Cureus
Part of SPRINGER NATURE

Open Access Case Report

Vestibular Epilepsy Presenting With Isolated Recurrent Vertigo and Postictal Headache: A Case Report

Cempaka Thursina Srie Setyaningrum [1], Sri Sutarni [1], Desin Pambudi Sejahtera [1], Natasha Yang [1]

1. Department of Neurology, Faculty of Medicine, Public Health, and Nursing, Universitas Gadjah Mada, Sardjito Hospital, Yogyakarta, IDN

Corresponding author: Natasha Yang, natasha.yang@mail.ugm.ac.id

Review began 10/08/2026
Review ended 10/15/2026
Published 10/20/2026

© Copyright 2026
Srie Setyaningrum et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.118213

Abstract

Vestibular epilepsy, also referred to as epileptic vertigo syndrome, is a rare and underrecognized cause of recurrent episodic vertigo. Currently, there are no universally accepted diagnostic criteria for vestibular epilepsy. A 38-year-old woman presented with an eight-month history of recurrent spontaneous episodes of rotational vertigo lasting one to two minutes and occurring multiple times daily. Episodes were accompanied by nausea and vomiting and were followed by bilateral throbbing headaches. Neurological examination, bedside vestibular assessment, and cognitive screening were unremarkable. Brain magnetic resonance imaging demonstrated no structural epileptogenic lesion, while brainstem evoked response audiometry was normal. Electroencephalography revealed frequent generalized epileptiform discharges with a photoparoxysmal response during intermittent photic stimulation. The patient was diagnosed with vestibular epilepsy with postictal headache after exclusion of alternative causes of episodic vertigo. Treatment with extended-release divalproex sodium resulted in a marked reduction in attack frequency and improvement in symptoms during follow-up. Vestibular epilepsy represents an uncommon seizure manifestation in which vestibular symptoms predominate and may occur in isolation. In this patient, the brief stereotyped attacks, normal brain neuroimaging, absence of peripheral vestibular abnormalities, epileptiform electroencephalographic findings, and favorable response to antiseizure therapy supported the diagnosis. In cases of recurrent episodic vertigo presenting concurrently with headache, vestibular epilepsy remains a diagnostic challenge, mainly because overt neurological manifestations are often absent. Recent literature suggests that electroencephalography should be considered in patients with recurrent unexplained brief vertigo, particularly when episodes are stereotyped or resistant to conventional vestibular therapies.

Categories: Neurology
Keywords: epileptic vertigo, episodic vestibular syndrome, recurrent vertigo, vertigo management, vestibular epilepsy

Introduction

The International Classification of Vestibular Disorders (ICVD) recognizes three major vestibular syndromes, distinguished according to symptom duration. Acute vestibular syndrome (AVS) is characterized by a sudden onset with a single disease episode lasting from several days to weeks. Episodic vestibular syndrome (EVS) is defined by recurrent attacks lasting from a brief period up to several days. In contrast, chronic vestibular syndrome (CVS) refers to persistent vestibular symptoms lasting for at least three months [1]. EVS is a vestibular disorder characterized by transient episodes that typically last from seconds to hours and only rarely persist for several days. These episodes are usually accompanied by brief nausea, the appearance of nystagmus, and sudden falls [2]. Symptoms may occur as recurrent attacks caused by an inherently episodic disorder or as the initial manifestation of a chronic progressive disease presenting with transient or recurrent episodes. EVS is classified into triggered EVS (t-EVS), in which attacks are provoked by specific triggers, and spontaneous EVS (s-EVS), in which episodes occur without identifiable triggers. The diagnosis of s-EVS relies primarily on a thorough clinical history [3]. EVS may result from disorders of either the peripheral or central vestibular system.

