

# Cerebellar mutism risk factors

- Cerebellar Mutism/Posterior Fossa Syndrome Following Resection of Posterior Fossa Tumor in Pediatric Patients: Assessing Pathophysiology, Risk Factors, and Neuroradiographic Features
  - Radiological Predictors of Cognitive Impairment in Paediatric Brain Tumours Using Multiparametric Magnetic Resonance Imaging: A Review of Current Practice, Challenges and Future Directions
  - Postoperative word-finding difficulties in children with posterior fossa tumours: a crosslinguistic European cohort study
  - Predicting cerebellar mutism syndrome in children using lesion map combined with clinical features
  - Risk factors for domain-specific neurocognitive outcome in pediatric survivors of a brain tumor in the posterior fossa-Results of the HIT 2000 trial
  - Medical management for cerebellar mutism syndrome following posterior fossa surgery: A systematic review
  - The Cerebellar Mutism Syndrome: Risk Assessment, Prevention and Treatment
  - Long-term postoperative quality of life in childhood survivors with cerebellar mutism syndrome
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Proven risk factors include brain stem invasion, diagnosis of medulloblastoma, midline localization, tumor size, invasion of the fourth ventricle, invasion of the superior cerebellar peduncle, left-handedness, and incision of the vermis<sup>1)</sup>

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For Grønbæk et al. data do not support the hypothesis that handedness should be of clinical relevance in the risk assessment of CMS<sup>2)</sup>.

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Findings enhance an already hypothesized role of cerebellar language lateralization<sup>3)</sup>.

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Risk factors for post-op cerebellar mutism following surgery for medulloblastoma in children: brainstem involvement & midline location<sup>4)</sup>.

Postoperative cerebellar mutism has been observed in ≈ 1% of adults following posterior fossa surgery<sup>5)</sup>

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Male sex and cerebro-cerebellar circuit damage are independent risk factors for pCMS. The cerebro-cerebellar circuit score indicates the duration of mutism<sup>6)</sup>

Male patients had a higher risk of developing CMS after a posterior fossa tumor resection. Midline location, solid tumor consistency, and hydrocephalus were independent risk factors for CMS<sup>7)</sup>.

Chua et al., performed a clinical study that evaluated possible risk factors for permanent PFS in paediatric medulloblastoma patients. Analysis of collated results found that post-operative DWI restriction in bilateral regions within the surgical cavity demonstrated statistical significance as a predictor of PFS permanence-a novel finding in the current literature<sup>8)</sup>.

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Children who receive treatment for medulloblastoma have a high survival rate, but also a high likelihood of developing posterior fossa syndrome.

Diffusion abnormalities were identified in 10 cases, 7 of which involved the proximal efferent cerebellar pathway (pECP). A retrospective evaluation revealed evidence of PFS in 6 cases. There was a significant association between abnormalities involving pECP structures ( $P = .001$ ) and the development of PFS. Bilateral involvement of pECP ( $P = .006$ ) was a highly specific risk factor for predicting the development of PFS. Diffusion abnormality of the inferior vermis was significantly associated with PFS ( $P = .001$ ) but may not represent a risk factor in isolation<sup>9)</sup>.

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Infratentorial glioblastoma recurrence is a severe recurrence type in GBM patients. Its symptoms are neurologically unspecific and can be overlooked or misdiagnosed as side effects of treatments. Carefully checking the infratentorial region, especially around the fourth ventricle, is essential during the GBM patient follow-up. The primary symptoms of ITR were gait disturbance (100%, n = 6), dizziness (50.0%, n = 3), nausea (33.3%, n = 2), and cerebellar mutism (16.7%, n = 1). In four cases (66.7%), symptoms were presented before ITR development<sup>10)</sup>

<sup>1)</sup>

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<sup>2)</sup>

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<sup>3)</sup>

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<sup>4)</sup>

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<sup>5)</sup>

Dubey A, Sung WS, Shaya M, et al. Complications of posterior cranial fossa surgery—an institutional

experience of 500 patients. *Surg Neurol.* 2009; 72: 369–375

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