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CASE REPORT

Transient mutism after posterior fossa surgery

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Abstract An 8-year-old girl developed mutism after removal of a vermian medulloblastoma. The mutism was not accompanied by long tract signs or cranial nerve palsy. The girl started to regain her speech 2 weeks postoperatively, showing marked improvement 2 months after the operation, after passing through a dysarthric phase. Magnetic resonance imaging showed only normal postoperative changes without abnormalities of dentate nuclei or supranuclear region. Nineteen similar cases of transient mutism following cerebellar operations have been reported in the literature, most of them with a delayed postoperative onset. In all patients the recovery of speech started to appear 4 days to 4 months postoperatively, and all patients passed through a monotonous, dysarthric phase. The absence of long tract or other brain stem signs, together with the presence of dysarthria during the recovery of speech, suggested a cerebellar cause of the transient mutism. Various hypotheses advanced to explain the pathogenesis of this speech disorder are analyzed.

Key words Mutism · Posterior fossa
Children · Computed tomography
Magnetic resonance imaging

Introduction

Total absence of speech is not generally recognized as a possible complication of posterior fossa surgery. Recently, mutism caused by cerebellar damage has been reported [1, 2, 4–7, 9, 10]. The cerebellar site responsible for mutism is still the subject of controversy. The present authors report on a pediatric patient who developed transient mutism following removal of a large vermian tumor. Similar cases in the literature are reviewed and analyzed. The possible pathogenesis of this form of mutism is discussed.

Case report

An 8-year-old girl presented with a 1-month history of headache and occasional morning vomiting.

Examination

Neurological examination on admission showed increased intracranial pressure signs and truncal ataxia. Computed tomography (CT) scans showed a slightly hyperdense, moderately enhanced vermian tumor with calcification. Magnetic resonance imaging (MRI) scans showed a low-intensity signal and a moderately enhanced tumor on T_1-weighted imaging. The extension of the tumor into the lateral recess was clearly shown on enhanced proton imaging. There was associated obstructive noncommunicating hydrocephalus (Fig. 1).

Operation

After ventricular drainage, the tumor was approached through a midline suboccipital craniectomy with the patient prone. When the dura was opened, the cerebellar tonsils were found to have herniated to the level of C_1. Retraction of the tonsils and incision of the inferior vermis exposed a soft, grayish tumor. The tumor originated from the inferior vermis and chiefly involved the vermis, protruding into the IV ventricle and obstructing the aqueduct. The pathological diagnosis was typical medulloblastoma.

