

Lamotrigine with Anti-Anxiety Medication may be an Effective Treatment for Patients with Vestibular Migraine: A Retrospective Study

Lixin Zhang, MD, PhD;* D. Merika W. Sanders, PhD; Celia Zhang, AuD, PhD; Cora J. Blackwell, BA; Jamie So, MSc; Karen M. Stephenson BS; Maxwell G. Kahn, JD, LLM; Jennifer Cox, PhD

Dizziness, Balance & Tinnitus Center, Dent Neurologic Institute, Amherst, NY

Vestibular migraine (VM) is a condition characterized by prolonged vertigo, typically lasting minutes to days, with or without headaches. VM has a high comorbidity with anxiety and other mood disorders. Persistent postural-perceptual dizziness (PPPD) is chronic non-spinning vertigo, lasting seconds to minutes, which also has a high comorbidity with anxiety and mood disorders. Lamotrigine, a sodium channel blocker may be an effective prophylactic for VM, and anti-anxiety medication may be effective in reducing lingering vertigo symptoms in VM patients by eliminating symptoms of PPPD. Researchers conducted a retrospective chart review of 95 patients diagnosed with VM and prescribed lamotrigine to explore lamotrigine's effect on VM symptoms. Of the 95 patients examined, 68 patients continued lamotrigine and had at least one follow-up visit. 64 of the 68 patients (94%) taking lamotrigine reported significant improvement in their vertigo and migraine attacks from prior to treatment, while 4 (6%) reported no improvement. 67 patients (71%) of the 95 initially reviewed had comorbid anxiety/mood disorders. Of the 68 patients consistently taking lamotrigine, 43 (67%) of patients were also on anti-anxiety medication. Lamotrigine may be an effective prophylactic to reduce the number and severity of VM episodes. Furthermore, lamotrigine in conjunction with anti-anxiety medication may further reduce PPPD symptoms in patients and overall improve patient's quality of life. We propose a "Dizziness threshold theory" to explain this phenomenon. Patients with PPPD are hypersensitive to positional changes and visual stimulation; this suggests they have a lowered "dizziness threshold," the threshold neuronal activity has to meet trigger symptoms of dizziness. For patients with VM, we propose that while their "dizziness threshold" may remain normal, they have periods of neuronal hyperactivity which triggers episodes of vertigo.

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INTRODUCTION

Vestibular migraine (VM), also known as migraine associated vertigo, migraineous vertigo, and vertiginous migraine, is a debilitating vestibular disorder that has a lifetime prevalence of 2.7%.¹ It is defined as vertigo accompanied by headache, photophobia, phonophobia, or visual aura.² Table 1 lists the full criteria to diagnose VM from the International Classification of Headache Disorders 3 (ICHD-3) 2012, and Neuhauser's definition of migraine-associated vertigo (MAV), 2001.^{3,4} While terminology varies slightly, there are no meaningful differences between the two definitions.

VM is often misdiagnosed or underdiagnosed.⁵ Patients with VM describe varying dizziness symptoms, including spontaneous vertigo, positional vertigo, or visual/motion induced vertigo.⁶ VM attacks can last from a few minutes to days and can have a significant impact on a patient's quality of life.^{2,6} The condition shares many traits with Meniere's Disease and migraine with brainstem aura, and there is a lack of consistent testing for VM, making physician diagnosis difficult.^{5,7} Patient self-reported symptoms are almost identical between patients diagnosed with VM and patients diagnosed with migraine headache with brainstem aura that do not meet the VM diagnostic criteria but still experience vertigo, suggesting that VM might be underdiagnosed among migraine patients.⁸ Mallampalli et al. suggests that migraine and vestibular migraine may be two disorders on a singular spectrum that has a consistent underlying pathophysiology.⁶

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*Corresponding Author: Dizziness, Balance & Tinnitus Center, Dent Neurologic Institute, Amherst, NY. (Email: lzhang@dentinstitute.com)

Table 1. VM diagnostic criteria based on Neuhauser et al., 2001 and IHS Classification ICHD-3 criteria.^{3,4}

