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ORIGINAL ARTICLE

Longitudinal assessment of vestibular evoked myogenic potentials during one year of levetiracetam monotherapy in generalized epilepsy

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ABSTRACT

Objectives: This study aimed to evaluate the effects of one-year levetiracetam monotherapy on vestibular system function in patients with newly diagnosed generalized epilepsy using objective neurophysiological tests.

Patients and methods: In this prospective study, 12 patients (7 males, 5 females; mean age: 26.91 ± 11.36 years; range, 18 to 65 years) with generalized epilepsy who initiated levetiracetam treatment and 19 healthy controls (1 males, 18 females; mean age: 39.14 ± 11.88 years; range, 20 to 65 years) were included between March 2021 and March 2022. Cervical and ocular vestibular evoked myogenic potential (VEMP) tests were performed at baseline and after one year of treatment. Latencies, amplitudes, interpeak intervals, and asymmetry ratios were recorded. Intra- and intergroup comparisons were analyzed.

Results: After one year of levetiracetam treatment, significant reductions were observed in right cervical VEMP P13 and N23 peak amplitudes (p = 0.01; p = 0.001) and in right cervical VEMP interpeak latency (p = 0.03). In ocular VEMP measurements, right P1 latency and right N1-P1 amplitude showed significant decreases following treatment (p = 0.02). No significant pretreatment amplitude differences were observed between patients and controls.

Conclusion: One-year levetiracetam monotherapy was associated with measurable changes in selected VEMP parameters, suggesting mild effects on vestibular reflex pathways. Although these alterations were objectively detectable, their clinical impact appeared limited. Larger prospective studies are needed to clarify the long-term vestibular effects of levetiracetam.

Keywords: Epilepsy, levetiracetam, vestibular evoked myogenic potentials.

Epilepsy is a common neurological disorder affecting nearly 50 million people worldwide, and many patients require long-term antiseizure therapy.[1] Broad-spectrum, well-tolerated medications are preferred, particularly in generalized epilepsy. Levetiracetam, a widely used second-generation antiseizure drug, is effective in both focal and generalized epilepsies and is characterized by minimal pharmacokinetic interactions and favorable tolerability.[2] Nevertheless, adverse effects such as dizziness, imbalance, and ataxia have been reported with its use.[3,4] Older antiseizure medications, including phenytoin and carbamazepine, are well known to induce vestibular dysfunction, particularly at higher doses.[3] Although levetiracetam is generally considered safer, data regarding its potential impact on vestibular system function remain limited.

The vestibular system plays a crucial role in balance and eye movement coordination. Vestibular evoked myogenic potential (VEMP) testing is a

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noninvasive and reproducible method used to assess otolith organ function and vestibular reflex pathways.[5] Cervical VEMP (cVEMP) evaluates the saccule and inferior vestibular nerve through responses recorded from the sternocleidomastoid muscle, whereas ocular VEMP (oVEMP) reflects utricular and superior vestibular nerve function via recordings from extraocular muscles.[5,6] Due to its practicality and diagnostic value, VEMP has become an important tool in detecting peripheral and central vestibular disorders, including superior canal dehiscence syndrome, Meniere's disease, vestibular neuritis, and vestibular schwannoma.[6] Combined cVEMP and oVEMP testing provides complementary information regarding different components of the vestibular pathway.

In recent years, vestibular function in epilepsy has attracted increasing attention. Studies suggest that a considerable proportion of epilepsy patients may exhibit subclinical vestibular abnormalities. A 2021 study reported significant interictal VEMP abnormalities in epilepsy patients compared to healthy controls, with pathological cVEMP and oVEMP responses detected in a substantial percentage of cases.[7] Abnormalities in VEMP were also associated with disease duration, seizure frequency, and treatment type.[7] Furthermore, comparative research on valproate and levetiracetam monotherapies demonstrated objective impairments in vestibulo-ocular and vestibulocolic reflexes, although deterioration was more pronounced with valproate.[8] Despite these findings, studies specifically investigating the longitudinal effects of levetiracetam on vestibular function remain limited. In this context, the present study aimed to evaluate the impact of one-year levetiracetam monotherapy on cVEMP and oVEMP parameters in patients with generalized epilepsy.

