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CASE REPORT

Fluorodeoxyglucose positron emission tomography/computed tomography findings in a patient with cerebellar mutism after operation in posterior fossa

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Summary Cerebellar mutism is a transient period of speechlessness that evolves after posterior fossa surgery in children. Although direct cerebellar and brain stem injury and supratentorial dysfunction have been implicated in the mediation of mutism, the pathophysiological mechanisms involved in the evolution of this kind of mutism remain unclear. Magnetic resonance imaging revealed dentatothalamocortical tract injuries and single photon emission computed tomography showed cerebellar and cerebral hypoperfusion in patients with cerebellar mutism. However, findings with ^{18}F-fluorodeoxyglucose positron emission tomography/computed tomography (FDG PET/CT) in this group of patients have not been documented previously. In this clinical case, we report a patient who experienced cerebellar mutism after undergoing a posterior fossa surgery. Right cerebellar and left frontal lobe hypometabolism was shown using FDG PET/CT. The FDG metabolism of both the cerebellum and the frontal lobe returned to normal levels after the resolution of the mutism symptoms.

1. Introduction

Posterior fossa syndrome consists of transient cerebellar mutism, cognitive symptoms, and neurobehavioral abnormalities.[1] It was first reported in 1985 as a complication of posterior fossa surgery.[2] Although a transient nature was recognized as the hallmark of this type of mutism,

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development after the resection of a cerebellar mass lesion, delayed onset after intervention, and association with other neurological, emotional, and behavioral disturbances were described as core features of this syndrome.[3]

The incidence of postoperative cerebellar mutism ranges between 11% and 29%.[4] It is more commonly observed after the resection of posterior fossa tumors in children; however, trauma, vascular incidents, or infections may also be followed by this complication.[4] Patients with medulloblastomas and/or brain stem invasion are at a greater risk of experiencing this condition compared with those who have pilocytic astrocytoma or ependymoma.[5]

After the first description of this syndrome appeared, more than 400 cases with cerebellar mutism have been reported, and several hypotheses have been suggested to explain the clinical features of this complex syndrome. Because surgery is the primary event, surgical injury to dentate nuclei of the cerebellum has been proposed as the primary cause of the initiation of mutism. However, the delayed onset of mutism after surgery and the presence of concomitant neurological symptoms have raised questions about the association of supratentorial structures to this clinical picture. On one hand, there were some studies with single photon emission computed tomography (SPECT) showing cerebellar hypoperfusion in mute patients with normalization of blood flow after mutism had resolved[6,7]; on the other hand, SPECT studies linking mutism to hypoperfusion of contralateral frontal cortex or other supratentorial targets were also reported.[1,8] This asymmetric blood flow in cortical areas contralateral to a remote cerebellar lesion, which is described previously as crossed cerebello-cerebral diaschisis, is a well-known phenomenon, and injury to the dentatothalamocortical pathway is suggested as the cause.[3]

We describe a patient who underwent cerebellar surgery and developed mutism after the procedure. Metabolic imaging with 18F-fluorodeoxyglucose positron emission tomography/computed tomography (FDG PET/CT) was performed at both mutistic and postmutistic phases. The FDG metabolism of the primary surgical site and the cortical areas is discussed, and the literature is reviewed.

2. Case report

A 17-year-old male patient was admitted to the hospital complaining of a headache. His history showed that he has been suffering from headaches and vomiting for 4 months and 3 weeks, respectively. His Glascow Coma Scale was 15, and his neurological examination yielded completely normal results. Cranial magnetic resonance imaging (MRI) showed a large arachnoid cyst formation (47 × 36 × 61 mm) at the fourth ventricle with significant brain stem and cerebellum compression accompanied with mild tetraventricular hydrocephalus (Fig. 1A and B). The patient underwent an operation at the neurosurgery department. The operation was performed with the patient placed in a sitting position, and a midline suboccipital approach was used. After the suboccipital craniotomy, the dura was opened and both cerebellar hemispheres were retracted

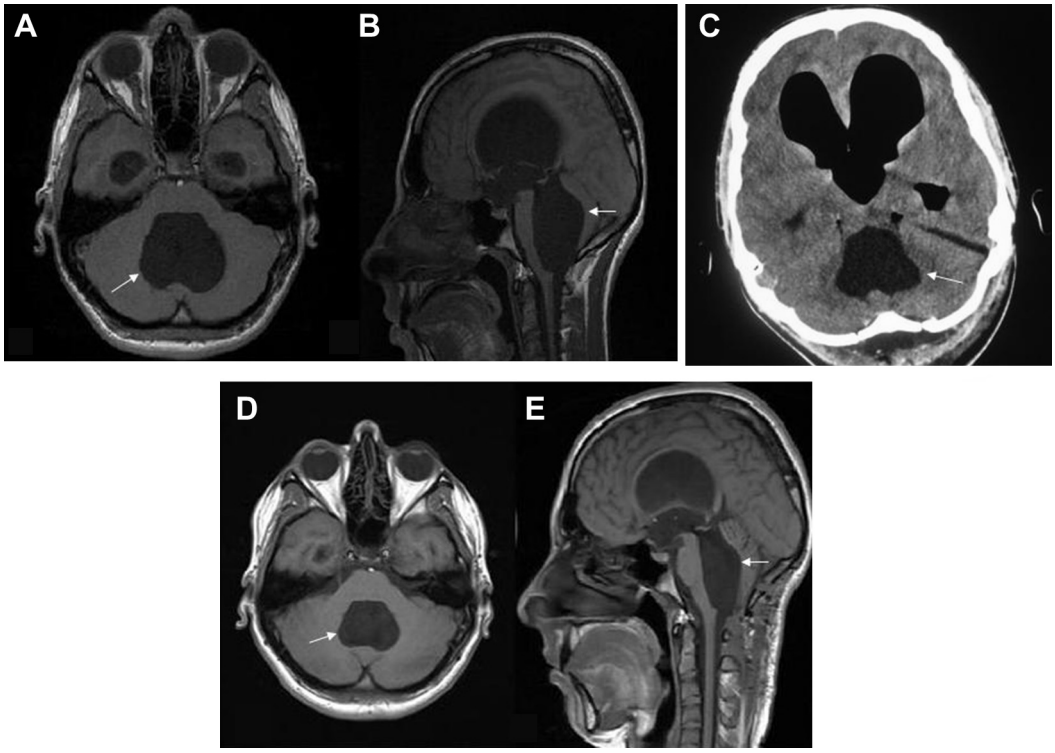

Figure 1 Preoperative (A) axial and (B) sagittal T1-weighted magnetic resonance imaging show large arachnoid cyst formation at the fourth ventricle (arrow) with significant brain stem and cerebellum compression. Pneumocephalus and ventricular dilatation are shown on (C) postoperative computed tomography. T1-weighted (D) axial and (E) sagittal magnetic resonance imaging performed 3 months after surgery reveal a decrease in cyst size (arrow) as well as compression of the cerebellum and brain stem.