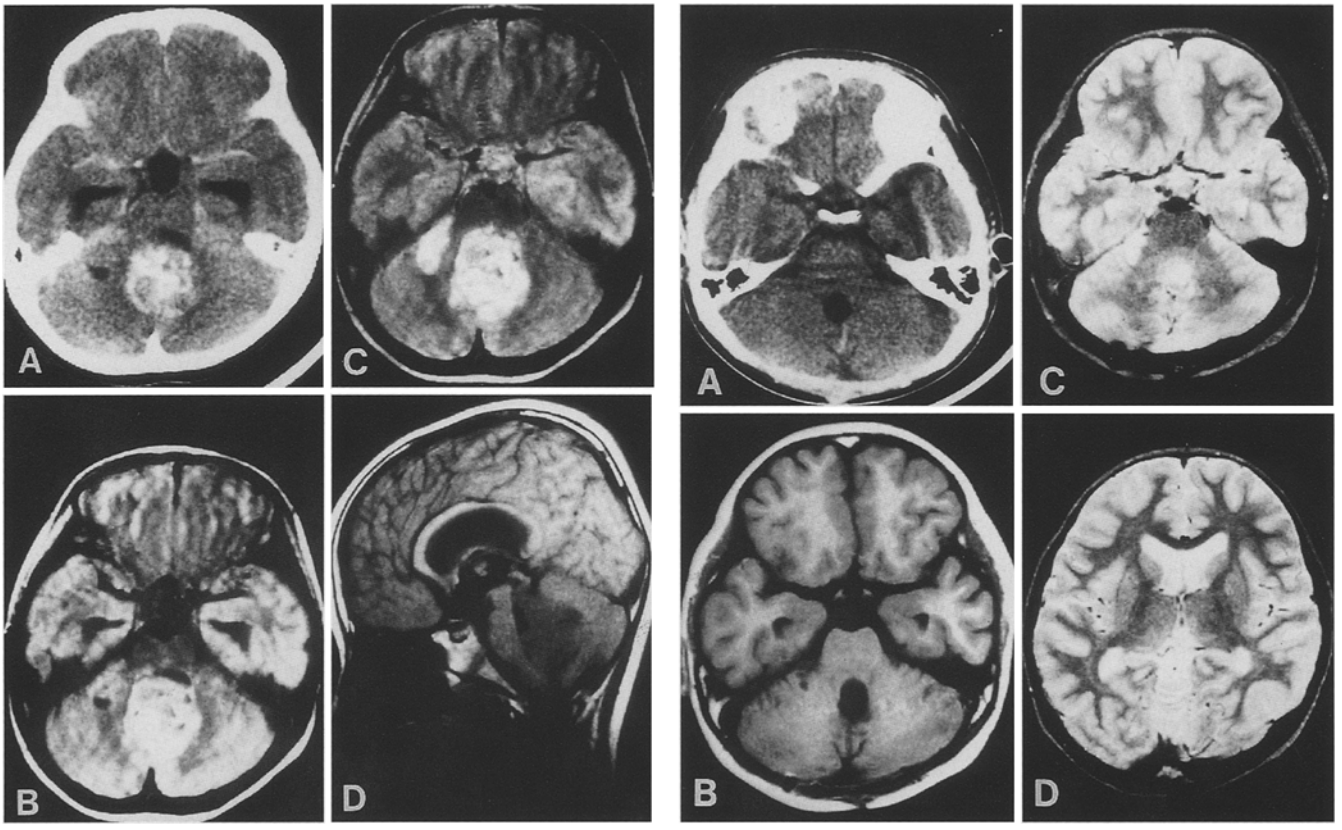

Fig. 1 **A** Computed tomographic (CT) scan with contrast enhancement demonstrating a large midline cerebellar tumor. **B** Magnetic resonance imaging (MRI): the same findings on enhanced T_1-weighted imaging. **C** The proton image showed the tumor with a lateral recess extension. **D** On the sagittal section, the large tumor almost obstructs the aqueduct

Fig. 2 **A** CT with contrast enhancement; **B** MRI, T_1-weighted image. Both scans were obtained 2 weeks after removal of the tumor and show no abnormality of either the dentate nuclei or supranuclear region. **C, D** T_2-weighted images

Table 1 Summary of 20 cases of mutism after posterior fossa tumor surgery (including the present one)

Patient age range	2–17 years	
Tumor location	Vermis–IV ventricle	16 cases
	Unknown	4 cases
Histology	Medulloblastoma	11 cases
	Astrocytoma	8 cases
	Ependymoma	1 case
IV ventricle adhesion	Present	11 cases
	Absent	9 cases
Postoperative hydrocephalus	Present	4 cases
	Absent	9 cases
	Unknown	7 cases
Lucid interval	12–96 h	
Duration of mutism	4 days–16 h	

Postoperative course

Postoperatively, the patient showed initial recovery and talked to the medical staff. However, over the next few fours her condition deteriorated; she refused food and became unable to speak. She was able to follow commands, but when asked to speak she would cry. She developed some degree of dysmetria on both sides. She did not have any long tract signs or cranial nerve palsy. CT and MRI scans showed almost complete tumor removal (Fig. 2), but no abnormalities could be recognized in either dentate nuclei or the supranuclear region. Auditory brain stem response also showed the normal reaction. No meningitis occurred after the operation. Two weeks postoperatively the girl regained monosyllabic speech. Her speech had a monotonous tone and was dysarthric with scanning.

Two months after operation, her speech had improved markedly. She spoke in sentences and more fluently. At that time, she received 40 Gy to the whole brain, 60 Gy to the tumor bed, and 20 Gy to the spinal cord. Clinically, she is progressing satisfactorily and at her 9-month follow up examination she was minimally dysarthric with mild ataxia and slight dysmetria.

Discussion

The main problem of dysarthria is disturbance in the articulation of syllables. Mutism caused by cerebellar damage can be regarded as the most extreme form of dysarthria, in which the patient cannot articulate at all. We have been able to trace 19 previously published similar cases (Table 1). After surgery, initial recovery was

seen in all cases, but over the next few hours the patients deteriorated with development of mutism. Neither brain stem nor cranial nerve signs were present. All of the patients exhibited some degree of whining or crying while being unable to speak. The duration of the speech disturbance ranged from 4 days to 16 weeks. Mutism in patients operated on for posterior fossa tumors may have a functional basis or organic basis, although very probably both sets of factors are involved [2].

There is a hypothesis that functional or emotional elements are involved, which is supported by the fact that there is recovery of verbal expression as soon as the child goes home, as noted by Humphreys [4]. However, the functional interpretation does not explain why mutism occurs in children operated on only for posterior fossa tumors and is also at variance with the fact that the intermediate phase of speech recovery is usually marked by dysarthria, which implies an organic basis in the cerebellum. It is highly probable that the emotional stress and the prolonged hospital stay may be related to the recovery of the faculty of speech and perhaps explain the refusal of food noted in the cases of Ferrante et al. [2] and also in the present case.