Neuhauser et al. 2001 MAV Diagnostic Criteria	Probably MAV	Definitive MAV
Episodic vestibular symptoms of at least moderate severity	X	X
Other causes ruled out	X	X
At least one of the following: • Migraine according to the IHS criteria • Migrainous symptoms during vertigo • Migraine-specific precipitants of vertigo • Response to antimigraine drugs	X	
Migraine according to IHS criteria		X
At least one of the following during at least two vertigo attacks: • Migrainous headache • Photophobia • Phonophobia • Visual or other auras		X

International Headaches Society – International Classification for Headache Disorders-3 for Vestibular Migraine	
A.	At least five episodes fulfilling criteria C and D
B.	A current or past history of migraine with or without aura
C.	Vestibular symptoms of moderate or severe intensity, lasting between 5min to 72hrs
D.	At least 50% of episodes are associated with at least one of the following three migrainous features: i) Headaches with at least two of the following four characteristics: • Unilateral location • Pulsating quality • Moderate or severe intensity • Aggravation by routine physical activity ii) Photophobia and phonophobia iii) Visual aura
E.	Not better accounted for by another ICHD-3 diagnosis or by another vestibular disorder

The migraine process can be split into four distinct phases: prodrome, aura, headache, and postdrome.⁹ The mechanism behind migraine pathophysiology is the subject of much debate; specifically, the pathophysiology of migraine aura is complicated as it can involve the visual, sensory, and language processing systems. One theory to explain migraine aura involves cortical-spreading depression (CSD), where a wave of depolarization travels through the brain followed by neuronal inhibition.¹⁰ CSD then activates the trigeminal-vascular system, which can lead to vasodilation associated with the headache phase of migraine.¹¹ Evidence suggests CSD originates in the cortex and then spreads. CSD passing through the occipital lobe can explain symptoms like photophobia and visual disturbances in aura, as well as the temporal relationship between aura and headache. There is also evidence that CSD can move through other brain structures, like the thalamus.¹² The parietal-insular-temporal area of the brain receives most of the vestibular sensory input.¹³⁻¹⁵ We theorize that CSD through the parietal-insular-temporal areas is the true cause of vestibular migraine, causing symptoms like nausea and vertigo associated with VM.

Vestibular disorders, migraine, and psychiatric disorders are closely related; it was reported that about half of all patients in dizziness clinics have comorbid anxiety and depression.¹⁶ Psychiatric disorders have also been associated with an

increased migraine frequency, resulting in a greater disability.^{17,18} The diagnosis of a primary vestibular disorder may cause secondary stress and anxiety conditions, thus creating a cyclical effect.¹⁹ Persistent postural-perceptual dizziness (PPPD), previously known as phobic postural vertigo or chronic subjective dizziness, is a common chronic dizziness condition often seen in tandem with patients diagnosed with anxiety.²⁰⁻²² Patients with PPPD have persisting dizziness or unsteadiness without vertigo that is worst during movement or with visual stimulation.²⁰ Diagnosis of PPPD requires symptoms for three months or more, and treatment can involve vestibular rehab, cognitive behavioral therapy, or medications like selective serotonin reuptake inhibitors.¹⁹⁻²² However, the current literature suggesting treatments for PPPD is often low-quality and inconsistent.

Similarly, the treatment guidelines for vestibular migraine are currently lacking.²³ Most treatment recommendations overlap with migraine: NSAIDs, antiemetics, triptans, beta-blockers, calcium channel blockers, antiepileptic drugs, etc., offering both abortive and prophylactic options for VM relief. Some antiepileptic drugs have been studied in relation to VM as a preventative treatment, including sodium valproate, topiramate, and lamotrigine. It was found that lamotrigine effectively reduced the amount of vertigo attacks in migraine patients, demonstrating its potential as a preventative

medication for VM.²⁴ Lamotrigine has also been shown to have a clinically significant effect on migraine aura reduction, and it has been proposed as an alternative for patients that do not tolerate Topiramate well.²⁵ Topiramate has several adverse effects, including metabolic acidosis, paresthesia and fetal toxicity.²⁶ Lamotrigine is generally more tolerated by patients than Topiramate, and has few serious side effects aside from allergic rash/Stevens-Johnson Syndrome.²⁷