The most common peripheral cause is Benign Paroxysmal Positional Vertigo (BPPV), which is characterized by brief episodes of vertigo triggered by changes in head position. Another peripheral cause is Ménière's disease, which presents with longer episodes of vertigo lasting 20 minutes to several hours and is accompanied by auditory symptoms such as tinnitus and fluctuating hearing loss. Among central causes, vestibular migraine is a significant etiology, presenting with recurrent vertigo that may occur with or without migraine headaches and is often associated with photophobia and phonophobia. In addition, transient ischemic attack (TIA) involving the posterior circulation should be considered, particularly in individuals with vascular risk factors, as it may represent an early warning sign of vertebrobasilar stroke [4]. Vestibular paroxysmia is a rare cause of EVS, typically producing attacks that last only a few seconds to less than one minute and are generally associated with vascular compression of the vestibulocochlear nerve. It is estimated to account for approximately 3-4% of patients presenting with vestibular symptoms. Vestibular epilepsy is another rare and frequently underdiagnosed condition, with an estimated incidence of approximately 1 per 100,000 population. Nevertheless, it is thought to account for 6-

15% of vertigo cases within the relevant patient population [5-7]. Here, we describe a patient presenting with isolated recurrent vertigo followed by postictal headache whose diagnosis was supported through clinical and EEG examination after extensive exclusion of more common vestibular disorders.

Case Presentation

A 38-year-old woman presented to the clinic due to recurrent vertigo attacks. The symptoms began eight months prior, with episodes of spinning vertigo lasting 1-2 minutes and occurring more than five times daily, with no attacks occurring during sleep. Vertigo was associated with nausea and vomiting. The patient was initially hospitalized at the referring hospital but was discharged with persistent symptoms. After three months, the patient noticed that the attacks became predominantly triggered by fatigue or exposure to bright light. Each episode was subsequently followed by a bilateral throbbing headache lasting approximately three hours after resolution of the vertigo. The patient denied positional worsening of vertigo or exacerbation with Valsalva maneuvers, including straining, coughing, or sneezing. There were no symptoms between attacks, and no epigastric rising sensation, déjà vu, jamais vu, olfactory or gustatory hallucinations, or sudden fear preceding the episodes of vertigo. The patient also denied periods of loss of consciousness, limb weakness, numbness or paresthesia, gait imbalance, seizures, memory impairment, bowel or bladder dysfunction, visual disturbances, hearing loss, or tinnitus. There were no identified epilepsy risk factors, including a history of brain insult or a family history of epilepsy. Due to the lack of clinical improvement, the patient was referred to our center.

The patient was hemodynamically stable, with normal vital signs and general physical examination findings. Neurological examination revealed an alert and fully oriented patient (GCS E4V5M6). Cranial nerve examination was normal, with equal and reactive pupils, intact corneal reflexes, and no spontaneous nystagmus. Motor strength was normal in all extremities, with normal muscle stretch reflexes and absent pathological reflexes. Sensory, cerebellar, and meningeal examinations were unremarkable. Coordination testing, including finger-to-nose, finger-to-finger, heel-to-shin, and rapid alternating movements, was normal without dysmetria or dysdiadochokinesia. Romberg, tandem gait, and bucket tests were normal. The HINTS examination showed a negative head impulse test, absence of nystagmus, and a negative test of skew, consistent with a normal bedside vestibular examination. Cognitive screening was normal, with a Montreal Cognitive Assessment Indonesian version (MoCA-Ina) score of 28/30 and preserved functional status (Barthel Index 100/100).

Contrast-enhanced brain MRI, including high-resolution imaging of the medial temporal structures, revealed no structural abnormalities, as shown in Figure *1*. Bilateral hippocampi, cerebellopontine angles, cranial nerve VII/VIII complexes, and inner ear structures were unremarkable, with no evidence of mesial temporal sclerosis, vestibular schwannoma, or abnormal contrast enhancement (Figure *2*). Brainstem evoked response audiometry (BERA) was within normal limits. Interictal electroencephalography (EEG) demonstrated a normal symmetric background rhythm without focal slowing (Figure *3*) but revealed frequent generalized broad sharp-wave epileptiform discharges (Figure *4*). Intermittent photic stimulation (5-30 Hz) elicited a spinning sensation concurrent with the emergence of a generalized photoparoxysmal response (PPR), as shown in Figure *5*. The patient was diagnosed with vestibular epilepsy with postictal headache. The patient responded well to routine antiseizure medication using extended-release divalproex sodium 250 mg twice daily, with a marked reduction in attack frequency to once or twice per month after two months and improvement of symptoms during follow-up.