The anatomical localization of this speech disturbance is still vague: the lack of long tract signs and the absence of supranuclear and/or nuclear cranial nerve palsy make it difficult to dismiss the temporary inability to speak. Wisoff and Epstein [10], reporting seven cases of mutism after posterior fossa surgery, suggested that mutism may be due to focal ischemic damage caused by retraction along the fiber pathways in the middle and superior cerebellar peduncles into the upper pons and midbrain. Trauma to the floor of the IV ventricle was the causative factor in view of the delayed onset. In the present case, even though there was no abnormality on CT and MRI scans, and also auditory brain stem response showed normal function postoperatively, it is impossible to deny the possibility that retraction along the pathway was a factor.

It is commonly accepted that dysarthria is a manifestation of cerebellar lesions. The fact that all children in whom recovery of speech was reported passed through a phase of dysarthria points indirectly to a recuperative cerebellar mechanism. In addition, the concept that acute cerebellar lesions may produce not only severe dysarthria but also mutism is also supported by the report of Ammirati et al. [1], who showed bilateral hypodense areas in the dentate nuclei on postoperative CT. Bilateral involvement of the dentate nuclei in this type of mutism had already been suspected by Rekate et al. [6]. Guidetti and Fraioli [3] reported two cases of mutism after stereotactic lesioning of the bilateral dentate nuclei. Therefore, retraction of these nuclei may play a role in the development of mutism. Moreover, mutism has also occurred after bilateral thalamotomy for Parkinson's disease. This may indicate a contribution of the superior cerebellar peduncles, which connect the dentate nucleus to the thalamus [5]. However, in the present case, both CT and MRI postoperative scans showed no abnormality in the bilateral dentate nuclei.

Clinicians need to be aware that mutism may follow the removal of midline cerebellar tumors; it may be due to a transient edematous lesion of the cerebellar nuclei or, when associated with supranuclear cranial nerve palsies, to braim stem edema. In the present case, the tumor extended from the IV ventricle to the cerebellopontine angle through the cerebellomedullary fissure around the lateral recess. The vein of the cerebellomedullary fissure which drains from the inferior medullary velum and dentate nucleus passes through this pathway. Indeed, postoperative edema may induce disturbance of the venous circulation. Dysfunction of dentate nuclei might occur in the same way, but without causing hypodense areas in CT or MRI. Most previous patients who suffered muteness had large masses with extension to the lateral recess, which supports this hypothesis.

Besides this venous influence, many small perforators from the posterior inferior cerebellar artery supply the brain stem which can be recognized intraoperatively near the outlet of the IV ventricle. Coagulating the bleeding source of such perforators may cause a disturbance of the blood supply to the pons.

Mutism can occur as a partial phenomenon of akinetic mutism. Postoperative meningeal reaction and disturbance of the CSF circulation have also been described. These two factors might have a precipitating effect [2].

Although compression of structures in the brain stem cannot be ruled out by the radiological and surgical findings in this case, the lack of long track findings or cranial nerve dysfunction would favor a purely cerebellar origin for this complication. The reversibility of the mutism with a resultant period of cerebellar dysarthria also suggests a cerebellar rather than a brain stem cause of this process. Even though the appearance of this mutism is disconcerting, especially when it occurs after a few days of uneventful postoperative recovery, it is nevertheless a relatively benign phenomenon, and improvement may generally be expected to start within a few weeks.

References

1. Ammirati M, Mirazai S, Sami M (1989) Transient mutism following removal of a cerebellar tumor. Child's Nerv Syst 5:12–14
2. Ferrante L, Mastrouardi L, Acqui M, Fortuna A (1990) Mutism after posterior fossa surgery in children. J Neurosurg 72:959–963
3. Guidetti B, Fraioli B (1977) Neurosurgical treatment of spasticity and dyskinesias. Acta Neurochir [Suppl] 24:27–39
4. Humphreys RP (1989) Mutism after posterior fossa tumor surgery. In: Marlin AE (ed) Concepts in pediatric neurosurgery, vol 9. Karger, Basel, pp 57–64
5. Nagatani K, Waga S, Nakagawa Y (1991) Mutism after removal of a vermian medulloblastoma: cerebellar mutism. Surg Neurol 36:307–309
6. Rekate HL, Grubb RI, Aram DM, Hahn JK, Ratcheson RA (1985) Muteness of cerebellar origin. Arch Neurol 42:697–698
7. Sakai H, Sekino H, Nakamura N (1980) Three cases of cerebellar mutism (in Japanese). Shinkeinaika 12:302–304
8. Siegfried J, Esslen E, Gretener U, Ketz E, Perret E (1970) Functional anatomy of the dentate nuclei in the light of stereotactic operations. Confin Neurol 32:1–10
9. Volcan I, Cole GP, Johnston K (1986) A case of muteness of cerebellar origin. Arch Neurol 43:313–314
10. Wisoff JH, Epstein FJ (1984) Pseudobulbar palsy after posterior fossa operation in children. Neurosurgery 15:707–709