

Incidence of and Risk Factors for Postoperative Paediatric Cerebellar Mutism Syndrome in Children with Posterior Fossa Tumours: A Retrospective Study

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Keywords

Cerebellar mutism; Medulloblastoma; Pilocytic astrocytoma; Posterior fossa

Abstract

Objective

Postoperative paediatric cerebellar mutism syndrome (pCMS) can occur in children after resection of a posterior fossa brain tumour. It is characterised by mutism or reduced speech and emotional lability, often with disturbances in motor and cognitive functions. This study aimed to analyse the clinical features and risk factors for pCMS in the Ghent University Hospital.

Methods

Retrospective data of children treated for posterior fossa tumours in the Ghent University Hospital between 2008 and 2022 were reviewed, using the 2016 Delphi consensus definition of pCMS.

Results

Twenty-two out of 69 patients (32%) developed pCMS. Motor deficits were most frequent. Dysarthria was the leading speech impairment, mutism was the primary language disorder. The most prevalent affective disorder was anxiety. Univariate analysis showed significant associations with medulloblastoma (OR 3.41; 95%CI 1.19-9.78), brain stem invasion (OR 4.76; 95%CI 1.24-18.31) and midline tumour location (OR 1.58; 95%CI 1.30-1.91). Conversely, pCMS was less frequent in pilocytic astrocytomas (OR 0.21; 95%CI 0.06-0.73) and in tumours larger than 5 cm (OR 0.08; 95%CI 0.02-0.39).

Conclusion

This study highlights the clinical features and risk factors of pCMS in children treated for posterior fossa tumours. While motor deficits were most commonly reported, disorders in speech, language, and affective and cognitive function may have been underrecognised. Risk factors such as tumour type, brain stem invasion and midline location, are consistent with existing literature, whereas a "protective" effect of larger tumours might be attributed to the predominance of large pilocytic astrocytomas in this cohort.

Introduction

Brain tumours are the second most common type of malignancy in children, and represent the most prevalent form of solid tumours in this age group (1). Among all paediatric brain tumours, 45-60% are located in the posterior fossa, more specifically cerebellar tumours and tumours in the fourth ventricle (2). Postoperative paediatric cerebellar mutism syndrome (pCMS) can develop in children following the resection of a posterior fossa tumour and is characterised by delayed onset mutism or reduced speech, and

emotional lability (3). The incidence of pCMS ranges from 11% to 29% in retrospective studies, but is higher in prospective studies, ranging from 24% to 30% (4-8). However, comparing studies and incidence rates is challenging, as a consensus on the definition was not established until 2016 (3). Mutism, defined as the (partial) absence of speech despite preserved physical speech ability, is the most characteristic symptom of pCMS, although it is not a diagnostic requirement (5). It typically manifests within hours to days after surgery, with vocalisations often limited to high-pitched crying (4). The mutism is always transient, lasting from

days to months with an average duration of five weeks. Some children continue to experience speech and language difficulties after the mutism resolves (8, 9). During the postoperative period, children with pCMS often develop additional symptoms related to motor and cognitive functions (3). The additional symptoms persist longer than the mutism, particularly ataxia, dysarthria and other impairments (10). The underlying mechanisms of pCMS remain incompletely understood. The current hypothesis attributes pCMS to diaschisis, a phenomenon in which damage to one brain region disrupts function in a distant but connected area due to reduced input (11, 12). In pCMS, injury to the dentato-thalamo-cortical (DTC) tract during posterior fossa tumour resection can lead to decreased cerebellar input, resulting in temporary hypoactivity in the cerebral hemispheres and affecting motor coordination, cognition, and emotional regulation (11, 13). Neural plasticity and compensatory mechanisms contribute to recovery, but persistent symptoms following the resolution of mutism are most likely attributable to direct injury to the cerebellum (9, 10, 12). Treatment is primarily supportive, involving multidisciplinary rehabilitation with physical, speech, and cognitive therapies (10, 14). It enables children to develop alternative strategies to compensate for functional deficits, supports the promotion of independence, and facilitates reintegration into activities with peers. However, rehabilitation alone is insufficient to prevent further decline in motor and cognitive functions, partly due to the impact of chemotherapy or radiotherapy in the postoperative period (8, 14). A limited number of studies and case reports have suggested a potential benefit of various pharmacological agents in the management of pCMS, including benzodiazepines and selective serotonin reuptake inhibitors (15-17). However, no large-scale studies have demonstrated consistent efficacy. Multiple risk factors for the development of pCMS have been identified in various studies, with three consistently confirmed: tumour type (medulloblastoma), midline tumour location, and brainstem involvement. This research aimed to evaluate the incidence of pCMS, identify its clinical features, and determine the associated risk factors at the Ghent University Hospital over the last 15 years.