PATIENTS AND METHODS

This prospective observational study was conducted at University of Health Sciences, Kartal Dr. Lütfi Kırdar City Hospital, Department of Neurology between March 2021 and March 2022. Twelve patients (7 males, 5 females; mean age: 26.91 ± 11.36 years; range, 18 to 65 years) with newly diagnosed generalized epilepsy were enrolled according to the current International League Against Epilepsy classification criteria valid at the time of enrollment. All patients initiated levetiracetam monotherapy after diagnosis and underwent VEMP testing at baseline (prior to treatment) and after one year of therapy.

Nineteen healthy volunteers (1 males, 18 females; mean age: 39.14 ± 11.88 years; range, 20 to 65 years) without a history of neurological, vestibular, or auditory disorders were included as the control group. None of the participants had previously used antiseizure medication. Exclusion criteria comprised known vestibular or hearing disorders, additional neurological or systemic diseases, use of medications other than levetiracetam, and pregnancy or breastfeeding. Written informed consent was obtained from all participants. The study protocol was approved by the University of Health Sciences Türkiye, Kartal Dr. Lütfi Kırdar City Hospital Clinical Research Ethics Committee (Date: 27.01.2021, Approval No. 514/194/22). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Vestibular evoked myogenic potential recordings were performed using an electromyography/evoked potential device (MEB-2300K; Nihon Kohden, Tokyo, Japan) in a quiet room under standardized conditions. Air-conducted tone burst stimuli (500 Hz, 95 dB nHL) were delivered binaurally at a repetition rate of 5 Hz. Electrode impedances were maintained below 5 kΩ.

For cVEMP, the active electrode was placed on the upper third of the sternocleidomastoid muscle, the reference electrode on the sternum, and the ground electrode on the forehead. Participants were instructed to rotate their head contralaterally to activate the sternocleidomastoid muscle during stimulation.

For oVEMP, the active electrode was positioned approximately 1 cm below the lower eyelid contralateral to the stimulated ear, the reference electrode 2 cm inferior to the active electrode, and the ground electrode on the forehead. Participants maintained an upward gaze to ensure adequate extraocular muscle activation.

For both cVEMP and oVEMP, latencies, peak-to-peak amplitudes, and asymmetry ratios were analyzed. For cVEMP, P13 and N23 latencies were recorded. For oVEMP, N10 and P15 latencies were measured. Peak-to-peak amplitudes were calculated as the voltage difference between the corresponding positive and negative waveform components.

The asymmetry ratio (AR) was calculated using the following formula: asymmetry ratio (%) = (Right-Left) / (Right + Left) × 100. This parameter

was used to quantify interaural differences and assess functional imbalance within the vestibular pathways.

Statistical analysis

Statistical analyses were performed using IBM SPSS version 25.0 software (IBM Corp., Armonk, NY, USA). Normality of continuous variables was evaluated with the Kolmogorov-Smirnov test. Data with normal distribution were expressed as mean ± standard deviation (SD). Paired t-tests were used to compare baseline and one-year measurements within the patient group. Independent samples t-tests or appropriate nonparametric tests were applied for comparisons between patients and controls. The level of statistical significance was set at $p < 0.05$.

RESULTS

Twelve patients with newly diagnosed generalized epilepsy completed baseline and 12-month follow-up assessments. Baseline comparisons and longitudinal within-group analyses were performed for all cVEMP and oVEMP parameters.

Cervical VEMP findings

In the right ear, significant post-treatment reductions were observed in P13 amplitude, N23 amplitude, P13-N23 interpeak latency, and peak-to-peak amplitude. No statistically significant differences were detected in right P13 or N23 latencies (Table 1a).

In the left ear, a statistically significant reduction was observed only in P13 peak amplitude.

