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Case Report

Bobble-head doll syndrome secondary to a giant posterior fossa arachnoid cyst: A rare case report

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ABSTRACT

Background: Bobble-head doll syndrome (BHDS) is a rare movement disorder characterized by rhythmic head oscillations at a frequency of 2–3 Hz, typically in a yes–yes or no–no pattern. It is most commonly associated with suprasellar arachnoid cysts, causing pressure on midbrain structures, thalamus, and cerebrospinal fluid (CSF) pathways, resulting in movement abnormalities and hydrocephalus. While it usually presents in children aged 3–5 years, late adolescent or adult presentations are exceedingly uncommon.

Case Description: We report a 23-year-old male with progressive involuntary side-to-side head movements since childhood, which had worsened over the preceding year and led to gait instability requiring support. He had a history of ventriculoperitoneal shunt placement in infancy for hydrocephalus, later removed due to infection. Magnetic resonance imaging revealed a large posterior fossa arachnoid cyst compressing the third ventricle, with intermittent CSF obstruction contributing to progressive neurological decline. The patient underwent endoscopic cyst fenestration, which restored CSF circulation and relieved mass effect. Head movements subsided almost completely within 48 h postoperatively, and gait improved significantly over 2 weeks. Six-month follow-up imaging confirmed resolution of ventricular compression and restoration of aqueductal flow.

Conclusion: This case highlights a rare delayed presentation of BHDS in adulthood, underscoring the importance of timely surgical intervention. Endoscopic fenestration proved highly effective in restoring CSF dynamics, resolving abnormal movements, and improving functional outcomes, and may offer advantages over CSF diversion alone.

Keywords: Arachnoid cyst, Bobble-head doll syndrome, Endoscopic fenestration

INTRODUCTION

Bobble-head doll syndrome (BHDS) is a rare neurological movement disorder characterized by continuous, rhythmic head oscillations at 2–3 Hz, typically anteroposterior (yes–yes) or lateral (no–no), which worsen with excitement and diminish during sleep.[1] It is most commonly associated with suprasellar arachnoid cysts exerting pressure on midbrain structures, including the red nucleus, dentatorubrothalamic pathway, and medial dorsal thalamus.[9] First described by Benton *et al.*[1] in 1966, fewer than 60 cases have been reported worldwide.[10] Although usually

recognized in children aged 3–5 years, late adolescent or adult presentations are exceptionally rare.[5]

The pathophysiology of BHDS remains incompletely understood. The mechanical compression hypothesis suggests cyst-related deformation of midbrain structures causes abnormal neuronal discharges, while the thalamic hypothesis proposes medial dorsal thalamic compression disrupts circuits involving the thalamus, basal ganglia, and cervical spinal motor neurons. The more recent cerebrospinal fluid (CSF) flow hypothesis postulates that head oscillations develop as compensatory movements to facilitate CSF passage through obstructed ventricular pathways, explaining their postural variation.[8]

Clinically, BHDS may begin as low-amplitude tremors and progress to disabling movements, hydrocephalus, gait instability, and cognitive decline. This case report highlights a rare adult presentation, emphasizing the role of early recognition, neuroimaging, and minimally invasive surgical management.

CASE REPORT

A 23-year-old male presented with progressive involuntary head movements for 19 years, which had worsened significantly in the past year. Initially slow, low-amplitude, and intermittent, the movements became continuous, rhythmic, and side-to-side, resembling a "no–no" pattern [Video 1]. While not functionally limiting during childhood, they gradually led to severe gait instability, rendering the patient unable to walk without support in the past 3 months. There was no history of headache, vomiting, seizures, fever, or neck stiffness.

His history included communicating hydrocephalus diagnosed at 2 months of age, for which a right Frazier's ventriculoperitoneal (VP) shunt was placed. The shunt was removed 2 months later due to infection, with no further neurosurgical intervention. He remained neurologically intact until the age of 4 years, when his parents noted head oscillations, initially dismissed as benign. These persisted and worsened into adulthood, eventually prompting medical evaluation.