Based on clinical observation, we hypothesize lamotrigine treatment may provide an effective, non-invasive preventive therapy for VM. Its low risk profile and inexpensive cost make it an ideal agent for repurposing.²⁸ Additionally, we examined the clinical comorbidity between mood disorders, VM, anti-anxiety medication, and PPPD, to evaluate if anti-anxiety medications could reduce PPPD-related episodes of dizziness and further improve patient quality of life.

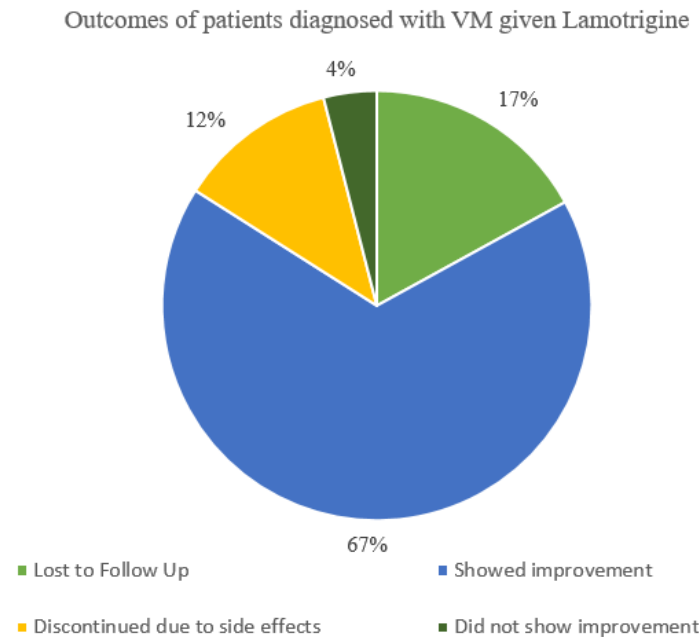


Figure 1. Pie chart describing the outcomes of VM patient's response to lamotrigine.