TABLE 1a
Right-sided cVEMP parameters before and after levetiracetam (n = 12)

	Before treatment	After 12 months	
Parameters	Mean ± SD	Mean ± SD	*p*
P13 latency (ms)	11.73 ± 2.53	9.45 ± 8.80	0.35
N23 latency (ms)	19.71 ± 3.12	12.60 ± 11.22	0.07
P13 peak amplitude (μV)	4.95 ± 5.83	1.13 ± 1.80	0.01
N23 peak amplitude (μV)	3.43 ± 2.33	0.89 ± 1.38	0.001
P13-N23 interpeak latency (ms)	8.08 ± 3.21	3.94 ± 3.64	0.03
P13-N23 amplitude (μV)	11.27 ± 11.54	4.00 ± 5.80	0.01

TABLE 1b
Left-sided cVEMP parameters before and after levetiracetam (n = 12)

	Before treatment	After 12 months	
Parameters	Mean ± SD	Mean ± SD	*p*
P13 latency (ms)	10.86 ± 4.14	11.16 ± 7.23	0.87
N23 latency (ms)	17.77 ± 5.94	15.33 ± 9.56	0.31
P13 peak amplitude (μV)	3.33 ± 3.34	1.45 ± 1.55	0.04
N23 peak amplitude (μV)	3.80 ± 4.61	1.81 ± 1.97	0.07
P13-N23 interpeak latency (ms)	7.41 ± 3.61	5.67 ± 4.23	0.14
P13-N23 amplitude (μV)	10.51 ± 11.53	6.59 ± 6.10	0.17

cVEMP, vervical vestibular evoked myogenic potential; SD, standard deviation.

TABLE 1c
Bilateral cVEMP differences and asymmetry (n = 12)

	Before treatment	After 12 months	
Parameters	Mean ± SD	Mean ± SD	*p*
Right-Left P13 latency difference (ms)	2.45 ± 3.61	5.54 ± 6.04	0.22
Right-Left N23 latency difference (ms)	3.49 ± 5.42	7.93 ± 9.90	0.25
Amplitude asymmetry ratio (%)	26.39 ± 28.77	47.36 ± 45.95	0.24

cVEMP, vervical vestibular evoked myogenic potential; SD, standard deviation.

TABLE 2a
Right-sided oVEMP parameters before and after levetiracetam (n = 12)

	Before treatment	After 12 months	
Parameters	Mean ± SD	Mean ± SD	*p*
N1 latency (ms)	10.69 ± 3.12	8.88 ± 5.21	0.19
P1 latency (ms)	13.88 ± 5.08	7.83 ± 8.48	0.02
N-P interpeak latency (ms)	4.02 ± 1.91	2.47 ± 2.85	0.12
N1-P1 amplitude (μV)	0.97 ± 0.84	0.36 ± 0.72	0.02

TABLE 2b
Left-sided oVEMP parameters before and after levetiracetam (n = 12)

	Before treatment	After 12 months	
Parameters	Mean ± SD	Mean ± SD	*p*
N1 latency (ms)	10.06 ± 2.49	8.64 ± 5.74	0.42
P1 latency (ms)	13.53 ± 4.60	18.63 ± 22.17	0.42
N-P interpeak latency (ms)	4.05 ± 2.07	2.38 ± 2.83	0.10
N1 amplitude (μV)	0.67 ± 0.71	0.24 ± 0.35	0.01
P1 amplitude (μV)	0.55 ± 0.57	0.21 ± 0.44	0.03
N1-P1 amplitude (μV)	1.53 ± 1.58	0.58 ± 1.28	0.03

cVEMP, vervical vestibular evoked myogenic potential; SD, standard deviation.

TABLE 2c
Bilateral oVEMP differences and asymmetry (n = 12)

	Before treatment	After 12 months	
Parameters	Mean ± SD	Mean ± SD	*p*
Right-Left N1 latency difference (ms)	1.60 ± 0.99	2.18 ± 3.07	0.51
Right-Left P1 latency difference (ms)	1.12 ± 1.11	11.39 ± 24.10	0.17
oVEMP asymmetry ratio (%)	34.17 ± 20.25	17.06 ± 20.97	0.03

oVEMP, ocular vestibular evoked myogenic potential; SD, standard deviation.

