaited, ballistic), and/or dysnomia
Criterion C	Presence of notable changes in mood/affect characterized by irritability (excessive tearfulness, crying, agitation, or anger), emotional lability (rapid changes in mood), and/or flat affect
Criterion D	Presence of motor dysfunction defined as apraxia (inability to execute purposeful movements on command, despite having the physical capacity to perform the movement), ataxia (difficulty coordinating muscle movements), dysmetria (undershoot or overshoot of intended position with the hand, arm, or leg), hypokinesia (abnormally diminished motor activity), and/or hemiparesis (weakness one side of the body)

Note: Criteria A and B1 (mutism) are sufficient for a diagnosis of PFS. In the absence of B1 (mutism), Criteria A, B2, and C or D must be met for diagnosis of PFS.

Although diagnostic consensus are not widely applied in clinical practice. The following procedures are suggested for all posterior fossa patients: pre-operative neurological examination and assessment of speech and language, post-operative imaging within 48 h, and post-operative assessment score based on a scoring scale [28]

Treatment

Six review articles focused on the treatment of cerebellar mutism and were included in our study.

Medical treatment in the acute phase is tailored on patients symptoms. The medications with the greatest demonstrated benefit in our patient population have been atypical antipsychotics, primarily risperidone. Other authors suggested that zolpidem, a benzodiazepine-like medication, could have a paradoxical effect on the cortico-striatopallidal-thalamocortical pathway and reverse the damage to this area. Partial dopamine agonists such as aripiprazole may have a role in treating mental status changes after posterior fossa tumor resection [29]. Moreover, carbamazepine has been used for severe dysphoria and irritability and aripiprazole for agitation and related mental status changes [30]. Corticosteroids, fluoxetine, thyrotropin-releasing hormone, bromocriptine, midazolam, and zolpidem have all been used with inconsistent results [10, 31]. However, there are no evidence-based recommendations for the pharmacologic treatment of PFS [29].

Another issue that is of great clinical significance relates to the rehabilitation of speech and language deficits. Currently, no treatment modality centered on the speech and language deficit of CMS is available [10] and no rehabilitation trials focused on reducing or eliminating the consequences of post-operative CMS [30].

Research on cognitive remediation programs fall into two distinct categories: face-to-face therapeutic sessions targeting specific functions such as executive function, attention, memory, and academic achievement; and computer-based intervention programs that are implemented within the home setting. The most comprehensive research program of interventions target: hierarchically graded massed practice; strategy acquisition;

and cognitive-behavioral therapy. The results indicated significant improvements in academic achievement, attention, and improved implementation of metacognitive skills [31].

The use of technologies such as brain computer interfaces, robotics, virtual reality, and augmented reality tools currently adopted in the treatment of cognitive disorders in adult patients and children could also lead to advantages in terms of motivation in posterior fossa tumor post-surgery rehabilitation [32].

Long-term outcomes

Four review articles focused on the long-term outcomes of cerebellar mutism and were included in our study.

A significant proportion of patients with CMS will experience clinical improvement after the acute phase postoperatively. However, studies have also reported permanent disabling symptoms in a large proportion of mutism patients after remission [33].

In one series, 91.7% of CMS patients persisted with some motor speech deficits up to 12 years after surgery. Moreover, wide spectrum of neurocognitive and neuroemotional deficits are found during their follow-up after their operative treatment [10]. Patients with mutism for greater than 4 weeks are at an increased risk of speech and language diseases at 1-year follow-up [34].

Vermis injuries are associated with a higher risk of cognitive and affective impairments. A tumor in the right cerebellar hemisphere is associated mainly with linguistic and logical reasoning deficits, whereas a left hemisphere lesion induces attentional and visual spatial deficits. In children with PFT, lesions to the dentate nuclei on postoperative imaging were associated with lower IQ and more severe motor dexterity difficulties [5].

On neuropsychological tests, these patients were found to have significantly lower performance in processing speed, attention, working memory, executive processes, cognitive efficiency, reading, spelling and mathematics compared with

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a control group of children treated for medulloblastoma that did not develop CMS postoperatively [34].

The most prominent long term speech deficits in CMS were distorted vowels, slow speech rate, voice tremor and monopitch [34]. Meanwhile, speech rate has been shown to be an indicator of the severity of CMS, with a slower speech rate indicating greater severity of CMS [33].