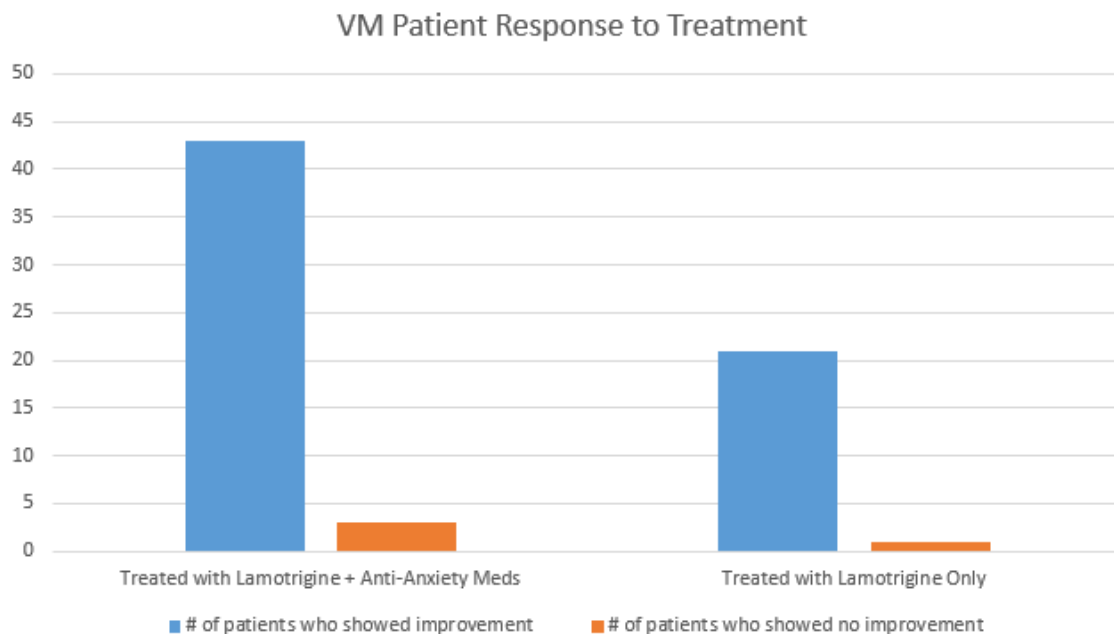


Figure 2. A comparison of patients who showed improvement or who showed no improvement on lamotrigine only, or lamotrigine and anti-anxiety medication therapy.

METHODS

Ethics

Protocol and consent forms for this prospective case review were approved by The State University of New York at Buffalo, Institutional Review Board. All procedures were followed in accordance to the approved protocol and all subjects gave written informed consent in accordance with the Declaration of Helsinki.

Participants

A retrospective chart review was performed at the Dent Dizziness, Balance, and Tinnitus Center (DBTC). Charts reviewed for this study were from 95 patients, all over the age of 18 who visited the Dent DBTC between February 2011 and December 2012. Inclusion criteria included being diagnosed with VM of at least moderate severity (as assessed by diagnosing physician) and prescribed lamotrigine as an off-label treatment. VM diagnosis criteria were based on Neuhauser's criteria (2001) and the ICHD-3 criteria, with active vertigo defined as two or more vertigo episodes per month for three months prior to the initial visit. Patients were also assessed for PPPD and diagnosed based on the following criteria:

- A) One or more symptoms of dizziness, unsteadiness, or non-spinning vertigo most days for 3 months or more,
- B) Persistent symptoms occur without specific provocation, but are exacerbated by upright posture, motion, and moving stimuli,
- C) Symptoms preceded by conditions that cause vertigo, unsteadiness, dizziness, or balance issues,
- D) Symptoms cause significant distress or functional impairment,
- E) Symptoms not better accounted for by another diagnosis.²⁰

Medication Dosage and Schedule

Patients maintained their pre-existing treatments and therapies. Patients with VM were prescribed lamotrigine as an off-label preventative treatment for their VM. Those prescribed lamotrigine underwent a four-week titration period starting at 25mg BID for two weeks, then 50mg BID for two weeks, then proceeded to the full 100mg BID dose. There is a known 8.3% incidence of adverse dermatological reaction while taking lamotrigine, which can develop into fatal Stevens-Johnson syndrome, so patients were informed to immediately stop taking the drug if they developed a rash anywhere on their body.²⁹ If patients could not tolerate 100mg lamotrigine, they were lowered to a dose of minimum 25mg BID; if patients still had VM symptoms while taking 100mg BID lamotrigine, their dose was increased up to 150mg BID lamotrigine. Dose adjustments occurred at regular follow-up patient visits.

Clinical Data

Data collected included demographic information, history of migraines and vertigo episodes, prior medical treatment, clinical presentation, clinical diagnosis, and frequency and severity of vertigo, dizziness, and migraines. Pre-existing history and family history of anxiety/depression and mood disorders, such as bipolar disorder and substance abuse, and

use of anti-anxiety medication was also recorded. In this study, anti-anxiety medication was defined as medication prescribed by a treatment provider to treat anxiety disorder.

Outcome Variables

Patient reports about increased/decreased symptoms of migraine, vertigo, and quality of life were measured at follow-up visits, including during the titration period and after three months of the full maintenance dosage treatment. Secondary outcomes included diagnosis of anxiety, use of anti-anxiety medications, and any reported changes with addition of anti-anxiety medications in patient report of quality of life. Adverse events were also monitored and data was collected from review of physicians' notes in clinical records.

RESULTS

Clinical Characteristics

Ninety-five adult patients (Age: $\mu = 42.7$, $\sigma = 13.5$) matched the criteria for this study (**Figure 1**). The demographics were 21 males and 74 females, 83 Caucasian, 6 African-American, 3 Hispanic, and 3 Unknown/Not Reported. Of the 95 patients, 67 (71%) had a diagnosis of anxiety/depression or other mood disorders. Out of the patients diagnosed with a mood disorder, 59 (88%) were on anti-anxiety medication during the study period.