Materials and Methods

This retrospective study included children up to 18 years of age who were diagnosed with a posterior fossa tumour at the Ghent University Hospital in Belgium over a fifteen-year period, between January 1st, 2008, and December 31st, 2022. The study was approved by the Ethics Committee affiliated with the Ghent University Hospital. The analysis focused on the first surgical intervention, data regarding surgery for recurrent tumours were not analysed. Patients whose initial surgery was performed at a different hospital before referral, were excluded. Patients under one year of age, in whom assessment of language and speech is challenging, were also excluded. Demographic data, patient characteristics, tumour characteristics on imaging and histopathology, and surgical details were collected. The selection of possible risk factors was based on those most frequently reported in the literature and included tumour type, midline location, brainstem involvement, tumour size, left-handedness, and surgical technique. Postoperative clinical information was analysed to determine the occurrence of pCMS. All children were evaluated postoperatively within a multidisciplinary rehabilitation program, including assessments by a speech therapist, physiotherapist, occupational therapist, and rehabilitation physician. Reports from all specialists were reviewed and consulted. These findings were subsequently compared with those reported in literature. This study adopts the 2016 Delphi consensus definition of pCMS, which is characterised by mutism or reduced speech and emotional lability following surgery for a cerebellar or fourth ventricle tumour in children (3). All patients were evaluated for development of

symptoms related to language, speech, or emotional affect during the postoperative period. Additional symptoms, as delineated in the 2016 consensus article, were systematically evaluated. All possible symptoms were categorised into five distinct domains:

- Motor deficits; including hypotonia, dysmetria, dysphagia, ataxia, hemiparesis, oculomotor problems, oropharyngeal dysfunction, urinary retention or incontinence, and cranial neuropathies
- Speech impairments; including dysarthria, disturbed prosody, and reduced speech production such as speaking in short sentences or single words
- Language disorders; including mutism, word-finding difficulties, and reduced verbal fluency
- Affective disorders; including emotional lability, personality changes with apathy or inappropriate behaviour
- Cognitive impairments; defined as intellectual impairments, including reduced executive functions, attentional problems, visuospatial disorganisation, and impaired visuospatial memory

Statistical analyses were performed using SPSS Statistics 28. Statistical significance was set at $P < 0.05$. Univariate analyses were conducted using the Mann-Whitney U test for continuous variables and the Pearson's chi-square test for categorical variables. The Fisher's exact test was used when the expected values were less than five. For survival analysis, the Kaplan-Meier method and the Mantel-Cox log-rank test were used.

Results

Demographic Data, Patient and Tumour Characteristics

Table 1 presents the demographic and clinical data of the cohort. During the observational period, 76 paediatric patients were admitted to the Ghent University Hospital with a tumour in the posterior fossa. Seven patients were excluded from the analysis: three patients whose surgery was performed at the referring hospital, one patient with an inoperable tumour, and three patients younger than 1 year at time of diagnosis (11 months, 9 months, and 5 months of age). Data from 69 patients were analysed. The median duration of follow-up was 66 months, or 5.5 years. Within the cohort, 58% of patients were male, resulting in a male-to-female ratio of 1.38. The median age at diagnosis was 7 years. The three main tumour types were medulloblastoma, pilocytic astrocytoma and (anaplastic) ependymoma. Other diagnoses on histopathology, including atypical teratoid rhabdoid tumour (ATRT, 1 case), choroid plexus papilloma (1 case), diffuse low-grade astrocytoma (3 cases), hemangioblastoma (1 case), and grade 2 low-grade glioma (1 case), are categorised as "other" due to their low incidence. In the majority of tumours (88%), imaging revealed brainstem involvement due to invasion, external compression, or a combination of both. Most tumours (87%) were located at the midline. The tumours had a median size of 4.8 cm (largest diameter on imaging), and 44% of the tumours exceeded 5 cm in size. When fundoscopy was performed at diagnosis, papilledema was present in 42% of patients. Imaging revealed hydrocephalus in 78% of cases, for which surgical intervention—most commonly the placement of an external ventricular drain—was required prior to tumour resection. Detailed information on the surgical procedure was available for 41 patients, of whom 15 (37%) underwent splitting of the vermis. Postoperative imaging demonstrated complete resection in 58% of cases. During the 15-year follow-up period, 14 out of 69 children died. The 5-year overall survival rate reached 90% in this cohort, and the 10-year overall survival rate was 77%. Among patients with medulloblastoma, 10 patients died (37%). No deaths occurred in the group with pilocytic astrocytoma. In each of the groups with (anaplastic) ependymoma and combined other tumours, there were two deaths (29%). The

log-rank test (Mantel-Cox) confirmed a statistically significant difference in survival between all groups (p = 0.012).