CONCLUSION

Posterior fossa surgery in pediatric patients can lead to the development of postoperative pediatric cerebellar mutism syndrome (POPCMS), which is characterized by language impairment and other associated symptoms. The physiopathology of POPCMS involves injury to cerebellar tracts and cerebellar-cerebral circuits, resulting in supratentorial dysfunction and disruption of connections. Several risk factors have been identified, including midline tumor location, medulloblastoma diagnosis, younger age at diagnosis, preoperative language impairment, brainstem invasion, and tumor histology. Diagnostic criteria for POPCMS are not universally agreed upon, but they generally include language impairment, emotional lability, hypotonia, oropharyngeal dysfunction, dysphagia, and associated cerebellar motor and cognitive affective syndromes. Treatment options for POPCMS are limited, and medical management is primarily focused on symptomatic relief. Rehabilitation programs targeting speech and language deficits are lacking, although cognitive remediation programs have shown some promise. Long-term outcomes of POPCMS vary, with some patients experiencing clinical improvement while others have persistent motor speech deficits and a wide range of neurocognitive and neuroemotional impairments. Further research is needed to better understand the pathophysiology, optimize diagnostic criteria, develop effective treatments, and improve long-term outcomes for patients with POPCMS.

ACKNOWLEDGMENTS

DISCLOSURES

Ethical approval

Not applicable

Consent to participate

The patients gave consent to use their information and images for research purposes. *Consent for publication*

The patient gave consent to use his information and images for publication.

Conflict of interest

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Funding

This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors

CONTRIBUTIONS

-Matheus Ballesterro: Conceptualization, Data curation, Formal Analysis, Investigation, Methodology, Project administration, Writing – original draft, Writing – review & editing

-Rodrigo Inácio Pongeluppi: Data curation, Formal Analysis, Investigation, Writing – original draft, Writing – review & editing

-Ricardo Santos de Oliveira: Conceptualization, Supervision, Validation, Writing – original draft, Writing – review & editing

REFERENCES

1. Thacker N, Bouffet E. Posterior fossa syndrome—time to unmute the silence on cerebellar mutism. *Neuro Oncol.* 2021 Sep 1;23(9):1427–8.
2. Mariën P, Keulen S, van Dun K, De Smet HJ, De Deyn PP, Verhoeven J, et al. Cerebellar mutism syndrome in children and adults. In: Manto M, Gruol D, Schmahmann J, Koibuchi N, Sillitoe R, editors. *Handbook of the cerebellum and cerebellar disorders*. Cham: Springer International Publishing; 2020. p. 1–23.
3. Moher D, Shamseer L, Clarke M, Ghersi D, Liberati A, Petticrew M, et al. Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement. *Syst Rev.* 2015 Jan 1;4(1):1.
4. Malbari F, Gill J, Daigle A, Rodriguez LL, Raghubar KP, Davis KC, et al. Cerebellar Mutism Syndrome in Pediatric Neuro-oncology: A Multidisciplinary Perspective and Call for Research Priorities. *Pediatr Neurol.* 2022 Jul;132:4–10.
5. Doger de Spéville E, Kieffer V, Dufour C, Grill J, Noulhiane M, Hertz-Pannier L, et al. Neuropsychological consequences of childhood medulloblastoma and possible interventions: A review. *Neurochirurgie.* 2021 Feb;67(1):90–8.
6. Pitsika M, Tsitouras V. Cerebellar mutism. *J Neurosurg Pediatr.* 2013 Dec;12(6):604–14.
7. Pettersson SD, Kitlinski M, Miękisiak G, Ali S, Krakowiak M, Szmuda T. Risk factors for postoperative cerebellar mutism syndrome in pediatric patients: a systematic review and meta-analysis. *J Neurosurg Pediatr.* 2022 Apr 1;29(4):467–75.
8. Fabozzi F, Margoni S, Andreozzi B, Musci MS, Del Baldo G, Boccuto L, et al. Cerebellar mutism syndrome: From

