

CASE REPORT

Vertigo or dizziness as the first presentation of epilepsy: A case report

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Abstract

Vertigo or dizziness is a common neurological symptom, but it is rarely regarded as the first presenting symptom of epilepsy. Patients who present with vertigo as the first symptom of epilepsy may be misdiagnosed or missed diagnose. We report a case presenting with vertigo or dizziness as the first manifestation of epilepsy and review relevant literature. A 71-year-old female patient sought medical attention in view of paroxysmal dizziness for a year that progressively worsened over the course of 1 week. Initially, her dizziness occurred 2 or 3 times a month, lasting for less than a minute each time. However, her attacks became more frequent and severe over the course of 1 week. Twenty-four-hour electroencephalogram (EEG) monitoring showed spike-and-slow-wave complex discharges at both sphenoidal electrodes, with the left being more prominent. The patient was treated with oral valproic acid. After a 9-month follow-up, the frequency of dizziness was markedly reduced. Although the pathogenesis of epileptic vertigo remains ambiguous, the current literature indicates that vestibular cortical neurons may be closely associated with periodic abnormal discharge of the brain lobe. Based on comprehensive medical history and EEG results, we should exercise caution when diagnosing this kind of epilepsy.

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Keywords: Epilepsy; Vertigo; Dizziness; Electroencephalogram**1. Background**

Vertigo or dizziness is a common neurological symptom that is related to peripheral and/or central nervous system disorders. The vestibular system extends from the peripheral vestibular part of the inner ear to the brainstem and cerebral cortex. Any lesion throughout the entire tract could lead to dizziness or vertigo. A growing number of literature has suggested that vertigo or dizziness is deemed as one of the clinical manifestations of epileptic seizures^[1]. Especially the focal and intermittent epileptic discharges, which are accompanied by episodic vestibular symptoms^[2], have been referred to as vertiginous seizures, epileptic vertigo or dizziness (EVD), vestibular epilepsy or seizures, and so on^[3-6]. Herein, we report a case presenting with vertigo or dizziness as the first manifestation of epilepsy and review relevant literature.

2. Case presentation

A 71-year-old female patient presented to our hospital in August 2016 in view of paroxysmal dizziness for a year that progressively worsened over a week, accompanied

by heavy-headedness, floating sensation, and instability when walking. Originally, her dizziness would occur 2 or 3 times a month, with each episode lasting for less than a minute. However, over a course of 1 week, she experienced more frequent (≥ 5 times a day) and severe episodes of vertigo. Her symptoms were unpredictable and unrelated to position change. She did not experience any intense spinning sensation during the onset of vertigo, nor was the dizziness accompanied by hallucination, unconsciousness, headache, orbital discomfort, sweating, nausea, vomiting, abdominal pain, or diarrhea. She had no fluttered feelings, chest tightness, palpitations, pruritus over her abdomen, or abdominal discomfort, including "ascending epigastric sensation." There were no concurrent neurological, ophthalmic, or auditory disorders. She did not experience agoraphobia, panic attacks, or any recent stressful situations. She had a previous history of lumbar disk herniation more than 4 years ago, and she denied history of hypertension, coronary heart disease, and diabetes. She had no history of motion sickness, migraine, trauma, surgery, or diseases that resulted in epileptic seizures. Personal history and family history were insignificant. Physical examination showed that her general condition was good. Cardiovascular, respiratory, abdominal, and nervous system examinations were normal. No abnormalities observed in vestibular and cerebellar function examinations. Her temperature and hearing examination were normal. The laboratory investigation results were as follows: C-reactive protein (CRP), liver function, kidney function, blood lipid, blood glucose, electrolytes, homocysteine, thyroid function, erythrocyte sedimentation rate (ESR), coagulation function, protein S, protein C, antinuclear antibodies (ANAs), antiphospholipid antibodies, and antiganglioside antibodies were all within the normal range. Serologic tests for human immunodeficiency virus (HIV), hepatitis B/C, and cytomegalovirus were all negative. There were no obvious abnormalities in chest radiograph, echocardiogram, abdominal ultrasound, and electrocardiogram examinations. Brain magnetic resonance imaging (MRI) showed old lacunar cerebral infarction lesions. Evaluation of intracranial and extracranial large vessels such as carotid ultrasound, computerized tomography angiography (CTA), and magnetic resonance angiography (MRA) did not show obvious stenosis nor atherosclerotic plaque. Hyperventilation did not trigger the onset of the dizziness, and no spontaneous nystagmus was observed during the episode. On otolaryngology examination, her ear canals and eardrums were normal. In addition, tests for benign paroxysmal positional vertigo (BPPV) such as bilateral Dix-Hallpike maneuver, supine roll test, Unterberger test, bilateral Dix-Hallpike maneuver, supine roll test, Unterberger test, fistula test, video head