Postoperative paediatric Cerebellar Mutism Syndrome

pCMS was retrospectively diagnosed in 22 children (32%), all of whom exhibited symptoms across multiple domains. Motor deficits were present in the majority of these patients (19/22), with hypotonia being the most prevalent (N=9), followed by hemiparesis, oculomotor dysfunction, and ataxia, each observed in 4 children. Speech impairments were identified in 13/22 children, most commonly dysarthria (N=9), and language disorders were present in 9/22 children, with mutism being the most frequent (N=7). Affective disorders were reported in 8/22 children, including anxiety (N=3) and autistic behaviour (N=2). Cognitive impairments

were the least common, with only one child demonstrating diminished executive skills. Further information regarding the clinical features is presented in Figure 1. The demographic and clinical data of patients with and without pCMS are compared in Table 2. A statistically significant difference in the incidence of pCMS was observed based on tumour type, as determined by histopathological analysis. Patients with medulloblastoma had a higher probability of developing pCMS (odds ratio [OR] 3.41; 95% confidence interval [CI] 1.19–9.78). The molecular subgroups of medulloblastomas were also analysed. Only having been systematically examined since 2016, this could only be ascertained in 11 patients. Of these patients, three exhibited the wingless (WNT) type (one developed pCMS), two presented with the Group 3 type (one developed pCMS), and six manifested the Group 4 type (three developed pCMS). No patient with the sonic hedgehog (SHH) type was identified. The diagnosis of pilocytic astrocytoma was associated with a decreased likelihood of developing pCMS following surgical intervention (OR 0.21; 95%CI 0.06–0.73). Tumours with brainstem invasion (OR 4.76; 95%CI 1.24–18.31) or a midline tumour location (OR 1.58; 95%CI 1.30–1.91) were associated with a significant increased likelihood of developing pCMS. There was also a significant correlation for tumour size: children with tumours larger than 5 cm exhibited a significantly lower risk of developing pCMS (OR 0.08; 95%CI 0.02–0.39). To further elucidate this finding, the tumour size was evaluated per tumourtype. The median tumour size of the medulloblastomas associated with pCMS was 4.3 cm (maximum 7.0 cm), whereas medulloblastomas that were not associated with pCMS had a smaller median tumour size of 4.1 cm (maximum 6.4 cm). This finding was not statistically significant (P = 0.895), suggesting only a minimal difference between groups. In contrast, the four children with pilocytic astrocytomas who developed pCMS had a median tumour size of 4.1 cm (maximum 4.9 cm), whereas the remaining 24 pilocytic astrocytomas had a larger median tumour size of 5.3 cm (maximum 8.7 cm).

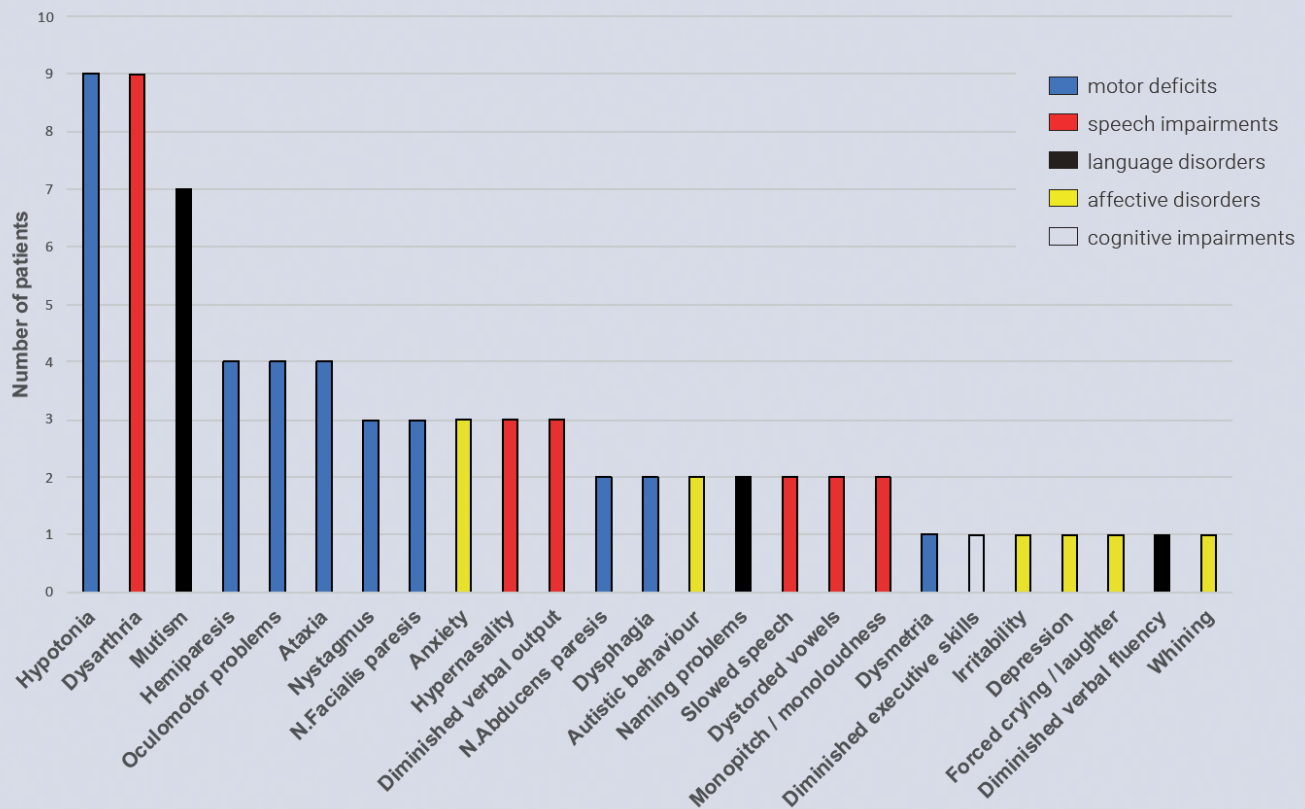
TABLE 1: Demographic and clinical data of the cohort.