No significant differences were found in latency measures or other amplitude parameters (Table 1b).

No statistically significant changes were detected in bilateral latency differences or amplitude asymmetry ratios over the one-year period (Table 1c).

Ocular VEMP findings

In the right ear, significant reductions were observed in P1 latency and N1-P1 amplitude following treatment. Other right-sided parameters did not demonstrate statistically significant differences (Table 2a).

Significant reductions were observed in left N1 amplitude, P1 amplitude, and N1-P1 amplitude. No statistically significant differences were found in latency measures (Table 2b).

A statistically significant change was observed in the oVEMP amplitude asymmetry ratio after treatment. No significant changes were detected in bilateral latency differences (Table 2c).

Baseline comparison of the patient and control groups

At baseline, right and left P13 latencies were significantly lower in patients compared to controls. No statistically significant differences were observed in other cVEMP parameters (Table 3a).

The mean age of the control group was significantly higher than that of the patient group (p = 0.013). No statistically significant differences were observed between patients and controls in baseline right or left P1 amplitudes (Table 3b).

TABLE 3a
Baseline cVEMP comparison between patient and control groups

	Patient group (n = 12)	Control group (n = 19)	
Parameters	Mean ± SD	Mean ± SD	*p*
Right P13 latency (ms)	11.73 ± 2.53	14.12 ± 2.34	0.012
Left P13 latency (ms)	10.86 ± 4.14	14.03 ± 2.03	0.008

TABLE 3b
Comparison of age and baseline oVEMP P1 amplitudes between patient and control groups

Parameters	Mean ± SD	*p*
Age (year)		
Patient group (n = 12)	26.91 ± 11.36	0.013
Control group (n = 19)	39.14 ± 11.88	
Right P1 amplitude (μV)		
Patient group (n = 12)	1.47 ± 4.24	0.630
Control group (n = 19)	2.07 ± 2.65	
Left P1 amplitude (μV)		
Patient group (n = 12)	0.55 ± 0.57	0.307
Control group (n = 19)	1.59 ± 3.43	

VEMP, vervical vestibular evoked myogenic potential; oVEMP, ocular vestibular evoked myogenic potential; SD, standard deviation.

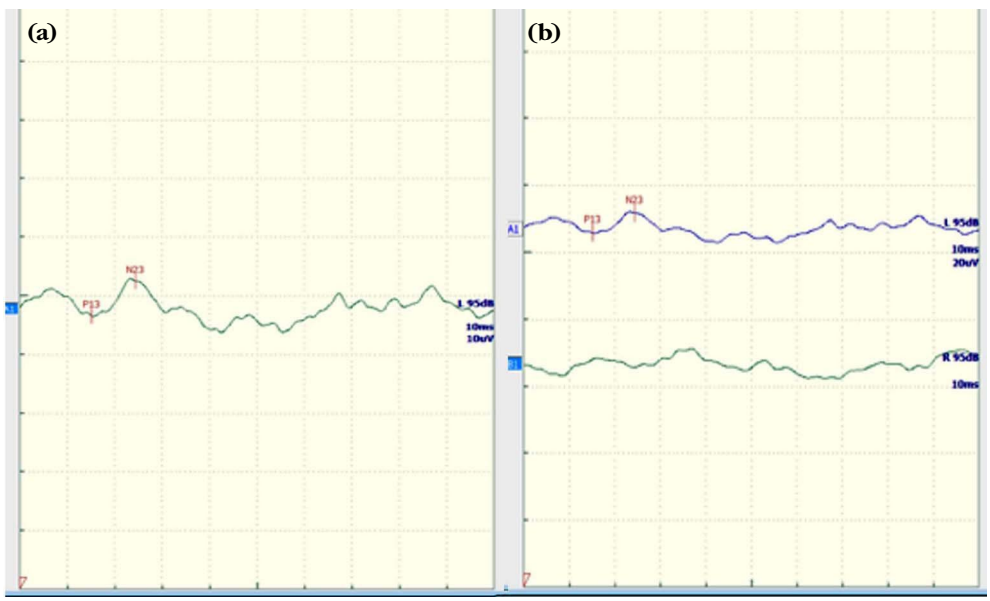

Figure 1. (a) Cervical vestibular evoked myogenic potential (cVEMP) results obtained from the left sternocleidomastoid muscle in a patient before medication (Nihon Kohden MEB-2300K). **(b)** In the same patient, a decrease in N23 amplitude is observed in the cVEMP response obtained from the left sternocleidomastoid muscle in the end of the 1st year of drug use (Nihon Kohden MEB-2300K).