impulse test, head thrust test, and caloric reflex test were all negative. Twenty-four-hour electroencephalogram (EEG) monitoring showed spike and slow wave complex discharges occurring at both sphenoidal electrodes, with the left being more prominent (Figure 1). Oral sodium valproate (500 mg/day) was administered and the dosage was increased to 1000 mg/day after a week. A 9-month follow-up showed that the patient had no further episodes of paroxysmal dizziness.

3. Discussion

Epileptic vertigo is a rare form of epilepsy. The incidence of epilepsy presenting with vertigo or dizziness is only about 8.5%. Especially, the sole symptom of vertigo or dizziness, which is rarely reported in the literature, only occurs in approximately 0.8% of all epilepsy^[3]. Its incidence is higher in children and adolescents compared with adults, especially in female^[7]. Due to the differences in race, sample size, etiology, and evaluation criteria between different studies^[3], a complete and consistent criterion for epileptic vertigo has yet to be established.

Although the pathogenesis of epileptic vertigo remains elusive, the current literature has indicated that vestibular cortical neurons are associated with periodic abnormal discharge of the brain lobe^[8,9]. Vestibular symptoms may also occur as a result of the permanent damage of the vestibular cortical area due to chronic epilepsy^[10]. The hallmarks of epilepsy, including paroxysmal, transient, stereotypical, and repetitive, are also present in epileptic vertigo. Its main clinical manifestations are recurrent attacks of dizziness and vertigo, accompanied by the rotation of the visual scene, with or without nausea, vomiting, pallor, sweating, palpitation, headache, and so on. These symptoms are unpredictable and are not associated with positional variation. With each episode, there is no loss of consciousness. Some patients may present with peripheral illusion sensation, such as rotating, drifting, tilting, swaying, heavy headedness, and unsteadiness while walking.

Each attack varies from several seconds to minutes without an exact time. However, studies have reported that the duration of almost all temporal lobe-associated vertigos usually lasts less than a minute, whereas that of non-temporal lobe-associated vertigo lasts longer^[3]. This may indicate that the temporal lobe is associated with transient epileptic vertigo. Hence, missed diagnosis or misdiagnosis with transient ischemic attacks (TIA), vestibular migraine, Meniere's disease, BPPV, vestibular paroxysmia, hypoglycemia, arrhythmia, panic attacks, neurosis, somatization, or psychogenic dizziness may likely occur in such patients. In EEG, abnormalities are mainly observed in the frontal, temporal, and top area of