Neurological examination revealed continuous, rhythmic horizontal head oscillations that attenuated during sleep and could be voluntarily suppressed briefly. There were no limb tremors, dystonia, or bradykinesia. Cranial nerves were intact, with no optic atrophy or raised intracranial pressure. Motor strength and reflexes were normal, and coordination was preserved. However, gait was broad-based and unsteady, requiring support. Cognitive functions were intact.

Magnetic resonance imaging (MRI) demonstrated a large posterior fossa arachnoid cyst ($7 \times 8.2 \times 10.5$ cm) compressing the third ventricle. The lesion was hypointense on T1 and hyperintense on T2, with no contrast enhancement, excluding craniopharyngioma or cystic glioma. Mild ventricular dilatation suggested obstructive hydrocephalus from the foramen of Monro compression [Figure 1]. Cine MRI flow studies showed absent CSF pulsations at the aqueduct of Sylvius, indicating intermittent obstruction. Based on clinical and radiological findings, a diagnosis of BHDS secondary to a posterior fossa arachnoid cyst was established. Differential diagnoses, including craniopharyngioma, cystic choroid plexus papilloma, and aqueductal stenosis, were excluded on imaging.

Given progressive neurological decline, endoscopic cyst fenestration was planned. The procedure was performed under general anesthesia with the patient in the prone position. A 30° rigid neuroendoscope (Karl Storz, Germany) was introduced through a left occipital burr hole. On entering the cyst cavity, clear CSF-like fluid was encountered. The cyst wall was sharply opened using cold microscissors, and fenestration was widened using bipolar coagulation under direct visualization. The cerebellar tonsils, medulla, and floor of the fourth ventricle served as key anatomical landmarks during the dissection. The fenestration into the cisterna magna measured approximately 1.5 cm in diameter, ensuring adequate CSF communication between the cyst and basal cisterns. Free CSF flow was confirmed before the gradual withdrawal of the endoscope [Figure 2]. The procedure was completed without any complications. Postoperatively, the patient exhibited >90% reduction in head oscillations within 48 h, with complete resolution by the end of the 1st postoperative week [Video 2]. Gait performance was objectively assessed using the Tinetti Balance and Gait Scale, improving from 13/28 preoperatively to 27/28 2 weeks after surgery, indicating near-complete restoration of functional mobility. The patient was able to ambulate independently without support on postoperative day 14. He was discharged on postoperative day 3, having experienced an uneventful recovery with no neurological or wound-related complications. At the 6-month follow-up, both clinical and MRI evaluations confirmed sustained cyst decompression, restored CSF flow, and complete resolution of symptoms.

DISCUSSION

BHDS is a rare and poorly understood movement disorder, most often linked to suprasellar arachnoid cysts. The hallmark rhythmic head oscillations are believed to result from mechanical compression of midbrain structures, including the red nucleus, dentatorubrothalamic tract, and medial dorsal thalamus. Several pathophysiological mechanisms have been proposed. Benton *et al.*[1] first suggested that cystic lesions distort adjacent neural structures, leading to abnormal motor control, though this lacked direct

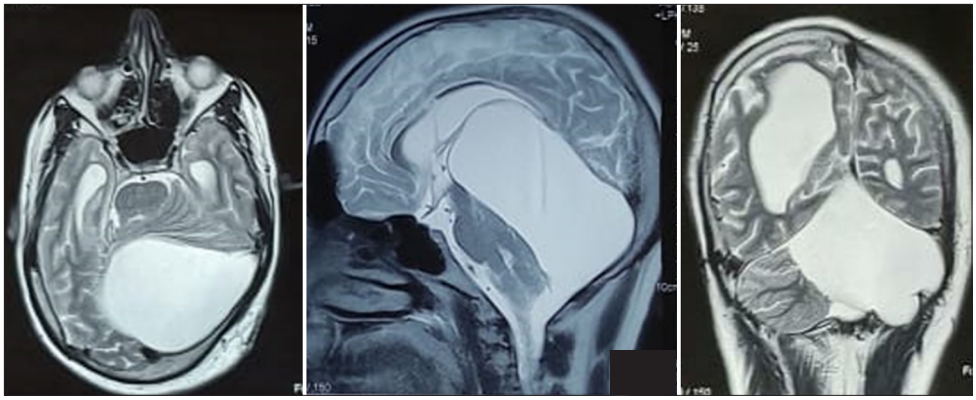


Figure 1: Magnetic resonance imaging T2 axial, sagittal, coronal sections showing posterior fossa arachnoid cyst of size 7 × 8.2 × 10.5 cm size causing mass effect on cerebellum, brainstem, 4th and 3rd ventricles with hydrocephalus respectively.

