J Hosp Palliat Care 2024 December;27(4):177-179
https://doi.org/10.14475/jhpc.2024.27.4.177

Case Report

pISSN 2765-3072 • eISSN 2765-3080

The Use of Baclofen to Control Vertical Nystagmus in a Patient with Advanced Glioma: A Case Report

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Nystagmus, characterized by involuntary eye movements, can arise from several causes, with benign paroxysmal positional vertigo being the most prevalent. Additionally, central lesions such as tumors may also induce nystagmus. This case report describes the amelioration of vertical nystagmus in a patient with advanced glioma after treatment with the GABAergic drug baclofen.

Key Words: Pathologic nystagmus, Glioma, Palliative care, Baclofen

Received October 8, 2024
Revised November 4, 2024
Accepted November 18, 2024

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INTRODUCTION

Nystagmus and vertigo are common symptoms encountered in primary healthcare settings, frequently attributed to benign positional vertigo [1]. However, central causes of nystagmus account for about 11% of cases and may arise from various central nervous system (CNS) conditions. Tumoral involvement of the brainstem, although rare, represents a significant cause that has not been widely reported in the literature [2-5].

This report presents a rare and intriguing case of vertical nystagmus that showed significant improvement after baclofen treatment in a patient with advanced glioma. The uncommon nature of this case highlights the unique challenges and opportunities in our field, underscoring the critical importance of our work in addressing these challenges.

CASE PRESENTATION

A 23-year-old man was diagnosed with a brainstem glioma, as confirmed by radiological imaging. Due to the risks associated with a biopsy in such a location, surgery was not pursued. Instead, he underwent concurrent chemoradiation, including focal radiation to the brain, followed by treatment with temozolomide. As the disease progressed, he experienced increased intracranial pressure, which required intubation and the placement of a ventriculoperitoneal shunt. After surgery, his condition showed significant improvement; in particular, his level of consciousness improved enough for him to be discharged home with a normal mental status, although his palliative performance scale was at 30%. Subsequently, he was admitted to the palliative care unit due to a urinary tract infection. During this hospital stay, he developed persistent vertical nystagmus and vertigo, which worsened with changes in head position. This journey, marked by both hope and setbacks, underscores the resilience of our patients and highlights the ongoing challenges in our field.

Physical examination revealed involvement of the third, sixth, and seventh cranial nerves, accompanied by vertical nystagmus. His blood count and renal profile were normal, with the following values: hemoglobin: 14 g/dL, platelet count: 311×

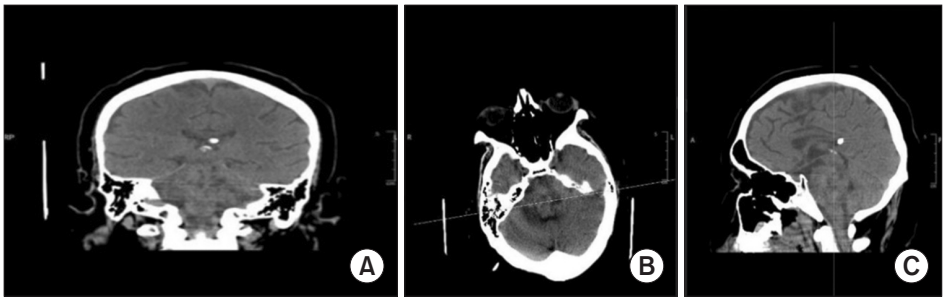

Figure 1. CT scan obtained in a 23—year-old man with pontine mass. (A) Cornoal view. (B) Axial view. (C) Sagittal view.
Technique: Pre-and post-contrast enhancement CT scan of the brain with sagittal and coronal reformats.
Findings: Known case of brainstem lesion. Post right parietal approach VP shunt with tip is seen in place.
Redemonstration of the heterogeneously enhancing ill-defined expansile pontine mass which is measuring approximately 4.2×4.2×3.7 cm it is surrounded by mild peritumoral edema, mass-effect on the cerebellum, fourth ventricle resulting in crowding of the foramen magnum.
No other focal masses or abnormal enhancement could be seen.
No evidence of intra or extra axial hemorrhage. No well-established acute territorial infarction. No midline shift.