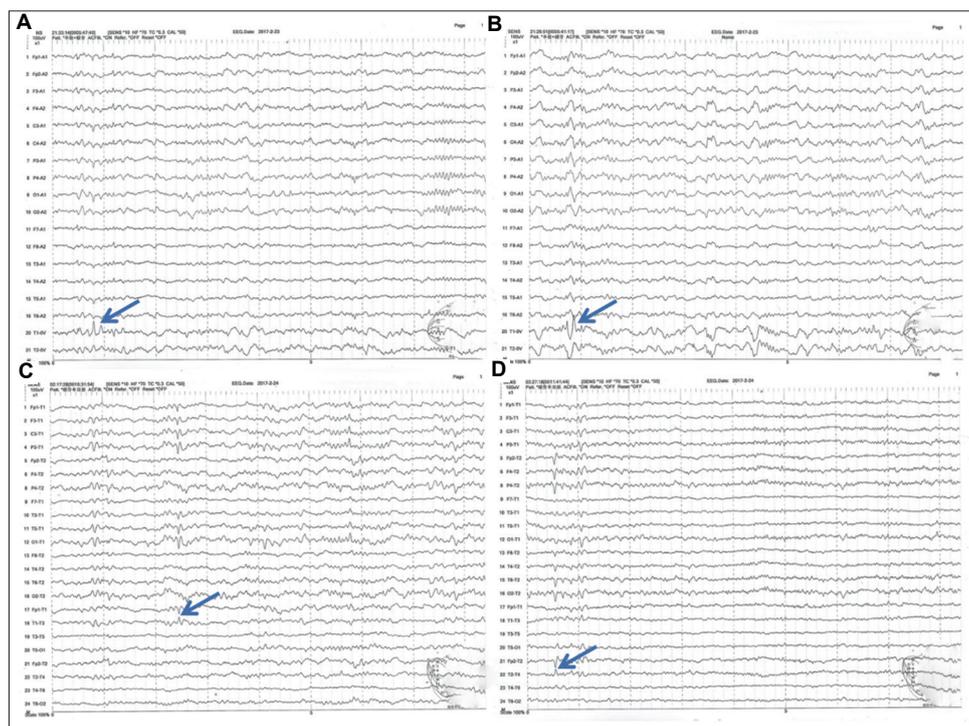


Figure 1. Electroencephalogram findings. (A–B) EEG shows paroxysmal spike and slow wave complex discharges in both temporal regions, with the left being more prominent. (C) EEG shows phase reversal in the T1 lead, located at the left temporal region. (D) EEG shows phase reversal in the T2 lead, located at the right temporal region. These epileptic waves are marked by the blue arrows.

the brain. These areas, especially the temporal lobe, release paroxysmal spikes or sharp waves, spike and slow wave complexes, or high amplitude slow waves^[1,11].

The diagnosis of epileptic vertigo still remains a clinical problem. The incomplete medical history and the absence of ancillary examinations increase the rate of missed diagnosis and misdiagnosis of this disease^[12]. The differential diagnosis for episodic vertigo/dizziness is broad and spans across various medical specialties. In fact, only a few patients would undergo thorough brain MRI, complete vestibular testing, ictal EEG, and professional molecular genetic testing^[13]. Besides, if antiepileptic drugs (AEDs) are effective in treating vertigo/dizziness, the diagnosis of epileptic vertigo may also be a false-positive diagnosis. For instance, the use of AEDs is a successful strategy for the treatment of vestibular paroxysmia, vestibular migraine, and migraine^[14,15]. Although the treatment response rate of AEDs in vertigo is as high as 90%, the diagnostic rate of epileptic vertigo is only 10%.

We should be alert in diagnosing cases of epilepsy presenting with vertigo as a standalone symptom since this condition is often underdiagnosed. This case demonstrates that to diagnose this condition, there is a need for a detailed history, a comprehensive physical examination, and a wide range of ancillary examinations and differential diagnoses, including those related to neurology, otolaryngology,

internal medicine, and psychiatry. Multidisciplinary collaboration has the potential to cover areas that are underemphasized in a single professional context.

4. Conclusion

This patient initially experienced simple vertigo, and her EEG revealed abnormal wave discharges at both sphenoidal electrodes, which were located at both temporal lobes. Her symptoms remarkably improved with regular antiepileptic therapy. This case may provide some reference for clinical diagnosis and treatment. However, there are some limitations observed in this case. Video EEG test was not done for the patient, neither was flash stimulation or sleep induction used during her EEG examination. Since this case report only covers one case, there may be certain contingencies; hence, more practical cases and theoretical studies are needed to make up for the deficiency. In the future, we must pay close attention to the history of such epileptic patients.

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Conflict of interest

The authors declare that they have no competing interests.

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Ethics approval and consent to participate

This study was approved by the Local Ethics Committee of the Affiliated Hospital of Weifang Medical University. The research ethics approval number was wyfy-2022-ky-179. The written informed consent for this study was obtained from the patient.

Consent for publication

The written informed consent and permission to publish the patient's data and/or images were obtained from the patient. We did our best to mask or conceal any identifying information of the patient that appears in writing or within photograph.

Availability of data

Not applicable.

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