DISCUSSION

The effects of antiseizure medications on vestibular function have been increasingly investigated in recent years. Previous studies demonstrated that a substantial proportion of patients with epilepsy had abnormal VEMP responses compared to healthy controls.[7] Pathological cVEMP responses were reported in

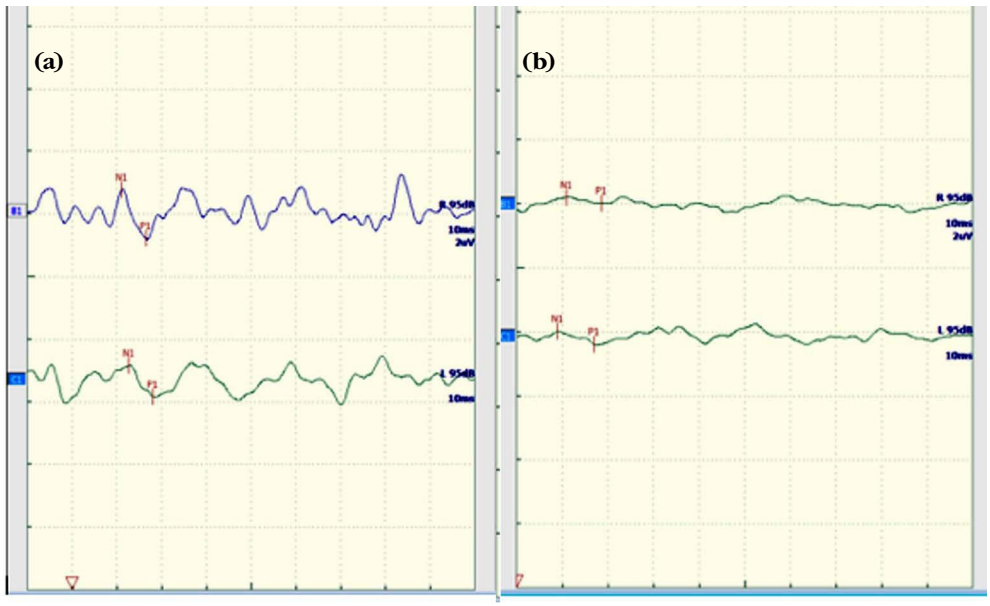

Figure 2. **(a)** Ocular vestibular evoked myogenic potential (oVEMP) results obtained from the right and left inferior oblique muscles in a patient before medication (Nihon Kohden MEB-2300K). **(b)** In the same patient, a prolongation of N1-P1 latencies and a decrease in amplitudes were detected in oVEMP responses obtained from bilateral inferior oblique muscles in the end of the first year of drug use (Nihon Kohden MEB-2300K).

65% and oVEMP abnormalities in 53% of epilepsy patients, with nearly half exhibiting bilateral involvement.[7] Moreover, VEMP abnormalities were correlated with seizure frequency, disease duration, and treatment exposure.[7]

In the present study, our cohort consisted of newly diagnosed generalized epilepsy patients who had not yet received antiseizure therapy at baseline. When pretreatment values were compared with those of the control group (n = 19), no statistically significant differences were observed in baseline oVEMP P1 amplitudes or most cVEMP amplitude parameters. Although shorter P13 latencies were detected in the patient group, the control group was significantly older (p = 0.013), and it is well established that VEMP amplitudes decline while latencies increase with advancing age.[9] Therefore, the latency difference observed at baseline is more plausibly attributable to age-related physiological variation rather than intrinsic vestibular hyperexcitability. Taken together, these findings suggest that vestibular reflex pathways are largely preserved in early-stage generalized epilepsy prior to prolonged treatment exposure.

