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Review paper

Transient mutism following removal of a cerebellar tumor

A case report and review of the literature

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Abstract. A 14-year-old boy developed mutism 24 h after the removal of a vermian low-grade astrocytoma. The mutism was not accompanied by long tract signs or cranial nerve palsies. He started to regain his speech 3 weeks postoperatively, and 4 months after the operation he was minimally dysarthric. Seven similar cases of transient muteness following cerebellar operations and not accompanied by long tract signs or cranial nerve palsies have been reported in the literature. In most of them there was delayed postoperative onset of the mutism. In all patients the recovery of speech started to appear 2 weeks to 3 months postoperatively and passed through a dysarthric phase. The absence of long tract or other brain stem signs, together with the presence of dysarthria during the recovery of speech, suggests a cerebellar cause for the transient muteness.

Key words: Cerebellar neoplasm – Surgery – Mutism.

Total absence of speech is not generally recognized as a possible complication of posterior fossa surgery [5–7], and when it occurs the neurosurgeon is usually baffled. Efforts to clarify the nature of this speech disturbance lead to unsuccessful investigation of multiple diagnostic possibilities, including that of a conversion reaction [9]. We report the case of a child who developed transient mutism following removal of a large vermian tumor. Similar cases reported in the literature are reviewed and analyzed.

Case report

A 14-year-old boy presented with a 2-week history of generalized headache. Neurological examination was positive for a right abducens paresis, horizontal and vertical nystagmus, right-sided dysmetria and ataxia of gait. Computerized tomography (CT) scans showed a hypodense, moderately enhancing vermian tumor partially occupying the IV ventricle. There was associated obstructive non-communicating hydrocephalus (Fig. 1). He underwent a suboccipital craniectomy in the sitting position with splitting of the vermis and complete, uneventful, removal of a prevalently cystic cerebellar tumor chiefly involving the vermis and bulging into the IV ventricle. The pathological diagnosis was grade 1 astrocytoma. Immediately after the surgery he was awake and fully oriented with no new neurological deficit. On the 2nd post-operative day he suddenly became unable to speak. He was able to follow commands, but when asked to speak he would cry. The right-sided dysmetria increased and he developed some degree of dysmetria on the left side as well. He remained able to swallow and did not have any long tract signs or cranial nerve palsies. CT scans showed complete tumor removal, unchanged ventricular size and bilateral hypodense areas at the approximate level of the dentate nuclei (Fig. 2). Three weeks post-operatively he was able to pronounce two-syllable words. Four weeks after the surgery he underwent placement of a ventriculoperitoneal shunt because of progression of the hydrocephalus. Five weeks after the first operation he was dysarthric but able to speak in sentences of two or three words. He was discharged 6 weeks after removal of the tumor with an ataxic gait, positive cerebellar tests (right more than left) and a markedly dysarthric speech. When last seen at follow-up, 4 months post-operatively, he was minimally dysarthric, had minimal gait ataxia and almost unnoticeable right-sided dysmetria. CT scans showed absence of the previously noted cerebellar hypodensities and good ventricular decompression (Fig. 3).

Discussion

Rekate, in 1985, reported six children, aged 2–11 years, who were unable to speak after posterior fossa operation for vermian neoplasms. No brain stem sign was present [8]. Both of the patients exhibited some degree of whining or crying while unable to speak; one of the two developed his speech problem on the 4th post-operative day. The duration of the speech disturbance ranged from 3 weeks to 3 months, and when the speech returned it was at first dysarthric. Volcan, in 1986, reported an 8-year-old girl who, immediately after removal of a vermian medulloblastoma, was unable to speak, though she was able to cry and whine [9]. She was alert, able to follow commands, and had no new postoperative neurological deficit. Neuroradiological investigations did not give any clue as to the cause of the problem. Two weeks after surgery she regained monosyllabic speech, and 1 month post-operatively she was dysarthric but able to speak in sentences. Our patient had a similar clinical course in that he lost his speech 2 days post-operatively, remained alert with no long tract findings or cranial nerve deficit, and