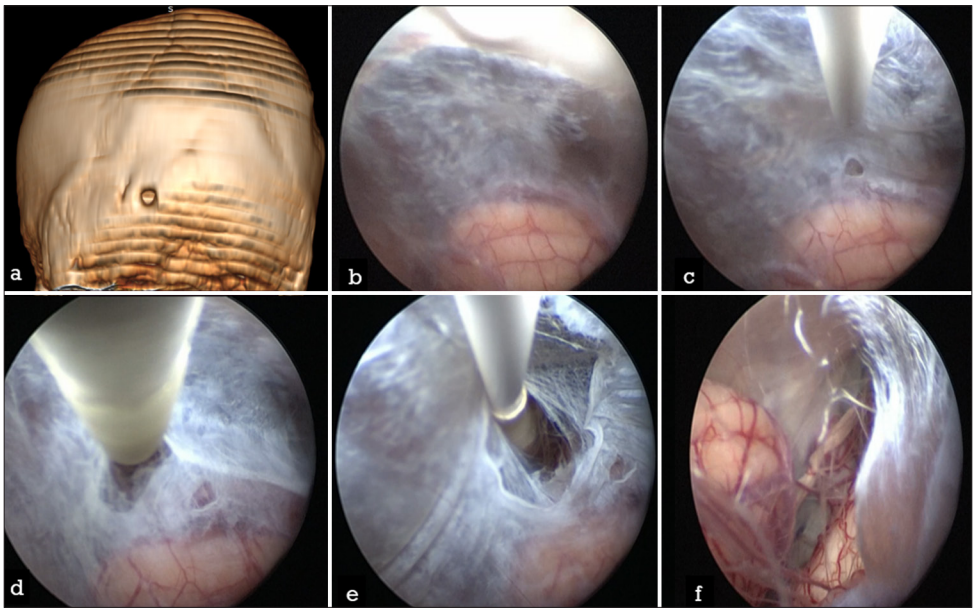
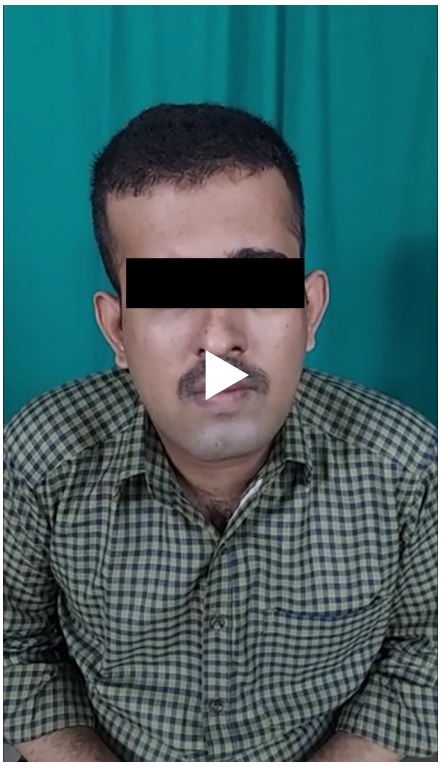


Figure 2: Operative images; (a) 3D illustration of left occipital burrhole. (b) Endoscopic view of cyst. (c) Initial fenestration. (d) Expansion of fenestration. (e) Post fenestration inspection. (f) Confirming cerebrospinal fluid flow before Final scope withdrawal.

evidence of extrapyramidal dysfunction.[4] Russo and Kindt hypothesized that medial dorsal thalamic compression disrupts communication between the thalamus, basal ganglia, and cervical motor neurons, generating involuntary rhythmic discharges. However, this fails to explain why similar lesions do not always produce BHDS. Wiese *et al.*[10] proposed a dynamic CSF-flow hypothesis, suggesting that oscillatory head movements develop as an adaptive behavior to intermittently relieve obstructed CSF pathways. This is supported by the observation that movements worsen when upright and diminish during sleep, when hydrostatic pressure is altered.[3,6] The ability to voluntarily suppress movements briefly also suggests basal ganglia involvement. Thus, a combination of neural compression, CSF flow disturbance, and compensatory mechanisms likely underlies BHDS.