After one year of levetiracetam monotherapy, statistically significant reductions were observed in several cVEMP amplitude parameters, particularly in right P13 and N23 peak amplitudes, as well as bilateral peak-to-peak amplitudes. In oVEMP testing, significant reductions were detected in right P1 latency and bilateral N1-P1 amplitudes. Importantly, asymmetry ratios did not demonstrate consistent statistically significant changes. This pattern indicates that the principal measurable effect was amplitude attenuation rather than marked interaural imbalance.

These findings are consistent with previous comparative investigations demonstrating that both levetiracetam and valproate monotherapies may produce objective vestibular alterations.[8] In the study by Cengiz et al.,[8] both drugs were associated with significant changes in cVEMP and oVEMP parameters, although deterioration was more pronounced in the valproate group. Our results align with these observations, showing measurable but relatively moderate changes under levetiracetam monotherapy. The absence of prominent asymmetry changes further supports the interpretation that the effect may represent generalized modulation of vestibular reflex excitability rather than focal unilateral dysfunction.

The possible mechanism underlying these findings may relate to the central inhibitory effects of antiseizure medications. Levetiracetam binds to synaptic vesicle protein 2A and modulates neurotransmitter release, thereby influencing

neuronal excitability. Increased inhibitory tone and modulation of ion channel activity within brainstem vestibular nuclei may attenuate synaptic transmission along vestibulocolic and vestibulo-ocular pathways.[7] Hamed[3] emphasized that long-term antiseizure therapy may be associated with dizziness, imbalance, ataxia, and nystagmus, potentially reflecting both central and peripheral vestibular involvement. The amplitude reductions observed in our cohort were compatible with such central modulation mechanisms.

Despite objective VEMP alterations, most patients in our study did not report clinically significant balance disturbances. This observation is consistent with the known tolerability profile of newer-generation antiseizure medications.[10] Compared to first-generation drugs such as phenytoin and carbamazepine, agents like levetiracetam and lamotrigine are associated with fewer balance-related adverse effects.[11] Dizziness and balance disorders are significantly more frequent with phenytoin than with levetiracetam (24% *vs.* 8%).[12] Therefore, although levetiracetam produces measurable electrophysiological changes, the magnitude of these alterations appears limited in clinical terms and milder than those described with older antiseizure drugs.[8,12]

Another relevant aspect is the broader impact of epilepsy and its treatment on patient well-being. Aggarwal et al.[13] demonstrated that drug-resistant epilepsy significantly impaired quality of life and increases neuropsychological burden. Although our cohort consisted of newly diagnosed patients receiving monotherapy, the identification of subclinical vestibular alterations may still be clinically meaningful. Even subtle balance disturbances, if cumulative over time, could potentially contribute to functional limitations or reduced quality of life.[14] In this context, VEMP testing may serve as a sensitive objective tool for monitoring vestibular function during long-term antiseizure treatment.

Several limitations should be acknowledged. First, the sample size was relatively small (12 patients and 19 controls), which may have limited statistical power and the detection of smaller effect sizes. Second, a significant age difference was present between the patient and control groups (p = 0.013). Given the established influence of aging on VEMP amplitudes and latencies,[9] age may have confounded baseline comparisons. Third, the study did not include a placebo or alternative antiseizure drug control arm, precluding direct comparative evaluation of drug-specific effects. Additionally, cumulative dose and dose-dependent effects of levetiracetam were not analyzed. Follow-up duration was limited to one year, and longer-term electrophysiological evolution remains unknown. Finally, although VEMP alterations were objectively demonstrated, the low prevalence of overt vestibular symptoms limits immediate clinical generalizability.