	Total = 69 (range)	%
Sex		
Male	40	58
Female	29	42
Median age at diagnosis	7 years (13 months – 15 years)	
Brain stem involvement	52	88
External compression	47	83
Invasion	12	21
Tumour location		
Midline	60	87
Left cerebellar hemisphere	7	10
Right cerebellar hemisphere	2	3
Tumour size		
Median size	4.8 cm (2.0 cm – 8.3 cm)	
Larger than 5 cm	26	44
Smaller than 3 cm	7	12
Left-handedness	10	28
Papilledema	14	42
Hydrocephalus	52	78
Management of hydrocephalus		
None	11	21
External ventricular drain	40	77
Third ventriculostomy	1	2
Splitting of the vermis	15	37
Complete resection on radiology	40	58
Tumour type (as on histopathology)		
Medulloblastoma	27	39
Pilocytic astrocytoma	28	41
(anaplastic) ependymoma	7	10
Other	7	10
Development of pCMS	22	32
Deceased	14	20

Discussion

This retrospective single-centre study analysed data from 69 paediatric patients who presented with a tumour located in the posterior fossa. This study marks the first comprehensive analysis of data from Belgian children with posterior fossa tumours who are susceptible to developing pCMS. Tumour type was significantly associated with the development of pCMS. Medulloblastoma emerged as a strong predictor, consistent with the available literature (6, 18-21). These tumours often occur along the midline and invade the brainstem, increasing the risk of damaging the adjacent DTC-tract during surgery. In contrast, the diagnosis of pilocytic astrocytoma was significantly associated with a lower risk of developing pCMS—an association previously reported only once (21). This “protective effect” may be related to the typical lateral location of these tumours within the cerebellar hemispheres. Children with a tumour invading the brainstem had a significantly higher risk of developing pCMS, a result that matches several previous meta-analyses and retrospective studies (18, 19, 21, 22). A prospective study focusing exclusively on medulloblastomas also reported this risk factor (4). Patients with tumours situated on the midline had a higher probability of developing pCMS, which is also consistent with earlier research (6, 19-21, 23). This correlation can be attributed to the caudal structures of the DTC-tract being located medially (19). In this study, tumour size was significantly associated with pCMS, with larger tumours linked to a lower likelihood of developing pCMS. Overall, the pilocytic astrocytomas in this cohort were on average larger than medulloblastomas (Figure 2). In the subgroup of pilocytic astrocytomas, pCMS occurred in relatively smaller tumours, which may have contributed to the overall association between larger

FIGURE 1: Clinical features in children who developed pCMS.



tumour size and a lower likelihood of developing pCMS. In contrast, analysis of the medulloblastoma subgroup demonstrated a non-significant trend suggesting that larger medulloblastomas may be associated with the development of pCMS, with higher median and maximum tumour dimensions observed in the pCMS group. Although this finding did not reach statistical significance, it may suggest that increasing tumour size could play a role in medulloblastomas for causing pCMS. In a literature review, Ashida et al. reported an association between larger tumour size and pCMS (19). However, this conclusion was based on only three prior studies, the largest of which included exclusively medulloblastomas (24). This effect can be explained by the tendency of larger medulloblastomas to invade nearby anatomical structures, and thereby increasing the risk of damage to the DTC tract during surgery. The above-mentioned findings support the hypothesis that the development of pCMS is more strongly associated with the underlying histopathological tumour type, as each type has its distinct biological characteristics, with size or location reflecting the tumour type rather than acting as independent risk factors. In this cohort, medulloblastomas, which frequently invade adjacent tissues, were mostly located on the midline and smaller compared to pilocytic astrocytomas (Figures 2 and 3). In a large prospective cohort study it was observed that high-grade tumours, including medulloblastomas, were significantly linked to postoperative speech impairment, regardless of tumour location or surgical technique (20). In other studies, a significant correlation between increasing tumour size and pCMS was found only in medulloblastomas, likely due to larger medulloblastomas showing more invasion to adjacent tissues than other tumour types (18, 23). On the other hand, the “protective effect” of pilocytic astrocytomas against pCMS, as reported by Dhaenens et al.,