In most previously reported cases, the cystic lesion has been suprasellar, exerting mass effect on the third ventricle and adjacent thalamic and midbrain structures. However, the present case is unique in that the causative lesion was a posterior fossa arachnoid cyst. This anatomical distinction offers a novel opportunity to extend the understanding of BHDS pathophysiology beyond the classical suprasellar model. Compression of posterior fossa structures – particularly the cerebellar dentate nucleus, inferior olivary complex, and cerebellar peduncles – could disrupt the dentato-rubro-thalamic circuitry responsible for coordination

Video 1: Abnormal movement before surgery, Video is accessible from the portal.



Video 2: Complete resolution of abnormal movement after surgery, Video is accessible from the portal.

and rhythmic motor control. Such interference within the cerebellar-thalamic loop may generate oscillatory discharges analogous to those caused by suprasellar compression of midbrain or thalamic nuclei. This suggests that BHDS may not be site-specific but rather circuit-specific, arising whenever critical nodes of the cerebello-thalamo-cortical pathway are distorted, irrespective of cyst location.

MRI typically shows a suprasellar cyst compressing the third ventricle, with associated ventricular dilatation and hydrocephalus. Cine or phase-contrast MRI can demonstrate intermittent CSF flow obstruction at the foramen of Monro or aqueduct of Sylvius, correlating with symptom fluctuations.[7] In posterior fossa variants, as in the present case, cine MRI may reveal obstruction at the aqueduct of Sylvius or fourth ventricular outlets, reinforcing the role of dynamic CSF flow disturbance in symptom generation.

Surgical management is the mainstay of therapy, aiming to restore CSF circulation and relieve mass effect. Endoscopic cyst fenestration has emerged as the preferred approach, offering definitive decompression and high rates of symptomatic resolution without lifelong shunt dependence. Reports indicate rapid postoperative improvement, often with near-complete disappearance of head oscillations.[2] In contrast, VP shunting addresses hydrocephalus but not the underlying cyst, frequently resulting in persistent or recurrent symptoms.

Given these findings and the excellent postoperative outcome observed in this case, endoscopic fenestration should be regarded as the treatment of choice for cystic-obstructive forms of BHDS, whether suprasellar or posterior fossa in origin. Early intervention not only relieves CSF obstruction but also reverses secondary neural compression, leading to complete functional recovery. Advances in MRI and minimally invasive endoscopic techniques have markedly improved diagnostic accuracy and surgical outcomes, reinforcing the role of timely intervention.

CONCLUSION

BHDS is a rare movement disorder, usually caused by suprasellar arachnoid cysts compressing midbrain structures, though rarely linked to posterior fossa cysts. It presents with rhythmic head oscillations, worsened by excitement and reduced during sleep. While often seen in childhood, this case highlights an uncommon adult presentation. MRI with cine flow studies aids diagnosis. VP shunting, though historically used, frequently results in persistent or recurrent symptoms since it addresses hydrocephalus but not the primary cystic obstruction. In contrast, endoscopic fenestration – alone or combined with endoscopic third ventriculostomy – directly restores normal CSF circulation, decompresses the cyst, and achieves durable neurological recovery. Based on our

excellent postoperative outcome and consistent literature evidence,[4-6,9,10] endoscopic fenestration should be advocated as the first-line treatment for cystic-obstructive etiologies of BHDS, offering superior long-term efficacy and shunt independence. Early recognition and surgical intervention are crucial to prevent progressive neurological decline and ensure favorable outcomes.

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