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Cerebellar Mutism in Children: Report of Six Cases and Potential Mechanisms

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Abstract

Cerebellar mutism is a rare finding associated with resection of posterior fossa tumors or cerebellar hemorrhages. We reviewed the medical records of six children, aged 6 to 12 years, who developed cerebellar mutism after resection of a posterior fossa mass or as a result of posterior fossa trauma. From 1989 to 1994, 210 children underwent posterior fossa resection at our institution, and four developed mutism (an incidence of 1.6%). All four patients had primitive neuroectodermal tumors. The fifth patient experienced trauma, and another patient had an arteriovenous malformation (AVM). In four children, hydrocephalus developed as a result of their tumor or AVM. Four developed cerebellar mutism 24 to 48 hours after surgery or trauma, and one developed cerebellar mutism 5 days after surgery, coincident with hydrocephalus. In one, mutism occurred after a second resection was performed for a recurrence of his posterior fossa tumor. Cerebellar mutism lasted 10 days in one patient and 2 to 8 weeks in the other four. Dysarthria was apparent in four patients during the recovery phase. We suggest trauma to the dentate nucleus and/or its outflow tract, the superior cerebellar peduncle, as a cause of reversible mutism. Because posterior fossa tumors are common in children, mutism should be recognized as an important side effect of surgery.

Introduction

Cerebellar mutism is a rarely reported finding associated with lesions of the midline cerebellum considered to represent a form of severe dysarthria. This finding has been reported in several studies, either after resection of posterior fossa tumors [1-10], with arteriovenous malformation (AVM) [8,9], or with trauma [11]. It has been difficult to estimate the incidence of cerebellar mutism, and few studies have attempted to do so. We reviewed all cases of posterior fossa tumors at Childrens Hospital Los Angeles from 1989 to 1995 in an attempt to estimate incidence; we also included additional cases with head trauma and cerebellar hemorrhage.

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Methods

We performed a retrospective chart review, surveying all cases of posterior fossa tumors at Childrens Hospital Los Angeles from 1989 through 1995. During this period, 258 children and adolescents with primary posterior fossa tumors were followed on the brain tumor service. Among this group of children, we identified seven patients, 6 to 12 years of age, with mutism after resection of their posterior fossa malignancies. Three patients were excluded from the final analysis because clinical data were insufficient to allow clear definition of their onset and course of mutism. In addition, two other children with cerebellar mutism were followed on the pediatric neurology consultation service during this period: one after a motor vehicle accident and another after hemorrhage of an AVM. All six patients were included because they presented with mutism and had no other motor or cranial nerve deficits to explain their lack of speech. No child had speech problems or cognitive deficits before the onset of the cerebellar lesions that led to surgery.

Results

The four children with posterior fossa malignancies had primitive neuroectodermal tumors (medulloblastoma). Two tumors were small and located in the paramedian vermis; the other two were larger and involved both the vermis and adjacent midline hemispheres. Three children developed hydrocephalus and required a second surgical procedure for placement of a ventriculoperitoneal shunt. These four patients represented 1.6% of all patients with posterior fossa tumors treated at Childrens Hospital Los Angeles during the 6-year study period.

One patient presented with cerebellar mutism after a motor vehicle accident in which he sustained a small contusion of his left cerebellar hemisphere and a small focal hemorrhage in the left cerebellar peduncle. The sixth patient had an AVM in the paramedian vermis and adjacent hemispheres. Hydrocephalus developed, but he did not require shunting.

The chronology of the onset and course of cerebellar mutism in our patients was informative. Four of six patients developed cerebellar mutism 24 to 48 hours after surgery or trauma, and one developed cerebellar mutism 5 days after surgery, coincident with hydrocephalus. Cerebellar mutism lasted 10 days in one patient and 2 to 8 weeks in the other four. Dysarthria was evident in four patients during the recovery phase.

The sixth patient exhibited cerebellar mutism on undergoing a second resection for a recurrence of his posterior fossa tumor. His mutism lasted only 24 hours.

Discussion

We report a group of children who were transiently mute after a posterior fossa resection. These children did not have major cranial nerve or motor deficits, and their mental status and cognition were not significantly impaired.

Mutism is described as the inability of a cognitively alert patient with the ability to process speech without evidence of apraxia to produce verbal output [4]. It is distinct from aphasia, in which there are typical cerebral cortical lesions [12]. It is also distinguished from oropraxia, in which performance of learned motor skills of face, lip, and tongue are affected without an apparent neuroanatomic lesion [12]. Cerebellar mutism is a severe dysarthria or anarthria in which there is profound impairment of fluency, articulation, and modulation of speech [8,12]. Cranial nerve function is uniformly intact in patients with cerebellar mutism [8]. Cerebellar mutism is transient, and resumption of baseline function is common [4].

Patients with cerebellar mutism have lesions in the paramedian cerebellar vermis or floor of the fourth ventricle [1-9,11]. Cerebellar mutism apparently may be produced by insult to the cerebellar midline. The cerebellum is critical in the fluency of speech. Fraioli and Guidetti performed stereotaxic ablation on the dentate nucleus and its outflow tract, the superior cerebellar peduncle, which resulted in reversible mutism [13]. This may explain the development of mutism in patients with posterior fossa lesions. Hydrocephalus may be an exacerbating factor [9]. There is increasing interest in the potential role of the cerebellum in processing of learning and memory [14,15]. Although formal cognitive testing was not performed, cognitive deficits were not obvious in our patients, and they attempted to communicate nonverbally.

Cerebellar mutism may be more common than is appreciated. Our estimated incidence of cerebellar mutism in 1.6% of children and adolescents with posterior fossa tumor may be too low. Three children were excluded and we fear this finding may not have been recognized in others, and details are not available for our retrospective review. Further clinical studies are needed to explain and define clearly the incidence of cerebellar mutism.

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