persisted even in midline pilocytic astrocytomas (21). More recently it was also observed that even the molecular subtype in medulloblastomas correlates with pCMS development. Jabarkheel et al. observed that children with SHH-type medulloblastomas were less likely to develop pCMS, partially because of the characteristic lateral positioning of this tumour subtype, although a protective effect was also noted in medial positioning (25). To further elucidate the effect of tumour type on the development of pCMS, larger prospective studies are required. No significant correlation was observed for other parameters, including left-handedness. Only one study had identified this feature as being significantly associated with pCMS, whereas others found no such association (26-28), aligning with the findings of this analysis. As pCMS is a postoperative complication, substantial research has focused on surgical techniques to reduce its risk. This study included both the traditional transvermian approach, which involves splitting the vermis, and the telovelar approach, in which splitting is not required. No significant association was observed between vermis splitting and the development of pCMS, as previously reported in multiple studies (7, 20, 22). However, two meta-analyses and one retrospective study did find a significant association between surgical technique and pCMS incidence, favouring the telovelar approach (18, 19, 29). Additionally, it was reported that gentle dynamic retraction can reduce the risk of pCMS compared to the use of static retracting spatulae, further supporting the benefit of the telovelar approach (29). Thanks to modern surgical techniques, such as neuronavigation and the use of ultrasonic surgical aspirator suction cannulas, it is possible to perform gross central tumour debulking before determining tumour margins. This enables smaller operative windows and minimises the need for brain tissue retraction, thereby facilitating

TABLE 2: Possible risk factors for pCMS (**Bold**: significant P-values).

	pCMS (N = 22)	%	No pCMS (N = 47)	%	P-value
Tumour type (as on histopathology)					
Medulloblastoma	13	59	14	30	0.020
Pilocytic astrocytoma	4	18	24	51	0.010
(anaplastic) ependymoma	2	9	5	11	1.000
Other	3	14	4	9	0.672
Tumour location					
Midline	22	100	38	81	0.049
Left cerebellar hemisphere	0	0	7	15	-
Right cerebellar hemisphere	0	0	2	4	-
Brain stem involvement	17	94	35	85	0.422
External compression	13	77	34	85	0.464
Invasion	7	41	5	13	0.031
Tumour size					
Median size	4.3 cm		5.2 cm		0.042
Larger than 5 cm	2	11	24	60	<0.001
Smaller than 3 cm	2	11	5	13	1.000
Splitting of the vermis	7	44	8	32	0.446
Left-handedness	5	42	5	21	0.247

the less invasive telovelar approach and permitting gentle, dynamic retraction (22). Although tumour-related factors likely play the major role in the development of pCMS, surgical technique remains the only modifiable factor that may influence its incidence. It therefore represents an important focus of preventive strategies, and surgeons should consistently aim to optimise their technique in order to minimise the incidence of pCMS. It was remarkable that postoperative reports primarily focused on motor deficits like hypotonia and hemiparesis, while speech impairments, language disorders and affective disturbances, essential for diagnosing pCMS, were reported less frequently. These symptoms were documented in subsequent reports by speech therapists and rehabilitation specialists. It is imperative for healthcare providers to consider these more subtle manifestations in the postoperative period, as they are sufficient to independently establish a diagnosis of pCMS. Early identification is

essential for timely initiation of appropriate rehabilitation and follow-up. As previously mentioned, rehabilitation alone is not sufficient to prevent further decline in motor and cognitive functions in children with pCMS (14). The prognosis and symptom progression for individual patients are difficult to predict, as the course of recovery is highly variable. Speech impairments often persist for several months or even years, and mutism lasting longer than four weeks is associated with an increased risk of long-term speech and language deficits (8). Furthermore, pCMS appears to exert a substantial negative impact on cognitive function (30). The combination of these postoperative sequelae in children—who already contend with a brain tumour, the effects of chemotherapy and, in some cases, radiotherapy, and frequent school absenteeism—renders them particularly susceptible to learning difficulties and challenges in academic progression. Given that these impairments in speech, language, and cognition can persist throughout several years of childhood, it is therefore essential that paediatricians and other healthcare professionals involved in the long-term follow-up of these patients are familiar with pCMS and its clinical features. This study had several limitations, partly due to its retrospective single-centre design. A first limitation is the absence of correction for multiple testing. Although variables were selected a priori, multiple comparisons increase the risk of false-positive findings, and some associations may reflect chance. Given the small sample size and limited number of pCMS events, applying a strict correction would have reduced statistical power. Therefore, results should be interpreted cautiously and considered exploratory, requiring confirmation in larger cohorts. The limited sample size and missing values prevented a multivariate analysis. While imaging reports were reviewed, the images were not reanalysed by a (paediatric) radiologist, which could have reduced missing data regarding tumour characteristics on imaging. The diagnosis of pCMS was retrospectively established, based on the 2016 definition (3). A

FIGURE 2: Tumour size according to tumour typer.