$10^3/\mu L$, white blood cell count: $9\times10^3/\mu L$, creatinine: 29 μmol/L, corrected calcium: 2.3 mmol/L, sodium: 139 mmol/L, potassium: 3.5 mmol/L.

A brain computed tomography scan revealed a pontine mass accompanied by edema, affecting the fourth ventricle and cerebellum Figure 1. Consultations with neurology and ophthalmology determined that CNS involvement was responsible for the symptoms. Treatment began with betahistine, which proved ineffective. Since the patient was already taking dexamethasone 8 mg when the symptoms appeared, increasing the steroid dosage was not considered advisable. Consequently, we initiated treatment with baclofen, administering 5 mg orally three times daily. Two days after starting baclofen, there was a significant improvement in the patient's nystagmus and vertigo, which persisted at follow-up.

DISCUSSION

Our patient experienced persistent, debilitating nystagmus, which was accompanied by vertigo and nausea, significantly diminishing his quality of life. Given the severity of his condition and his functional limitations, our goal was to alleviate these symptoms with minimal intervention. Despite the tumor being inoperable and the patient having received the maximum feasible chemotherapy and radiation treatment, there was only a slight improvement in his nystagmus. However, the introduction of baclofen at a dosage of 5 mg orally three times daily resulted in a significant reduction in his nystagmus. This improvement offers hope for similar cases in the future and underscores the potential of our efforts to bring about positive change.

There is limited data on the symptomatic treatment of vertigo and nystagmus caused by refractory CNS tumors. However, a review of the literature on acquired nystagmus treatment suggests that certain drugs may reduce nystagmus speed and enhance visual acuity. In a double-blind controlled study, gabapentin significantly decreased the speed of acquired pendular nystagmus in multiple planes, whereas baclofen was effective only in the vertical plane [6]. Another study involving 4 aminopyridine, a channel blocker that boosts Purkinje cell activity in the cerebellum, demonstrated improvements in downbeat nystagmus. This effect was particularly pronounced in patients with cerebellar atrophy, as opposed to other central causes [7].

Baclofen, a derivative of gamma-aminobutyric acid and a specific agonist of B receptors, is commonly used to treat disorders such as spasticity and dystonia. It is thought to stabilize the vestibular system by influencing the velocity mediated by the vestibular-ocular reflex. Baclofen enhances the inhibitory influence of the vestibule-cerebellum on the vestibular nuclei. This action affects the velocity storage mechanism, which typically facilitates the adaptation of the vestibular-ocular reflex and tends to be exaggerated in central lesions associated with the cerebellum. Through its inhibitory effect, baclofen may help alleviate symptoms of nystagmus in some patients [8-10].

A recent study evaluated the impact of baclofen on the intensity of paroxysmal positional downbeat nystagmus. The

https://doi.org/10.14475/jhpc.2024.27.4.177

findings indicated a reduction in the slow phase velocity of this nystagmus type following baclofen administration, potentially due to its inhibitory effects on the vestibular nuclei. However, the primary causes of central nystagmus in that study were spinocerebellar ataxia and multiple systemic atrophy, rather than tumors, as observed in our case [9]. In a small trial involving patients with various etiologies of nystagmus, oral baclofen (5 mg three times daily) reduced the slow phase velocity of nystagmus by 25 to 75% [10]. Our case report contributes valuable insights by demonstrating the effectiveness of baclofen in a scenario that has rarely been addressed previously, involving a CNS tumor. Our findings suggest a potential benefit of baclofen for patients with nystagmus associated with CNS tumors. However, more rigorous investigations, such as case series, studies exploring underlying mechanisms, or well-designed intervention studies, are necessary to confirm these benefits in the future.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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AUTHOR'S CONTRIBUTIONS

Conception or design of the work: all authors. Data collection: all authors. Data analysis and interpretation: all authors. Drafting the article: all authors. Critical revision of the article: all authors. Final approval of the version to be published: all authors.

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