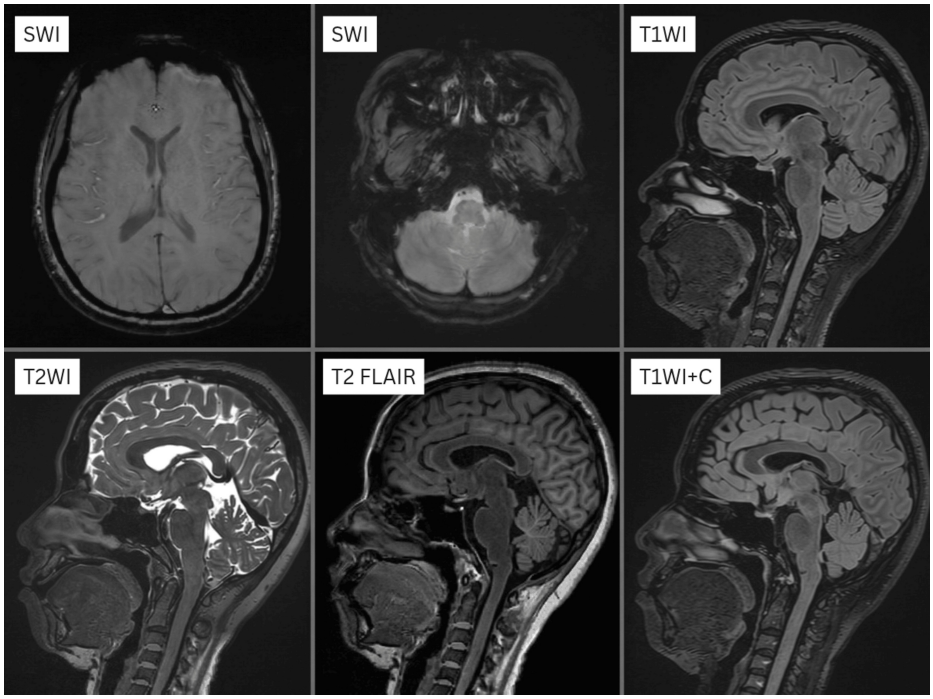


FIGURE 1: MRI brain with gadolinium contrast

Magnetic resonance imaging (MRI) of the brain with gadolinium contrast demonstrated normal brain parenchymal signal intensity without focal intra-axial or extra-axial lesions. No evidence of acute infarction, intracranial hemorrhage, mass effect, or hydrocephalus was identified. The ventricles, cortical sulci, and basal cisterns were normal in size and configuration.

T1WI: T1-weighted imaging; T1WI+C: contrast-enhanced T1-weighted imaging; T2WI: T2-weighted imaging; T2 FLAIR: T2-weighted fluid-attenuated inversion recovery; SWI: susceptibility-weighted imaging.

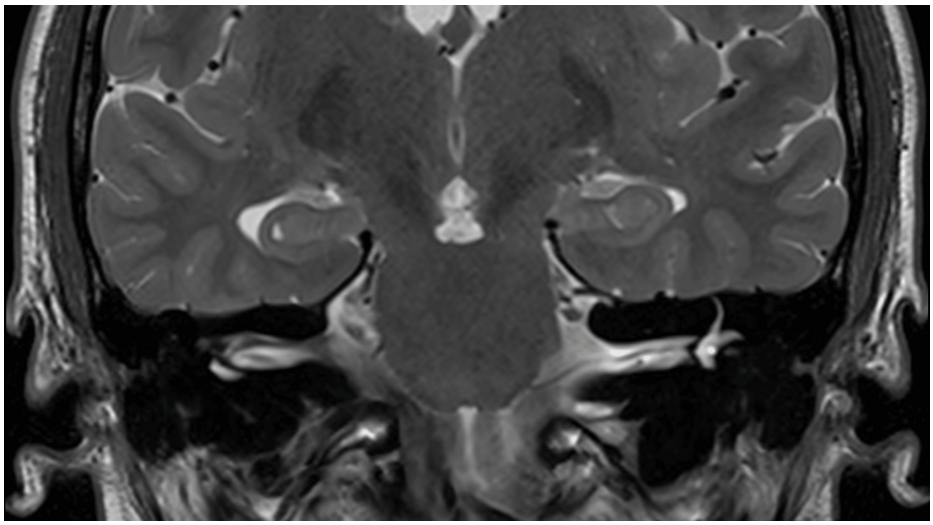


FIGURE 2: High-resolution coronal T2-weighted image through the medial temporal lobe

Coronal high-resolution T2-weighted imaging through the medial temporal lobes demonstrated symmetric hippocampi with preserved normal morphology, size, and internal architecture bilaterally. No hippocampal atrophy, sclerosis, abnormal T2/FLAIR signal intensity, or contrast enhancement was identified. The amygdalae and parahippocampal gyri were unremarkable.