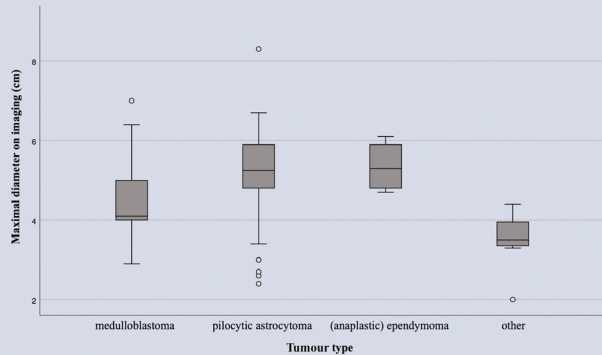
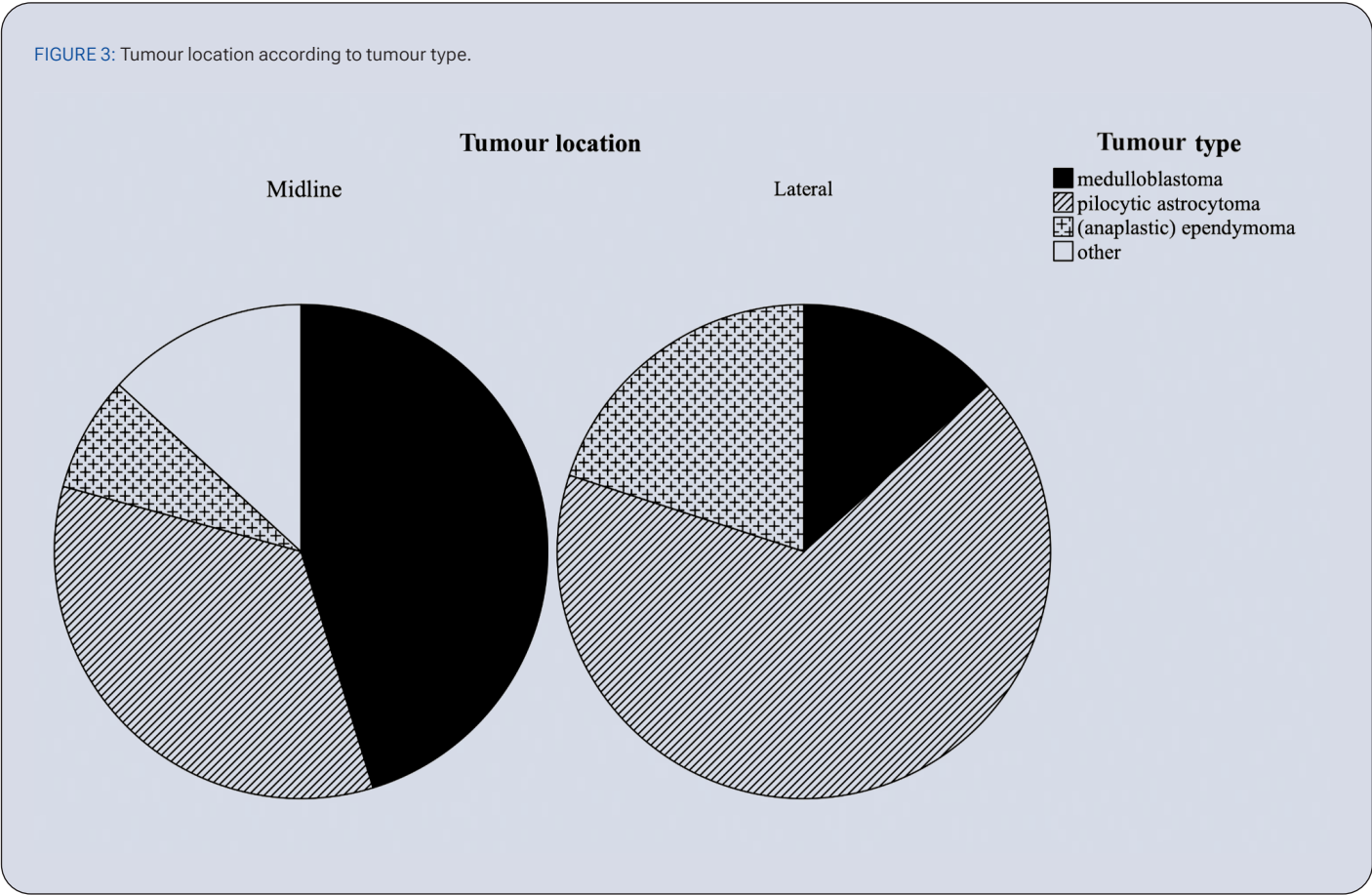


