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Short Communication

Is Vestibular Epilepsy an Underdiagnosed Form of Recurrent Vertigo?

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Vestibular epilepsy is often mentioned as a probable cause of episodic vertigo.

As a neurologist dedicated to neuro-otology for over 20 years, I have rarely seen it. Why?

We have to differentiate between Vestibular Epilepsy and Nystagmus (See table 1). In the first case, we have the symptoms reported by the patient. In the second, we can see a fixed-direction nystagmus, but its mechanism has nothing to do with vestibular nystagmus. Two hypotheses have been proposed to explain it: (1) primary, contraversive saccades due to epileptic activity in the saccade region, followed by centripetal drift due to impaired gaze holding function; and (2) secondary, reflexive contraversive saccades, which correlate for slow ipsiversive deviation across the midline, due to epileptic activation of cortical areas important for motion vision and smooth tracking (Lee and Zee, 2015).

Feature	Vestibular Epilepsy	Epileptic Nystagmus
Clinical Presentation	Vertigo, dizziness, sense of movement [1-4]	Rapid eye movements, visual blurring [5-7]
Onset	Paroxysmal, brief seizures [1-3]	Abrupt, coinciding with seizure activity [5-7]
Associated Symptoms	Autonomic symptoms, nausea, imbalance [2,3]	Consciousness often preserved, diplopia possible [6,7]
EEG Findings	Epileptiform discharges in temporo-parietal/insular regions [1-3]	Discharges in occipital/parietal regions [5-7]
Duration	Seconds to minutes [2-4]	Usually seconds, short-lived [5-7]

Table 1: *Comparison: Vestibular epilepsy vs. epileptic nystagmus.*

Consequently, nystagmus has the fast phase opposite the affected hemisphere.

This is a specific type of seizure caused by vestibular cortex discharges and should not be confused with other conditions: 1. The so-called "volvular epilepsy," where the seizure consists of the patient spinning in small circles without impairment of consciousness. The focal

location can be frontal or temporal (Donaldson, 1986); 2. epileptic nystagmus, in which the focal discharge results in nystagmus, and it can originate in the vestibular, visual (optokinetic), or oculomotor cortex (Furman., *et al*. 1990; Kaplan and Tusa, 1993); 3. "Vestibulogenic epilepsy," where the seizure is induced by vestibular stimulation, and is another form of reflex epilepsy (Behrman and Wyke, 1958; Barac, 1968).

The regions involved in both cases are known (See table 2). Given the incidence of epilepsy: 0.5% of the general population, and that 70% of these are focal, and taking into account the regions involved, we would have a statistically significant chance of 49% of vestibular presentations.

Condition	Brain Regions Involved	Notes
Vestibular Epilepsy	Temporo-parietal junction, superior temporal gyrus, parietal operculum, posterior insula	Associated with vertigo, spatial disorientation, sometimes auditory symptoms
Epileptic Nystagmus	Occipital cortex, parietal cortex, frontal eye fields, brainstem (vestibular nuclei)	Characterized by involuntary rhythmic eye movements; often contralateral to cortical focus

Table 2: *Brain regions involved in vestibular epilepsy and epileptic nystagmus.*

In fact, the incidence varies widely, ranging from 6% to 15% in epilepsy services, especially pediatric ones, and 0.001% in neuro-otology services (See table 3).

Feature	Vestibular Epilepsy
Incidence in pediatric dizziness presentations	6-15% [8]
Prevalence in neurotology clinic/temporal lobe epilepsy cases	0.001% overall; 19% with vertiginous seizures [9]

Table 3: *Incidence of vestibular epilepsy.*

In my personal experience with 9,200 patients followed over 20 years, I have had three cases:

1. A 30-year-old woman who, preceded by a characteristic odor, had minute-long episodes of vertigo and subsequent confusion. The cause was a large astrocytoma involving the inner aspect of the left temporal lobe, in which focal spikes were detected on the EEG.
2. A 20-year-old man, a model by profession, began experiencing minute-long vertigo attacks after photoshoots (photostimulation). He then added forced head deviation seizures to the right. Several ictal videonystagmography recordings showed purely horizontal nystagmus that changes direction between seizures. His MRI was normal, and the EEG showed bilateral frontotemporal spikes.
3. A 10-year-old boy presented with right-sided nystagmus seizures and facial automatisms. His MRI was normal, and the EEG showed left temporal spikes.

The differential diagnosis includes other simple or complex partial seizures: visual, adversive, atonic, and absence seizures. The specific symptomatology of each seizure is crucial for DD.

Vestibular paroxysm, where pure vertigo lasts less than 1 minute, with no other associated symptoms.

Vestibular migraine, where the much longer duration and the patient's medical history will be the diagnostic keys.

Posterior territory TIAs and vestibular and non-vestibular drop attacks, a history of ABCD2, and a lack of consciousness are the hallmarks of DD.

Paroxysmal central vertigo, where attacks are produced in the brainstem (usually due to demyelination), the characteristics of the attacks, and the absence of focal spikes on the EEG, will provide guidance in this regard.

Panic attacks and phobic postural vertigo should be considered.

In my personal statistics, the incidence was 0.03%, which means that consultation for vertigo in epilepsy is uncommon; however, it should be considered, especially when vertigo is accompanied by sensory and/or focal motor symptoms. And this seems to be the key to differentiating it from other conditions that produce episodic vertigo.

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