In conclusion, one year of levetiracetam monotherapy in patients with generalized epilepsy was associated with significant reductions in selected cVEMP and oVEMP amplitude parameters, while asymmetry ratios remained largely unchanged. Baseline vestibular function in newly diagnosed patients was comparable to that of controls, apart from age-related latency differences. The observed electrophysiological changes were objectively measurable but clinically mild and not accompanied by substantial balance complaints. Compared to older antiseizure medications, levetiracetam appears to exert a relatively limited negative impact on vestibular function. Vestibular evoked myogenic potential testing may provide a useful adjunctive method for detecting subclinical vestibular changes during antiseizure therapy. Larger prospective studies incorporating dose analysis and longer follow-up are warranted to clarify long-term vestibular effects.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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REFERENCES

1. World Health Organization. Epilepsy: key facts [Internet]. Geneva: World Health Organization; 2024 Feb 7 [cited 2026 May 1]. Available from: https://www.who.int/news-room/fact-sheets/detail/epilepsy.
2. Celdran de Castro A, Nascimento FA, Beltran-Corbellini Á, Toledano R, Garcia-Morales I, Gil-Nagel A, et al. Levetiracetam, from broad-spectrum use to precision prescription: A narrative review and expert opinion. Seizure 2023;107:121-31. doi: 10.1016/j.seizure.2023.03.017.
3. Hamed SA. The auditory and vestibular toxicities induced by antiepileptic drugs. Expert Opin Drug Saf 2017;16:1281-94. doi: 10.1080/14740338.2017.1372420.
4. Ogunsakin O, Tumenta T, Louis-Jean S, Mahbub A, Rabel P, Olupona T, et al. Levetiracetam induced behavioral abnormalities in a patient with seizure disorder: A diagnostic challenge. Case Rep Psychiatry 2020;2020:8883802. doi: 10.1155/2020/8883802.
5. Rosengren SM, Colebatch JG, Young AS, Govender S, Welgampola MS. Vestibular evoked myogenic potentials in practice: Methods, pitfalls and clinical applications. Clin Neurophysiol Pract 2019;4:47-68. doi: 10.1016/j.cnp.2019.01.005.
6. Dorbeau C, Bourget K, Renard L, Calais C, Bakhos D. Vestibular evoked myogenic potentials. Eur Ann Otorhinolaryngol Head Neck Dis 2021;138:483-8. doi: 10.1016/j.anorl.2021.01.001.
7. Kabel AEH, Afifi KH, ElFakhrany SM, Abdelghany HS, Kamel TB. Cervical and ocular vestibular evoked myogenic potentials in epileptic patients. Egypt J Otolaryngol 2021;37:51.
8. Cengiz DU, Çolak SC, Özdemir EA, Adıgüzel A. Effects of valproic acid and levetiracetam monotherapy on balance functions in patients with generalized epilepsy. Epilepsy Behav 2024;151:109622. doi: 10.1016/j.yebeh.2024.109622.
9. Hakami T. Efficacy and tolerability of antiseizure drugs. Ther Adv Neurol Disord 2021;14:17562864211037430. doi: 10.1177/17562864211037430.
10. Sayed SZ, Abdul Wahat NH. Effects of age on cervical vestibular evoked myogenic potentials and ocular vestibular evoked myogenic potentials using 750 hz tone burst stimuli among healthy adults. Malays J Med Sci 2022;29:53-64. doi: 10.21315/mjms2022.29.4.6.
11. Harris L, Hateley S, Tsang KT, Wilson M, Seemungal BM. Impact of anti-epileptic drug choice on discharge in acute traumatic brain injury patients. J Neurol 2020;267:1774-9. doi: 10.1007/s00415-020-09769-5.
12. Jory C, Oak K, Organ C, Mclean B, Shankar R. Head first - Review of epilepsy head injury risk and protection. Seizure 2019;71:66-79. doi: 10.1016/j.seizure.2019.06.013.
13. Wood AM, Thompson-Harvey A, Kesser BW. Vertiginous epilepsy in the pediatric population. Front Neurol 2024;15:1403536. doi: 10.3389/fneur.2024.1403536.
14. Aggarwal HK, Jain D, Bishnoi A. (2019). Quality of life in patients with drug resistant epilepsy. Turk J Neurol 2019;25:159-63.