Clinical Outcomes

Eleven patients (12%) discontinued lamotrigine due to side effects: seven developed a rash, one experienced jittery sensations, one developed feverish feeling in the cheek area, one felt as if the "skin was on fire," and one had an unknown side effect. Sixteen patients (17%) were lost to follow-up. Of the 68 patients that continued follow-up and did not discontinue lamotrigine, 64 (94%) reported improvement of VM symptoms with lamotrigine treatment, most notably in reduction of their vertigo attacks and a decrease in their migraine frequency and severity (**Figure 1**). Of the 64 patients who reported improvement, nine (14%) reported zero vertigo attacks after starting lamotrigine. 29 patients remained on the maintenance dose of 100mg BID; 11 patients were put on a lower dosage, and 24 patients had their dosage increased up to 150mg BID.

Among the 64 patients who reported improvement, 46 (72%) had comorbid anxiety/mood disorders and 43 (67%) were prescribed anti-anxiety medication. (**Figure 2**). Four patients reported no improvement of migraine or vertigo symptoms while on lamotrigine. Of these four, three (75%) had a comorbid mood disorder and were treated with anti-anxiety medication. Of the 28% (18/64) of improved patients who denied a personal history of anxiety/depression or mood disorders, 33% (6/18) of them have a noted family history of anxiety/depression or mood disorders.

Of the 43 patients on anti-anxiety medication, 56% (24/43) were on the medication prior to starting lamotrigine, 7% (3/42) started on the medication the same day they began lamotrigine, and 37% (16/43) were prescribed anti-anxiety medication after starting lamotrigine due to the patients complaints of non-

spinning dizziness or lightheadedness and subsequent diagnosis of PPPD, even after their major vertigo attacks were improved or resolved. 13 patients (30%) were prescribed/taking selective serotonin reuptake inhibitors like escitalopram or fluoxetine, 14 (33%) were on benzodiazepines as-needed, 10 (23%) were taking bupropion, 4 (9%) were taking serotonin-norepinephrine reuptake inhibitors, and 7

(16%) were unknown. 16 patients (37%) were on multiple anti-anxiety medications. Anti-anxiety medications were prescribed for PPPD symptoms and the dosage could be further adjusted based on the patient's response upon returning for a follow-up visit four to six weeks later. After taking anti-anxiety medications, further improvement of PPPD symptoms was reported in 73% (11/15) of patients.