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- pathophysiology to rehabilitation. *Front Cell Dev Biol.* 2022 Dec 2;10:1082947.
9. Jabarkheel R, Amayiri N, Yecies D, Huang Y, Toescu S, Nobre L, et al. Molecular correlates of cerebellar mutism syndrome in medulloblastoma. *Neuro Oncol.* 2020 Feb 20;22(2):290–7.
 10. Panagopoulos D, Stranjalis G, Gavra M, Boviatsis E, Korfiatis S, Karydakis P, et al. The entity of cerebellar mutism syndrome: A narrative review centered on the etiology, diagnostics, prevention, and therapeutic options. *Children (Basel).* 2022 Dec 30;10(1).
 11. Dellatolas G, Câmara-Costa H. The role of cerebellum in the child neuropsychological functioning. *Handb Clin Neurol.* 2020;173:265–304.
 12. Beez T, Munoz-Bendix C, Steiger H-J, Hänggi D. Functional tracts of the cerebellum-essentials for the neurosurgeon. *Neurosurg Rev.* 2021 Feb;44(1):273–8.
 13. Küper M, Timmann D. Cerebellar mutism. *Brain Lang.* 2013 Dec;127(3):327–33.
 14. Severo Bem L, Gemir J, Cysneiros RRM, Azevedo HC. The understanding of pediatric akinetic mutism. *Cureus.* 2021 Jan 9;13(1):e12593.
 15. Ahmadian N, van Baarsen KM, Robe PAJT, Hoving EW. Association between cerebral perfusion and paediatric postoperative cerebellar mutism syndrome after posterior fossa surgery-a systematic review. *Childs Nerv Syst.* 2021 Sep;37(9):2743–51.
 16. van Baarsen KM, Grotenhuis JA. The anatomical substrate of cerebellar mutism. *Med Hypotheses.* 2014 Jun;82(6):774–80.
 17. Stoodley CJ, Limperopoulos C. Structure-function relationships in the developing cerebellum: Evidence from early-life cerebellar injury and neurodevelopmental disorders. *Semin Fetal Neonatal Med.* 2016 Oct;21(5):356–64.
 18. Avula S, Mallucci C, Kumar R, Pizer B. Posterior fossa syndrome following brain tumour resection: review of pathophysiology and a new hypothesis on its pathogenesis. *Childs Nerv Syst.* 2015 Oct;31(10):1859–67.
 19. Grønbaek J, Molinari E, Avula S, Wibroe M, Oettingen G, Juhler M. The supplementary motor area syndrome and the cerebellar mutism syndrome: a pathoanatomical relationship? *Childs Nerv Syst.* 2020 Jun;36(6):1197–204.
 20. Ballesterio MFM, Viana DC, Teixeira TL, Santos MV, de Oliveira RS. Hypertrophic olivary degeneration in children after posterior fossa surgery. An underdiagnosed condition. *Childs Nerv Syst.* 2018 Mar;34(3):409–15.
 21. Schmähmann JD. The cerebellum and cognition. *Neurosci Lett.* 2019 Jan 1;688:62–75.
 22. Zyrianova Y, Alexander L, Faruqi R. Neuropsychiatric presentations and outcomes in children and adolescents with primary brain tumours: Systematic review. *Brain Inj.* 2016;30(1):1–9.
 23. Schmähmann JD. Pediatric post-operative cerebellar mutism syndrome, cerebellar cognitive affective syndrome, and posterior fossa syndrome: historical review and proposed resolution to guide future study. *Childs Nerv Syst.* 2020 Jun;36(6):1205–14.
 24. Bakhshi SK, Mitha R, Mushtaq N, Shamim MS. Cerebellar Mutism Syndrome after surgical resection of posterior fossa neoplastic lesions. *J Pak Med Assoc.* 2020 Sep;70(9):1667–8.
 25. Reed-Berendt R, Phillips B, Picton S, Chumas P, Warren D, Livingston JH, et al. Cause and outcome of cerebellar mutism: evidence from a systematic review. *Childs Nerv Syst.* 2014 Mar;30(3):375–85.
 26. Wickenhauser ME, Khan RB, Raches D, Ashford JM, Robinson GW, Russell KM, et al. Characterizing posterior fossa syndrome: A survey of experts. *Pediatr Neurol.* 2020 Mar;104:19–22.
 27. Ricci FS, D'Alessandro R, Somà A, Salvalaggio A, Rossi F, Rampone S, et al. Development and application of a diagnostic and severity scale to grade post-operative pediatric cerebellar mutism syndrome. *Eur J Pediatr.* 2022 Mar;181(3):941–50.
 28. Gudrunardottir T, Morgan AT, Lux AL, Walker DA, Walsh KS, Wells EM, et al. Consensus paper on post-operative pediatric cerebellar mutism syndrome: the Iceland Delphi results. *Childs Nerv Syst.* 2016 Jul;32(7):1195–203.
 29. Lanier JC, Abrams AN. Posterior fossa syndrome: Review of the behavioral and emotional aspects in pediatric cancer patients. *Cancer.* 2017 Feb 15;123(4):551–9.
 30. Molinari E, Pizer B, Catsman-Berrevoets C, Avula S, Keating R, Paquier P, et al. Posterior Fossa Society Consensus Meeting 2018: a synopsis. *Childs Nerv Syst.* 2020 Jun;36(6):1145–51.
 31. Paquier PF, Walsh KS, Docking KM, Hartley H, Kumar R, Catsman-Berrevoets CE. Post-operative cerebellar mutism syndrome: rehabilitation issues. *Childs Nerv Syst.* 2020 Jun;36(6):1215–22.
 32. Chieffo DPR, Lino F, Arcangeli V, Moriconi F, Frassanito P, Massimi L, et al. Posterior Fossa Tumor Rehabilitation: An Up-to-Date Overview. *Children (Basel).* 2022 Jun 16;9(6).
 33. Ashida R, Nazar N, Edwards R, Teo M. Cerebellar mutism syndrome: an overview of the pathophysiology in relation to the cerebrocerebellar anatomy, risk factors, potential treatments, and outcomes. *World Neurosurg.* 2021 Sep;153:63–74.
 34. Catsman-Berrevoets CE. Cerebellar mutism syndrome: cause and rehabilitation. *Curr Opin Neurol.* 2017;30(2):133–9.