CASE REPORT

Transient cerebellar eye closure after posterior fossa surgery in a 5-year-old child

Munir J. Nasser

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Abstract

Introduction Transient cerebellar eye closure (TCES) is a rare complication of cerebellar tumor surgery in children. The pathogenesis of this problem remains unclear, and controversy exists regarding whether it is a purely psychogenic disorder or an organic syndrome. The anatomical substrate for this transient eye closure remains unknown. Most of the cases reported were associated with the syndrome of mutism.

Case report We encountered a case of post-operative TCES in a 5-year-old girl with posterior fossa tumor.

Results We are presenting this rare complication with the hope of elucidating further on clinical course of illness and literature review on the possible pathophysiological mechanism.

Keywords Transient eye closure · Mutism · Cerebellar surgery

Introduction

Transient eye closure after cerebellar surgery was reported first time by Nashold and Slaughter [1] in 1969. This phenomenon was seen together with profound generalized hypotonia, hyporeflexia, and hypokinesis. Gaskill and Marlin [2] in 1991, reported four children with transient cerebellar eye closure (TCES) after posterior fossa surgery, in three of them the syndrome of mutism was present.

Coriene et al. [3], in 2002, reported a case of TCES in a 14-year-old girl where they extensively described the

patient's post-operative course of illness. In literature review, six cases with TCES has been reported; all of them were associated with variable features of posterior fossa syndrome (PFS).

In our case, TCES was the only post-operative complication that occurred after posterior fossa surgery, otherwise the patient was normal. This case may represent the first to report TCES without any other cerebellar findings.

Case report

History

A 5-year-old right-handed girl was admitted to our hospital with a history of repeated attacks of vomiting and mild torticollis toward the right side for 1 week the neurological examination was unremarkable. Magnetic resonance imaging (MRI) of the brain revealed a large contrast-enhancing mass lesion in the posterior fossa that invaded the right cerebellar hemisphere and going down through the foramen magnum (Fig. 1a, b, c). No hydrocephalus was seen in spite of the tumor filling most of the fourth ventricle.

The patient was operated in the prone position, after midline incision, suboccipital craniotomy was made, and then the dura was opened in Y-shaped fashion. The tumor was macroscopically resected initially using the cavitron ultrasonic surgical aspirator. Tumoral tissue adhering to the brain stem and to the floor of the fourth ventricle was resected with the use of a microscope; while shaving the tumor from the floor of the fourth ventricle, several attacks of hypotension occurred but the vermis was not touched.

Cerebellar retractors were not used intraoperatively. Post-operative MRI confirmed almost total resection of the tumor (Fig. 1d).

M. J. Nasser (⊠)

King Fahd Hospital, King Faisal University, P.O. box 40010, Al- Khobar 31952, Saudi Arabia e-mail: munirlnasser@yahoo.com



Fig. 1 a Pre-operative MRI T1 sag. view, the tumor seen as hypointense lesion in the posterior fossa. b Pre-operative MRI T1 sag. view with good enhancement of the tumor. c Pre-operative MRI, axial view post contrast, the tumor growth mainly to right side and compressing brain stem. d Post-operative MRI T1 sag. view post contrast, no tumor residual

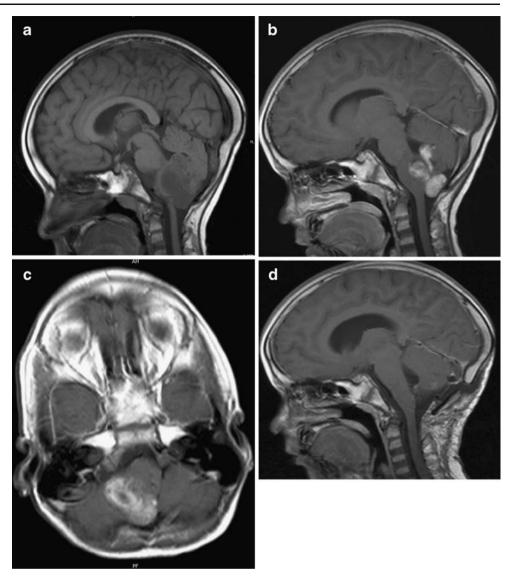
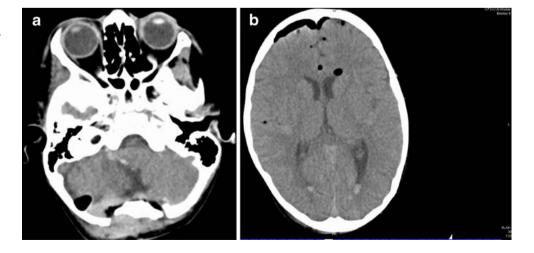


Fig. 2 a Post-operative CTscan of brain showing craniotomy site. b Post-operative CT scan of brain showing minimal blood in the occipital horns





Histological examination disclosed the diagnosis of a pilocytic astrocytoma. After the surgery, the patient was kept on elective ventilation till the next morning to avoid straining and CSF leak from the operative site; brain CT was performed (Fig. 2a, b) before extubation which showed minimal post-operative blood at the operative site with no hydrocephalic changes. When the patient woke up, she refused to open her eyes and we initially thought this problem was resulted from the general condition of the patient. On the next day, she became very alert and talkative although she still had this continuing problem without any other cerebellar findings. The patient had remained the same for about 10 days; she then started opening the left eye most of the time, while the right eye was closed. On follow-up examination, the right eye closure recovered gradually and on the sixth post-operative week, this finding was resolved completely.

Discussion

PFS is a well-known clinical condition following posterior fossa lesions in children. In 1958, Daly et al. [4], for the first time, described mutism following posterior fossa surgery in children for which Rekate et al. [5] introduced the term 'cerebellar mutism'. Since then, approximately 226 children and adolescents have been reported with this condition. Although posterior fossa tumor resection is the most common cause of the PFS, patients with a traumatic [6], vascular [7], and infectious etiology [8] have been described. With regard to the incidence, variable figures of cerebellar mutism ranging from 12% [9] to 29% [10] have been reported after posterior fossa surgery.

In children, TCES seems to be associated with cerebellar mutism syndrome (CMS). In the literature review, six cases were reported with TCES, all of them associated with CMS or other features of PFS.

In our case report, TCES was the only finding without any other cerebellar complications.

The underlying mechanism of (TCES) is not clear, Humphry [11] suggested a psychological mechanism of TCES and he used the term refusal to open the eyes.

Goldstien and Cogan [12] describe it as (apraxia of eye-lid opening). Pathophysiological substrate of 'cerebellar eye-lid apraxia' remains unclear and several hypothetical explanations

are suggested among which hypoperfusion of cerebellar, frontal, and occipital hemispheres that may explain connections disruption between the cortical regions and the cerebellum.

Conclusion

This is the first report on TCES as a single complication after posterior fossa surgery. Most of the reported cases of TCES are associated with mutism. The prognosis for recovery is excellent. The exact mechanism is not clear.

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