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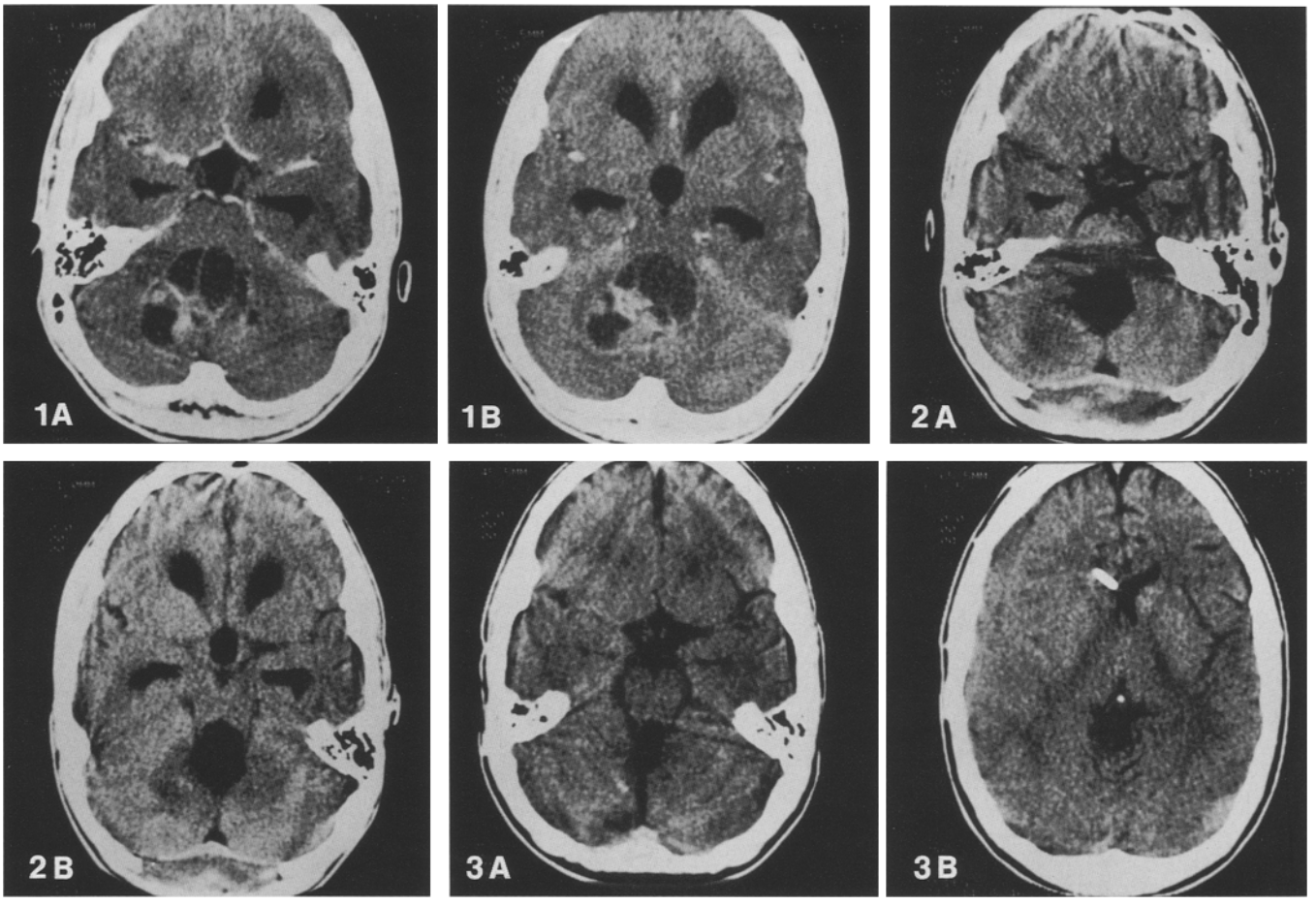

Fig. 1. CT scans show a partially hypodense mass involving the vermis and the right medial cerebellar hemisphere, **A** and **B**. **B** Hydrocephalus is present

