Transient cerebellar mutism caused by bilateral damage to the dentate nuclei after the second posterior fossa surgery

Case report

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The authors report on the case of a 6-year-old boy who underwent resection of a midline cerebellar tumor. The boy was able to speak fluently after the operation. Magnetic resonance (MR) imaging showed that the right dentate nucleus had been partially removed along with the tumor, but that the left dentate nucleus remained with the residual tumor. A second operation was performed to remove the residue, after which the child suffered mutism. Three weeks post-surgery, he could only communicate through gestures. He started speaking 1 week later and regained normal speech 2 months after the operation. Final MR imaging revealed gross-total removal of the tumor and dentate nucleus on the injured left side. The cerebellar mutism was considered to have been caused by bilateral damage to the dentate nuclei and not by unilateral damage.

**KEY WORDS** • cerebellar mutism • medulloblastoma • dentate nucleus

Cerebellar mutism has been described by Rekate, et al.,14 in 1985, and by others1,2,5,7,8,12,17,18 as a complication after resection of posterior fossa tumors. This rare condition also occurs after cerebellar hemorrhage,16 head trauma,3,6 and infectious diseases.13 Transient mutism generally occurs in young children and lasts from 1 day to 6 months, followed by a severe dysarthria, which resolves completely in 1 to 3 months.4 However, the correlation between the neuroanatomy and symptoms remains controversial. We describe a pediatric patient who presented with transient mutism after the second operation on a midline cerebellar tumor. In this report, the origin of cerebellar mutism is discussed based on pre- and postoperative MR images.

**Case Report**

**History.** This 6-year-old right-handed boy had a 3-month history of occasional vomiting and gait unsteadiness. A computerized tomography scan showed a mass in the posterior cranial fossa with obstructive hydrocephalus. He was referred to our service, and neurological examination showed bilateral papilledema, wide-based gait, and right-sided dysmetria. Magnetic resonance imaging revealed a midline posterior fossa tumor with tonsillar herniation (Fig. 1A and B).

First Operation. The patient underwent immediate surgery. Suboccipital craniotomy was performed using the Concorde position.11 The lower end of the vermis was incised longitudinally and the tumor was removed. The histological diagnosis was medulloblastoma. Magnetic resonance imaging, performed 1 day after the operation, revealed residual tumor, mainly in the left hemisphere (Fig. 1C and D). The patient was fully conscious, with no additional neurological deficit, and was able to speak fluently.

Second Operation. The second operation was performed 2 weeks later to remove the residual tumor via the same approach. The residual tumor was identified at the left cerebellar peduncle and extended caudolaterally to the left cerebellomedullary cistern. On the opposite side, a tiny tumor was confirmed at the surface of the removal cavity. Immediately after the second operation, the patient was unable to speak. He could follow commands, but cried when asked to speak. Truncal ataxia and dysmetria were also aggravated after the second surgery, but no long-tract sign or cranial nerve palsy was noted. He was also affected with behavioral abnormalities, such as emotional incontinence, poor oral intake, decreased voluntary movement, and urinary retention. Magnetic resonance images obtained 10 days after the operation demonstrated gross-total removal of the tumor with no sign suggesting damage adjacent to the dead cavity (Fig. 1E and F). Three weeks postoperatively, the boy could communicate with his parents by using hand gestures. For example, erecting one finger meant “yes,” and two fingers meant “no.” One week later, he was able to say two-sylla-
ble words such as “papa” and “mama”; 2 weeks later, he articulated two-word sentences such as “watch TV” and “eat something.” His speech was at first monotonous and dysarthric, but returned to normal 2 months after the operation.

**Discussion**

Mutism is defined as the total absence of speech in an awake patient. Recently, cerebellar mutism has become regarded as a common transient complication after the resection of posterior fossa tumors. However, the neuroanatomical correlation of the cerebellar mutism is controversial.

Guidetti and Fraioli reported that two of 47 patients who had undergone stereotactic dentatectomy for treatment of dyskinetic disease had total absence of speech postoperatively. The mutism lasted for 1 and 3 months, respectively, and resolved completely, but the authors did not describe the neuroanatomy in the two patients or the differences in anatomy among patients with and without the mutism. Rekate, et al., reported on six patients with transient mutism following removal of cerebellar tumors. The authors of the study suspected that acute bilateral damage to both cerebellar hemispheres, including the dentate nuclei, could cause the mutism.

Mutism may develop following the surgical removal of an extracerebellar lesion, such as a bilateral parasagittal meningioma or a brainstem cavernous angioma. This fact suggests that the bilateral interruption of the elements of the dentatothalamocortical pathway is responsible for the development of mutism. Pollack, et al., reported that 12 of 142 children in their study manifested postoperative mutism. The postoperative images revealed bilateral edema in the brachium pontis, and the authors postulated that mutism may have resulted indirectly from edema around the resection cavity, which may reversibly compromise the function of the dentate nuclei, and/or the afferent or efferent connections to this region bilaterally.

In the present case, the MR images obtained after the second operation revealed no sign of injury within a large portion of the cerebellar hemispheres (Fig. 1F). This consequence suggests that indirect damage was inflicted to the bilateral dentate nuclei. The patient did not suffer mutism after unilateral damage to the dentate nucleus, rather, he suffered mutism after bilateral damage.

**Conclusions**

We believe that the presence of bilateral damage is the determining factor for the development of transient cerebellar mutism. To our knowledge, this is the first report in which sequential MR images have demonstrated support for the theory that bilateral damage to the dentate nuclei is necessary for provoking transient cerebellar mutism (Fig. 2). Our experience gives further anatomical evidence for elucidating the cause of cerebellar mutism.
References


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