

Mutism After Total Removal Of An Exophytic Pontine Glioma

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Abstract : Total absence of speech is not generally recognized as a possible complication of posterior fossa surgery. Only a few reports of cerebellar mutism have been published so far. In this paper, we present the case of, a patient with a brain stem tumour,

who developed mutism without any additional neurological deficit after total tumour removal and which resolved six months after surgery.

Key Words : Brain stem, Mutism, Neoplasm, Surgery.

INTRODUCTION

The term 'mutism' describes patients, who lack spontaneous speech despite the appearance of alertness. According to Benson (2) there are five neurological conditions causing muteness. The first is damage to the Broca's area which may lead to total absence of speech. The second is damage to the supplementary motor area of the dominant hemisphere. Damage to the reticular formation of the mesencephalon may also leave a patient mute in the context of akinetic mutism. The fourth is pseudobulbar palsy due to diffuse bilateral cerebral hemispheric dysfunction. Muteness has been described also following bilateral thalamotomy for Parkinson's disease. In addition to these conditions, there are a few cases who developed transient mutism following removal of large vermian or IV. ventricle tumours (1,3,4,6,7,8,9). Although compression of structures in the brain stem cannot be ruled out by the radiological and surgical findings in these cases, the lack of long-track findings or cranial nerve dysfunction would favour a cerebellar origin for this syndrome.

In this report we present a patient with an exophytic brain stem tumour, who after total excision of the tumour developed mutism which resolved after six months.

CASE REPORT

A 13-year-old girl was admitted to our institution with a two months' history of headache, nausea and vomiting. Neurological examination revealed papilloedema with retinal haemorrhage and difficulty of walking on a straight line. MRI studies revealed a solid and homogeneous tumour 4 cm. in diameter which arose from the pons and exophytically grew into the fourth ventricle (Figure 1). The intensity of the tumour was the same as the pontine tissue itself and it did not enhance. In addition there was obvious hydrocephalus with periventricular oedema. The upper part of the IV. ventricle was also trapped and dilated, mimicking a tumour cyst.

Operation: A posterior fossa exploration was done in the supine position with suboccipital craniectomy and CI laminectomy. By splitting the inferior vermis the fourth ventricle was reached and a round shaped solid tumour was exposed. The tumour had no adherence to the cerebellar tissue and seemed to arise from the pons. The tumour including its intrapontine component was totally removed with the aid of an ultrasonic aspirator. After tumour excision the cerebrospinal fluid drainage was opened and the dilated aqueductus sylvii was visible. The periaqueductal region looked normal.

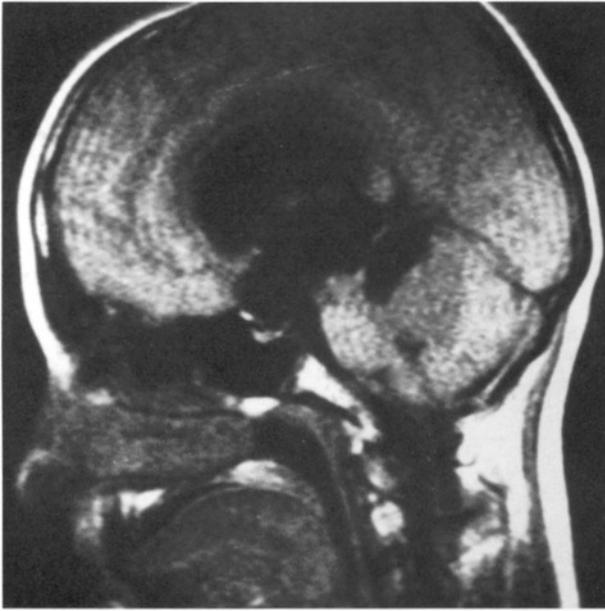


Fig. 1 : There is an exophytic brain stem tumor growing into the IV. ventricle. The upper part of IV. ventricle is dilated, mimicking a tumor cyst.

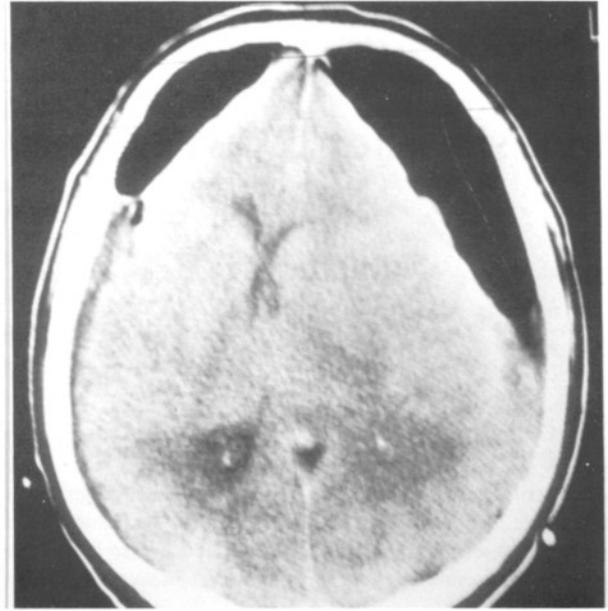


Fig. 2 : After tumor excision there is no residual tumor within the IV. ventricle. The IV. ventricle is dilated and aqueductus sylvii seemed to be open.

Histopathological examination of the tumour revealed a pilocytic astrocytoma. In the early postoperative period neurological examination of the patient was within normal limits except the mutism. Antioedematous therapy was started immediately and continued for six weeks. Since there was no remission in the size of the ventricles although the tumour seemed to be removed totally a V-P shunt was applied.

Follow-up: The postoperative period was uneventful. She was discharged on the 10th postoperative day. Radiotherapy and chemotherapy were not recommended. The mute state persisted for 4 months, was followed by a severe cerebellar speech disturbance and then returned to normal patterns after six months. A follow-up MRI after eight months was within normal limits with no evidence of recurrent tumour tissue (Fig. 2).

DISCUSSION

Although dysarthria produced by cerebellar dysfunction is a well known phenomenon, total absence of speech is not generally recognized as a possible complication of posterior fossa surgery. According to the study by Sakai et al (9), the main pro-

blem is disturbance in the articulation of syllables, but the exact anatomical location of this type of mutism is still unclear. Only a few reports of cerebellar mutism have been published so far (1,3, 6,7,8,9). All these reported that the patients, like ours, were fully alert. The receptive vocabulary understanding and the lower cranial nerves were not involved. The fact that in all the children reported the recovery of speech passed through a phase of dysarthria points indirectly to a recovering cerebellar mechanism. Guidetti and Fraioli (4,5) observed total inability to speak in two patients in whom simultaneous and bilateral lesions of the dentate nuclei were stereotactically created in order to treat spasticity. Ferrante (3) suggested postoperative spasm of the arteries supplying the cerebellum and the brain stem, causing ischaemia. This possibility may be supported by the fact that 60% of the reported cases were able to speak immediately after surgery when cerebellar perfusion was not disturbed which was not the fact in our case.

In any event, clinicians must be aware that mutism may follow after posterior fossa surgery. We hypothesize that it may be due to transient aedematous lesions of the cerebellar nuclei secondary to cerebellar retraction. For this reason we strongly recommend

constant retraction of both cerebellar hemispheres during surgery should be avoided and antioedematous therapy should be started immediately.

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