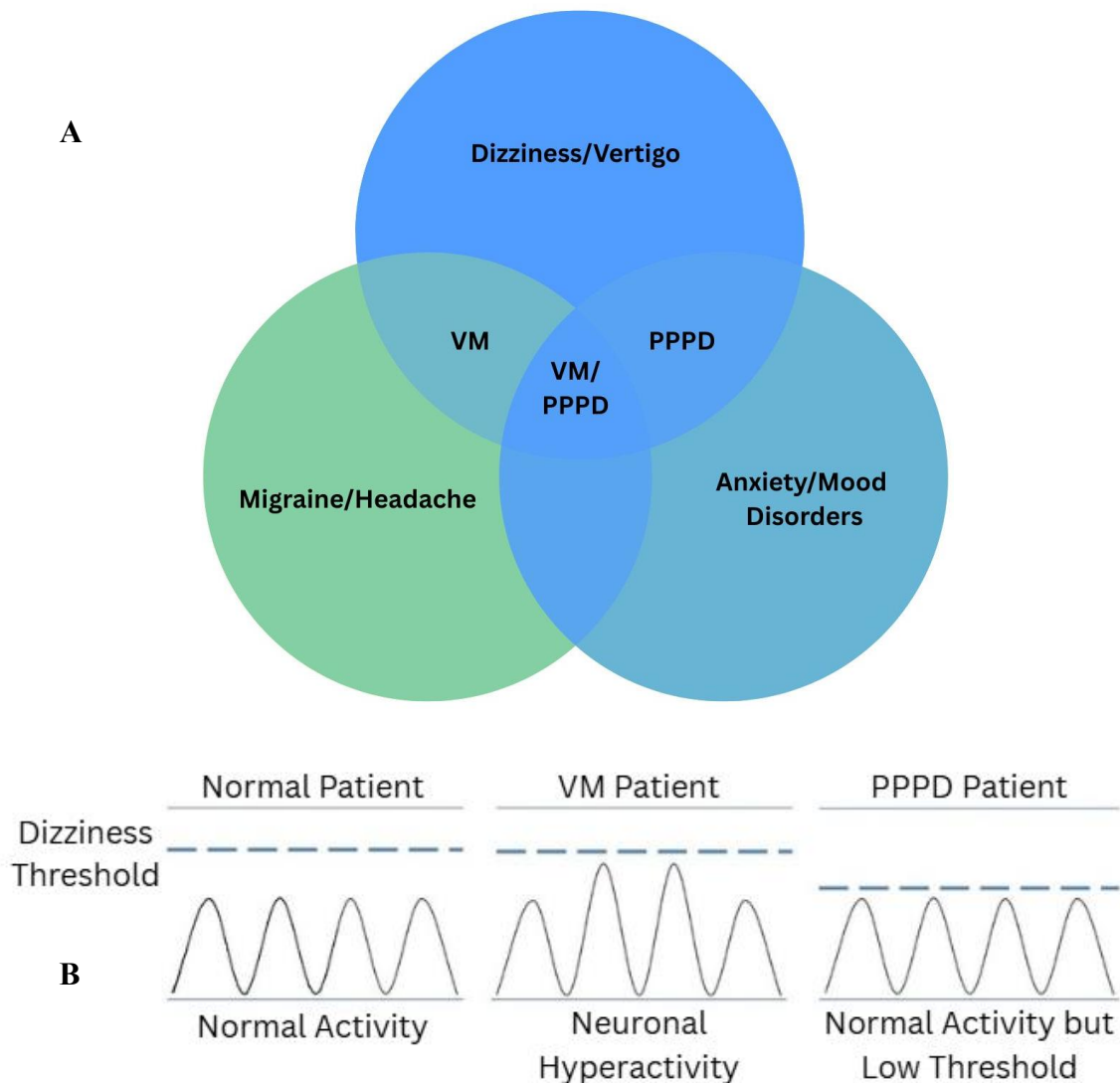


Figure 3. The central nervous system vestibular center “Dizziness Threshold Theory”: **(A)** The relationship between migraine, anxiety and vertigo/dizziness. **(B)** Dizziness threshold and neuronal activity of a normal healthy patient. VM patients show increased neuron activity in the vestibular center. Individuals with PPPD (anxiety-related dizziness) exhibit normal neuronal activity but a decreased dizziness threshold due to vestibular center hypersensitivity. Lamotrigine is used to prevent brain hyperactivity; anti-anxiety medication is used to raise dizziness threshold. The combination of the two treatments may be the best option for preventing migrainous vertigo attacks and treating PPPD.

DISCUSSION

In this retrospective chart review, we observed that lamotrigine was an effective preventative medication for VM reducing both the frequency and severity of vertigo and migraine attacks in the majority of patients. With few exceptions, dosages in the range of 100-150 mg BID were sufficient to achieve a consistent response of VM attack

reduction across the treatment period. The majority of VM patients that improved on lamotrigine had an underlying mood disorder and were already on anti-anxiety medications. We theorize that patients who began anti-anxiety medication after lamotrigine felt a greater decrease in dizziness related to positional changes than those who did not take anti-anxiety

medication, as anti-anxiety medication could treat a comorbid diagnosis of PPPD. Many of the patients without personal history of anxiety/depression or mood disorders had a family history, which suggests that genetic predisposition to mood disorders might also be a contributing factor to a patient's likelihood to develop dizziness disorders.