FLAIR: fluid-attenuated inversion recovery.

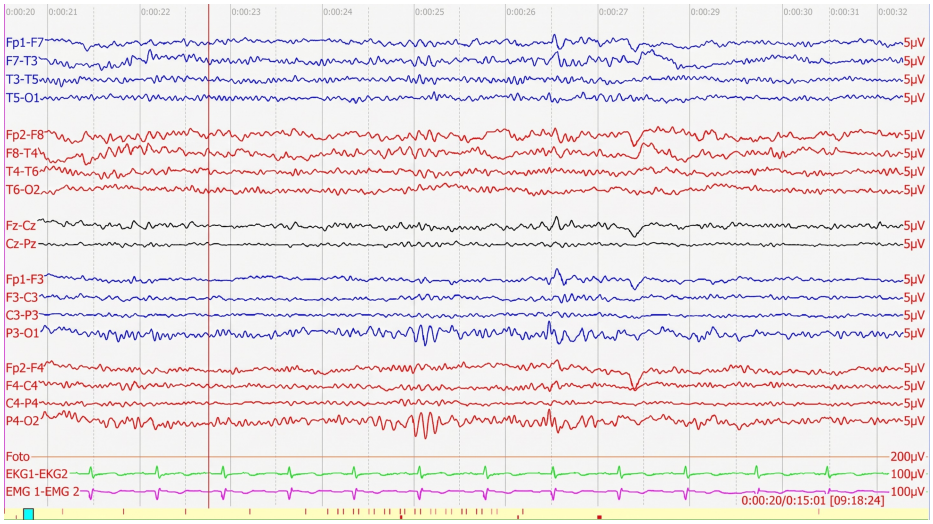


FIGURE 3: Interictal electroencephalography

Interictal EEG during wakefulness demonstrated a symmetric, regular low-amplitude alpha background without focal slowing or epileptiform discharges; the study was considered normal.

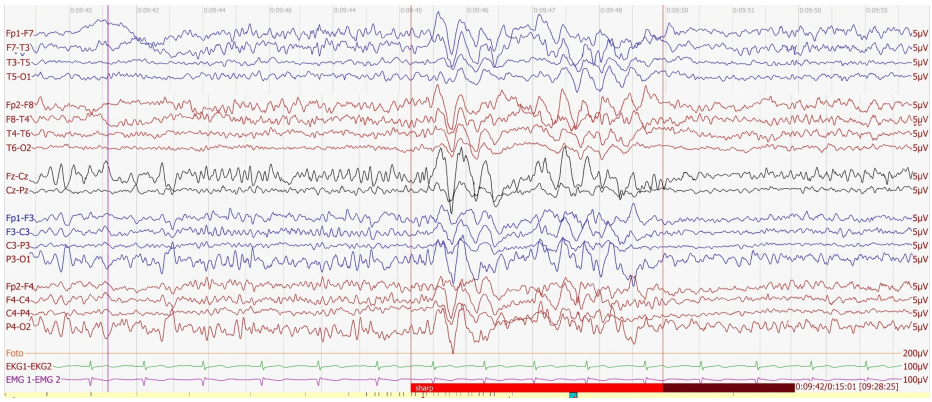


FIGURE 4: Epileptiform activity

Abnormal findings of frequent generalized broad sharp-wave epileptiform discharges, indicating cortical hyperexcitability and supporting a diagnosis of generalized epileptiform activity. No focal abnormalities or ictal events were identified during the recording.