FIGURE 3: Tumour location according to tumour type.



standardized evaluation of language and speech was not conducted preoperatively, as this was not standard practice at our centre. Postoperative symptoms related to language, speech, or emotional affect can be more subtle than motor deficits, which may lead to missing data in retrospective studies due to inadequate recognition or reporting. The observed pCMS incidence of 32% is consistent with previous studies. Despite its retrospective nature, the current study appears to be representative of children with pCMS, as the incidence and the identified risk factors align with the findings reported in the literature. Prospective longitudinal studies employing multivariate analyses are the most effective tools for identifying risk factors and elucidating their underlying aetiology, which remains under investigation. Currently, a large prospective study, referred to as the Nordic Study of Cerebellar Mutism Syndrome in Children with Posterior Fossa Brain Tumours, is being conducted in Europe (31).

Conclusion

This study describes the clinical features and risk factors associated with pCMS in children treated for posterior fossa tumours at Ghent University Hospital. Motor deficits were reported most frequently, while disorders in speech, language, and affective and cognitive function may have been underrecognised. Several risk factors showed a significant association with the development of pCMS. The diagnosis of medulloblastoma, brain stem invasion, and midline tumour location were associated with an increased risk of pCMS, whereas the diagnosis of pilocytic astrocytoma and tumour size greater than 5 cm were associated with a reduced risk. The latter may be explained by the fact that pilocytic astrocytomas in this cohort were, on average, larger than medulloblastomas, and supports the hypothesis that certain tumour characteristics, such as location and size, may reflect the underlying tumour type rather than constitute independent risk factors

for pCMS. Comprehensive postoperative monitoring of patients at risk for pCMS is crucial, with particular emphasis on language, speech, and affective and cognitive functions. This facilitates early detection of pCMS and initiation of rehabilitation, thereby enhancing patient outcomes and quality of life. Children with pCMS may experience lasting neurological deficits, which—combined with treatment effects and time away from school—can increase learning difficulties. Awareness of pCMS is therefore important for long-term follow-up by healthcare professionals.