Lamotrigine is an antiepileptic medication and mood stabilizer that acts as a blocker on voltage gated sodium channels and is reported to prevent CSD.³⁰ The drug is generally well tolerated and has a favorable safety profile with titration to minimize potential side effects.³¹ Lamotrigine has been shown to have an effect on visual auras during migraine attacks and previous work also suggested that lamotrigine could be effective for preventing vertigo attacks in VM.^{32,33} If vertigo is truly a type of aura caused by CSD, it explains why lamotrigine would be an effective treatment.³⁴ Previous studies have also elucidated the effectiveness of using lamotrigine to manage mood disorders, especially bipolar disorder.³⁵ It is suggested that lamotrigine reduces neuronal hyperactivity to reduce symptoms in patients with bipolar disorder.^{35,36} Similarly, lamotrigine could reduce neuronal hyperactivity, which prevents the onset of vertigo attacks.

PPPD is often observed concurrently in patients who are diagnosed with VM. One study found that approximately 24% of patients with PPPD were diagnosed with VM.³⁷ The best management for these patients would include pharmaceutical treatment for both conditions, as only treating one condition would still result in the patient experiencing dizziness/vertigo. Lamotrigine for VM plus an anti-anxiety medication for PPPD are potential candidates for an effective pharmacotherapy combination.

We propose a "Dizziness Threshold Theory" to explain VM and PPPD (**Figure 3**). Patients with underlying mood disorders are more likely to develop vestibular disorders, which may be related to neuronal hypersensitivity or hyperactivity in the central nervous system's (CNS) vestibular center. This is most likely located in the temporal lobe or limbic system; these areas are involved in peripheral vestibular information processing and have been shown to play a role in mood disorders.³⁸⁻⁴¹ Patients with neuronal hypersensitivity present with PPPD, dizziness lasting for less than a minute that can be elicited by positional changes or visual stimulation. Due to their hypersensitivity, the neuronal threshold required to trigger dizziness is lowered, and even normal positional changes can cause PPPD. Patients with VM have episodic hyperactive neuronal activity at the vestibular center. Hyperactivity means the dizziness threshold can be reached, possibly due to CSD, even with a "normal" activation threshold. Based on the CNS "Dizziness Threshold Theory," we further propose that lamotrigine may be able to treat VM and migraine attacks by preventing CSD and neuronal hyperactivity. Concurrently, vestibular hypersensitivity could be related to mood disorders due to the CNS chemical imbalance like lower serotonin, so, anti-anxiety medications can decrease neuronal sensitivity by raising serotonin level and the dizziness threshold. This treatment course with

combination of lamotrigine and anti-anxiety medications could prevent the dizziness threshold from being reached, so preventing VM and PPPD. Similar theories have been proposed by Sarna et al. and Zhang et al., describing vestibular migraine and Meniere's disease as a migraine-related phenomenon related to CSD.^{42,43} Mallampalli et al also discuss perceptual thresholds in the vestibular system, describing how VM patients have low perception thresholds and vestibular hypersensitivity.⁶ Our theory builds and expands on their work to propose a comprehensive dizziness threshold theory explaining the root cause behind vertigo attacks in patients, in relation to mood disorders.

Future work should further examine the effectiveness of lamotrigine on VM with prospective double blind multi-center clinical trials to expand on the findings from this center. This study was limited in that it assessed improvement through patient reports and provider notes but lacked an objective measure of dizziness improvement. Effective treatment with lamotrigine in combination with an anti-anxiety medication in VM patients could support a breakthrough pharmacological preventive treatment for VM attacks, or possibly migraine in its entirety, and may eventually lead to a better understanding of the neural mechanisms of the disease. Furthermore, it is important to consider this potential therapy because of the current lack of effective prophylactic pharmacological treatments for this recurrent disabling disorder.

CONFLICT OF INTEREST STATEMENT

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

AUTHORS AND CONTRIBUTORS

LZ designed the study and took care of all patients during their visits. MW, LY and CZ conducted medical reviews and drafted the manuscript, and CB finalized the manuscript with the help of LZ, JS, SZ, MK, KS, and JC. All approved the final version of the manuscript.

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