Fig. 2. CT scans obtained on the 2nd post-operative day show total removal of the tumor, **A** and **B**. Hydrocephalus is unchanged, **B**. Bilateral hypodense areas are noted at the approximate level of the dentate nuclei, **B**

Fig. 3. **A** CT obtained 4 months after the removal of the neoplasm shows disappearance of the hypodense areas at the level of the cerebellar hilus shown in the previous figure. **B** The ventricles are small and the tip of the shunt tube is in a good position in the right frontal horn

started to have a dysarthric speech few weeks post-operatively.

The anatomical localization of this speech disturbance is unclear: the lack of long tract findings, the absence of supranuclear and/or nuclear cranial nerve palsies and swallowing difficulties make it difficult to dismiss the inability to speak exhibited by these patients as aphonia in the context of a pseudobulbar palsy [2]. On the other hand, it is well accepted that dysarthria is a manifestation of cerebellar lesions [1]. The fact that in all of the children reported the recovery of speech passed through a phase of dysarthria points indirectly to a recovering cerebellar mechanism. That acute cerebellar lesions may produce not only severe dysarthria but also mutism is supported, as already noted by Rekate [8], by the report of Guidetti and Fraioli, who observed total inability to speak for up to 3 months in two patients in whom simultaneous and bilateral lesions of the dentate nuclei were sterotactically created in order to treat spasticity [3, 4]. Bilateral involvement of the dentate nuclei in this type of mutism had, therefore, already been suspected by Rekate [8]. The fact that in our patient the improvement of speech closely paralleled the disappearance of the bilateral hypodense areas at the approximate level of the dentate nuclei lends support to this theory. Perhaps serial magnetic resonance imaging scans could be of help in better pinpointing the anatomical location of this disturbance.

This type of muteness not associated with cranial nerve palsies must be distinguished from the type that is associated with cranial nerve palsies, mainly supranuclearly located, that was described after removal of cerebellar midline neoplasms by Wisoff and Epstein in 1984 [10]. This postoperative onset of supranuclear cranial nerve palsies, associated at times with inability to speak and emotional incontinence, has been thought to represent a pseudobulbar palsy due to edema secondary to cerebellar retraction, tracking along the cerebellar peduncles into the pons and midbrain [10].

Clinicians must be aware that mutism may follow removal of midline cerebellar tumors; it may be due to transient edematous lesions of the cerebellar nuclei or, when associated with supranuclear cranial nerve palsies, to brain

stem edema. Even though the appearance of this mutism is disconcerting, especially when coming about after a few days of uneventful post-opeative recovery, it is nevertheless a relatively benign phenomenon and improvement may usually be expected to start in 4–6 weeks.

References

1. Adams, RD, Victor M (1981) Principles of neurology. McGraw-Hill, New York St Louis, pp 65–66
2. De Jong RN (1979) The neurologic examination. Harper & Row, Philadelphia, pp 264–265
3. Fraioli B, Guidetti B (1975) Effects of stereotactic lesions of the dentate nucleus of the cerebellum in man. Appl Neurophysiol 38:81–90
4. Guidetti B, Fraioli B (1977) Neurosurgical treatment of spasticity and dyskinesias. Acta Neurochir [Suppl] 24:27–39
5. Horwitz NH, Rizzoli HV (1982) Postoperative complications of intracranial neurological surgery. Williams & Wilkins, Baltimore London, pp 35–181
6. Humphreys RP (1982) Posterior cranial fossa brain tumors in children. In: Youmans JR (ed) Neurological surgery. A comprehensive reference guide to the diagnosis and management of neurosurgical problems, vol 5. Saunders, Philadelphia London, pp 2733–2758
7. Matson DD (1969) Neurosurgery of infancy and childhood. Thomas, Springfield Ill, pp 410–479
8. Rekate HL, Grubb RL, Aram DM, Hahn JF, Ratcheson RA (1985) Muteness of cerebellar origin. Arch Neurol 42:697–698
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

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