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REFERENCES

1. Udaka YT, Packer RJ. Pediatric brain tumors. *Neurologic clinics*. 2018;36(3):533-56.
2. Formentin C, Joaquim AF, Ghizoni E. Posterior fossa tumors in children: current insights. *European Journal of Pediatrics*. 2023;182(11):4833-50.
3. 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. *Child's Nervous System*. 2016;32:1195-203.
4. Robertson PL, Muraszko KM, Holmes EJ, Sposto R, Packer RJ, Gajjar A, et al. Incidence and severity of postoperative cerebellar mutism syndrome in children with medulloblastoma: a prospective study by the Children's Oncology Group. *Journal of Neurosurgery: Pediatrics*. 2006;105(6):444-51.
5. Catsman-Berrevoets CE, Aarsen FK. The spectrum of neurobehavioural deficits in the Posterior Fossa Syndrome in children after cerebellar tumour surgery. *Cortex*. 2010;46(7):933-46.
6. Küpeli S, Yalçın B, Bilginer B, Akalan N, Haksal P, Büyükpamukçu M. Posterior fossa syndrome after posterior fossa surgery in children with brain tumors. *Pediatric Blood & Cancer*. 2011;56(2):206-10.
7. Zaheer SN, Wood M. Experiences with the telovelar approach to fourth ventricular tumors in children. *Pediatric neurosurgery*. 2011;46(5):340-3.
8. Gudrunardottir T, Sehested A, Juhler M, Schmiegelow K. Cerebellar mutism: review of the literature. *Child's nervous system*. 2011;27:355-63.
9. İldan F, Tuna M, Erman T, Göçer A, Zeren M, Cetinalp E. The evaluation and comparison of cerebellar mutism in children and adults after posterior fossa surgery: report of two adult cases and review of the literature. *Acta neurochirurgica*. 2002;144:463-73.
10. Küper M, Timmann D. Cerebellar mutism. *Brain and language*. 2013;127(3):327-33.
11. van Baarsen KM, Grotenhuis JA. The anatomical substrate of cerebellar mutism. *Medical hypotheses*. 2014;82(6):774-80.
12. Miller N, Reddick W, Kocak M, Glass J, Löbel U, Morris B, et al. Cerebellocerebral diaschisis is the likely mechanism of postsurgical posterior fossa syndrome in pediatric patients with midline cerebellar tumors. *American journal of neuroradiology*. 2010;31(2):288-94.
13. van Baarsen K, Kleinnijenhuis M, Konert T, van Cappellen van Walsum A-M, Grotenhuis A. Tractography demonstrates dentate-rubro-thalamic tract disruption in an adult with cerebellar mutism. *The Cerebellum*. 2013;12:617-22.
14. Decock M, De Wilde R, Van der Looen R, Vander Linden C. Motor functioning and intelligence quotient in paediatric survivors of a Fossa posterior tumor following a multidisciplinary rehabilitation program. *International Journal of Environmental Research and Public Health*. 2022;19(12):7083.
15. Shyu C, Burke K, Souweidane MM, Dunkel LJ, Gilheeney SW, Gershon T, et al. Novel use of zolpidem in cerebellar mutism syndrome. *Journal of pediatric hematology/oncology*. 2011;33(2):148-9.
16. Nicita F, Paiano M, Liberatore M, Spalice A, Papoff P, Ullo M, et al. Sudden benzodiazepine-induced resolution of post-operative pediatric cerebellar mutism syndrome: a clinical-SPECT study. *Acta neurochirurgica*. 2017;159:475-9.
17. Amor-García MÁ, Fernández-Llamazares CM, Manrique-Rodríguez S, Narrillos-Moraza A, García-Morín M, Huerta-Aragón J, et al. Bromocriptine for the treatment of postoperative cerebellar mutism syndrome in pediatric patients: Three case reports. *Journal of Oncology Pharmacy Practice*. 2021;27(7):1753-7.
18. 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. *Journal of Neurosurgery: Pediatrics*. 2021;29(4):467-75.
19. Ashida R, Nazar N, Edwards R, Teo M. Cerebellar mutism syndrome: An overview of the pathophysiology in relation to the cerebrotocerebellar anatomy, risk factors, potential treatments, and outcomes. *World Neurosurgery*. 2021;153:63-74.
20. Grønbaek JK, Wibroe M, Toescu S, Frič R, Thomsen BL, Møller LN, et al. Postoperative speech impairment and surgical approach to posterior fossa tumours in children: a prospective European multicentre cohort study. *The Lancet Child & Adolescent Health*. 2021;5(11):814-24.
21. Bae D, Catsman-Berrevoets C. Preoperative prediction of postoperative cerebellar mutism syndrome. Validation of existing MRI models and proposal of the new Rotterdam pCMS prediction model. *Child's Nervous System*. 2020;36:1471-80.
22. Renne B, Radic J, Agrawal D, Albrecht B, Bonfield CM, Cohrs G, et al. Cerebellar mutism after posterior fossa tumor resection in children: a multicenter international retrospective study to determine possible modifiable factors. *Child's Nervous System*. 2020;36:1159-69.
23. Kotil K, Mustafa E, Akcetin M, Bilge T. Cerebellar mutism following posterior fossa tumor resection in children. *Turkish neurosurgery*. 2008;18(1).
24. Pols SY, van Veelen MLC, Aarsen FK, Candel AG, Catsman-Berrevoets CE. Risk factors for development of postoperative cerebellar mutism syndrome in children after medulloblastoma surgery. *Journal of Neurosurgery: Pediatrics*. 2017;20(1):35-41.
25. Jabarkheel R, Amayiri N, Yecies D, Huang Y, Toescu S, Nobre L, et al. Molecular correlates of cerebellar mutism syndrome in medulloblastoma. *Neuro-oncology*. 2020;22(2):290-7.
26. McEvoy SD, Lee A, Poliakov A, Friedman S, Shaw D, Browd SR, et al. Longitudinal cerebellar diffusion tensor imaging changes in posterior fossa syndrome. *NeuroImage: Clinical*. 2016;12:582-90.
27. Grønbaek JK, Laustsen AF, Toescu S, Pizer B, Mallucci C, Aquilina K, et al. Left-handedness should not be overrated as a risk factor for postoperative speech impairment in children after posterior fossa tumour surgery: a prospective European multicentre study. *Child's Nervous System*. 2022;38(8):1479-85.
28. Law N, Greenberg M, Bouffet E, Taylor MD, Laughlin S, Strother D, et al. Clinical and neuroanatomical predictors of cerebellar mutism syndrome. *Neuro-oncology*. 2012;14(10):1294-303.
29. Cobourn K, Marayati F, Tsering D, Ayers O, Myseros JS, Magge SN, et al. Cerebellar mutism syndrome: current approaches to minimize risk for CMS. *Child's Nervous System*. 2020;36:1171-9.
30. Catsman-Berrevoets CE. Cerebellar mutism syndrome: cause and rehabilitation. *Current Opinion in Neurology*. 2017;30(2):133-9.
31. Wibroe M, Cappelen J, Castor C, Clausen N, Grillner P, Gudrunardottir T, et al. Cerebellar mutism syndrome in children with brain tumours of the posterior fossa. *BMC Cancer*. 2017;17:1-7.