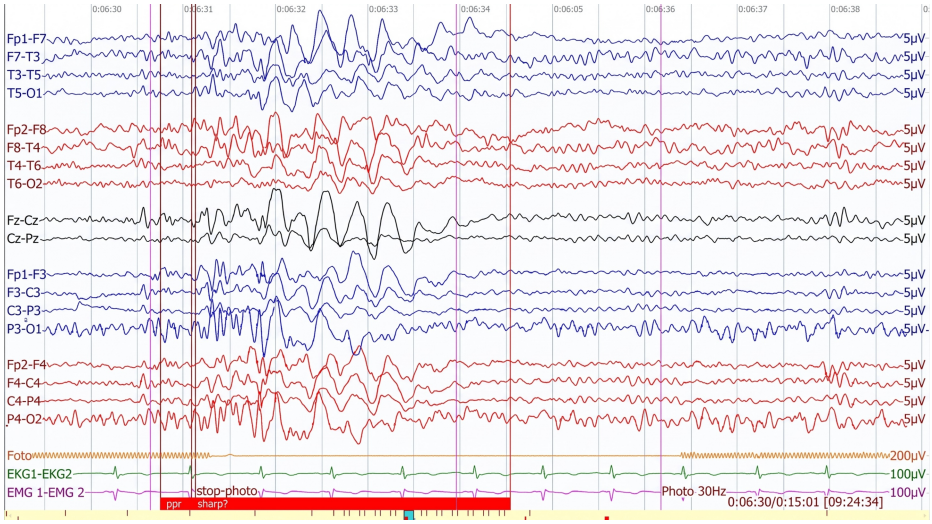


FIGURE 5: Electroencephalography provocation

Abnormal finding of EEG provocation include a photoparoxysmal response during intermittent photic stimulation, consistent with increased photosensitivity.

Discussion

This case is important because the diagnosis of vestibular epilepsy is often substantially delayed, with reported intervals averaging approximately three years and extending up to a decade, largely because its vestibular manifestations resemble those of more established vestibular disorders rather than typical epileptic seizures [8]. Clinically, the patient fulfilled the diagnostic criteria for probable vestibular paroxysmia. However, in this patient, given the very brief and recurrent nature of the attacks, as well as the lack of improvement with standard antivertigo therapy despite multiple outpatient and inpatient treatments, an EEG was performed to evaluate for epileptiform abnormalities suggestive of an ictal event. To date, no standardized diagnostic criteria have been established for seizure disorders presenting predominantly with vestibular symptoms, a condition commonly referred to as vestibular epilepsy or epileptic vertigo. Periguey et al. proposed diagnostic criteria for vestibular epilepsy consisting of at least two recurrent seizure episodes characterized by stereotyped paroxysmal vestibular symptoms, relatively brief seizure duration ranging from approximately one minute to less than one hour, the absence of structural abnormalities on brain imaging, EEG abnormalities recorded during wakefulness or sleep demonstrating epileptiform activity involving the temporal and/or parietal lobes or the temporoparieto-occipital junction, and clinical improvement or a favorable response to antiseizure medication [9].

In addition to vestibular manifestations, ictal symptoms may include visual, auditory, and autonomic disturbances, while impairment of consciousness occurs in a subset of patients [5,10]. Some patients also experience interictal symptoms, such as persistent imbalance, which may reflect postictal vestibular dysfunction [9]. A weighted mean prevalence of postictal headache of 33% (95% CI, 26-40%) has been reported, making it one of the most common postictal symptoms [11]. Postictal vasodilation has been suggested as a contributing mechanism, but its pathophysiological basis has yet to be fully elucidated [12]. Postictal headache occurrence in association with vestibular seizures has not been systematically investigated. The patient's clinical presentation and ancillary investigations support the possibility of vestibular epilepsy, with epileptiform discharges on EEG and a normal brain MRI, consistent with the proposed diagnostic criteria. Tarnutzer et al. reported that EEG-localized epileptic vertigo most commonly originated from the temporal lobe (79.8%). However, it remains uncertain whether this distribution reflects a true anatomical predilection or the higher underlying prevalence of temporal lobe epilepsy [13]. EEG remains the primary diagnostic modality, although interictal epileptiform abnormalities may only be detected during sleep recordings, as approximately 25% of patients demonstrate abnormalities exclusively during sleep. To date, no treatment guidelines have been established for vestibular epilepsy. Other than carbamazepine, several antiseizure medications, especially non-enzyme-inducing antiseizure medications, have been reported to be effective, including levetiracetam and lacosamide [10].

Conclusions

Vestibular epilepsy should be considered in patients presenting with recurrent, brief, stereotyped episodes of spontaneous vertigo, especially when routine vestibular testing and brain imaging fail to reveal another cause. In this case, the diagnosis of vestibular epilepsy was supported by isolated recurrent vertigo with a very brief onset, postictal headache, epileptiform abnormalities on electroencephalography, a photoparoxysmal response, normal brain magnetic resonance imaging, and a favorable response to

antiseizure therapy. The presence of headache following the vestibular episodes may provide an additional clinical clue, although it should not be considered diagnostic in isolation. Recognition of this uncommon presentation is important because vestibular epilepsy may closely mimic other episodic vestibular disorders and consequently lead to diagnostic delay. This case highlights that vestibular epilepsy, although rare and diagnostically challenging, is a potentially treatable cause of recurrent episodic vertigo.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Natasha Yang, Cempaka Thursina Srie Setyaningrum, Desin Pambudi Sejahtera, Sri Sutarni

Acquisition, analysis, or interpretation of data: Natasha Yang, Desin Pambudi Sejahtera

Drafting of the manuscript: Natasha Yang, Cempaka Thursina Srie Setyaningrum

Critical review of the manuscript for important intellectual content: Natasha Yang, Cempaka Thursina Srie Setyaningrum, Desin Pambudi Sejahtera, Sri Sutarni

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Kaski D, Tarnutzer AA, Agrawal Y, et al.: The International Classification of Vestibular Disorders: achievements, challenges, and future directions. J Vestib Res. 2025, 35:105-12. 10.1177/09574271251313803
2. Bisdorff AR, Staab JP, Newman-Toker DE: Overview of the International Classification of Vestibular Disorders. Neurol Clin. 2015, 33:541-50, vii. 10.1016/j.ncl.2015.04.010
3. Comolli L, Korda A, Zamaro E, et al.: Vestibular syndromes, diagnosis and diagnostic errors in patients with dizziness presenting to the emergency department: a cross-sectional study. BMJ Open. 2023, 13:e064057. 10.1136/bmjopen-2022-064057
4. Tarnutzer AA, Kerkeni H, Diener S, et al.: Diagnosis and treatment of vertigo and dizziness: interdisciplinary guidance paper for clinical practice. HNO. 2025, 73:357-69. 10.1007/s00106-025-01599-z
5. Philip A, Haripriya G, Mammen M, Lepcha A, Augustine A: Vestibular epilepsy: clinical presentation, diagnosis, and management. Indian J Otol. 2020, 26:147. 10.4103/indianjotol.INDIANJOTOL_154_20
6. Wood AM, Thompson-Harvey A, Kesser BW: Vertiginous epilepsy in the pediatric population. Front Neurol. 2024, 15:1403536. 10.3389/fneur.2024.1403536
7. Dieterich M, Brandt T: Vestibular paroxysmia: a systematic review . J Neurol. 2025, 272:188. 10.1007/s00415-025-12913-8
8. Widdess-Walsh P: A case of the "Spinnies": vestibular epilepsy . Epilepsy Curr. 2025, 25:315-6. 10.1177/15357597251352971
9. Perriguey M, Elziere M, Lopez C, Bartolomei F: Vestibular epilepsy: clinical and electroencephalographic characteristics with the proposed diagnostic criteria. J Neurol. 2024, 272:68. 10.1007/s00415-024-12796-1
10. Surmeli R, Yalcin AD, Surmeli M, Gunay G: Vertiginous epilepsy: documentation of clinical and electrophysiological findings of nine patients. Epileptic Disord. 2020, 22:775-81. 10.1684/epd.2020.1232
11. Förderreuther S, Henkel A, Noachtar S, Straube A: Headache associated with epileptic seizures: epidemiology and clinical characteristics. Headache. 2002, 42:649-55. 10.1046/j.1526-4610.2002.02154.x
12. Zhang S, Lv Z, Li J, Zhou D: Clinical characteristics of headache related to epilepsy: experience from a tertiary epilepsy center. BMC Neurol. 2025, 25:223. 10.1186/s12883-025-04217-1
13. Tarnutzer AA, Lee SH, Robinson KA, Kaplan PW, Newman-Toker DE: Clinical and electrographic findings in epileptic vertigo and dizziness: a systematic review. Neurology. 2015, 84:1595-604. 10.1212/WNL.0000000000001474