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Pupillary Nystagmus as an Objective Neuro-Otological Biomarker in Vestibular Migraine: A Quantitative Pupillometric Study

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Abstract

Background: Vestibular migraine (VM) is a common cause of episodic vertigo, yet its diagnosis remains primarily clinical and is often complicated by the absence of reliable objective biomarkers. Pupillary nystagmus, reflecting spontaneous oscillations of pupil diameter, has been proposed as a potential clinical sign of VM, but its quantitative characterization remains limited. **Objective**: To evaluate the diagnostic value of pupillary nystagmus in VM and to provide a quantitative assessment using infrared pupillometry. **Methods**: In this case–control study, 69 patients with vestibular migraine and 13 healthy controls underwent comprehensive neuro-otological evaluation, including vestibular testing and pupillometric assessment. Pupillary activity was recorded using a dedicated infrared pupillometer, and oscillatory dynamics were quantified using the Pupillary Unrest Activity Level (PUAL), derived through spectral analysis (Larson–Neice algorithm). Statistical comparisons were performed using non-parametric methods. **Results**: PUAL values differed significantly between VM patients and controls (Wilcoxon test, $p = 2.265 \times 10^{-11}$), demonstrating a clear separation between groups. A cut-off value of 0.393 was identified as the upper limit of normality, suggesting that elevated PUAL values may indicate vestibular migraine. **Conclusions**: Pupillary nystagmus represents a clinically accessible sign that can be objectively quantified through infrared pupillometry. The PUAL index provides a measurable parameter reflecting altered vestibulo–autonomic dynamics in VM and may serve as a promising neuro-otological biomarker. The integration of pupillometric analysis with clinical evaluation may improve diagnostic accuracy and support the development of objective diagnostic tools in vestibular migraine.

Keywords: pupillary hippus; pupillary nystagmus; vestibular migraine; vestibular examination; dizziness; episodic vertigo

1. Introduction

Vestibular migraine (VM) is currently recognized as one of the most common causes of episodic vertigo in adults, accounting for a substantial proportion of patients presenting with recurrent vestibular symptoms in both outpatient and emergency settings [1–3]. Since its initial description and subsequent inclusion in the International Classification of Headache Disorders (ICHD), vestibular migraine has progressively emerged as a distinct clinical entity at the interface between neurology and neuro-otology [4–6]. Despite this recognition, VM remains a diagnostically challenging condition due to its heterogeneous clinical presentation and the frequent absence of pathognomonic objective findings. According to the Bárány Society and ICHD-3 criteria, the diagnosis of vestibular migraine is primarily clinical, based on the temporal association between vestibular symptoms and migrainous features together with the exclusion of alternative vestibular disorders [5,7]. However, these criteria

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rely heavily on patient-reported symptoms and clinical history, which may be unreliable or incomplete, particularly in patients with overlapping vestibular conditions or atypical presentations [8,9]. Consequently, diagnostic uncertainty remains common, and vestibular migraine is frequently misdiagnosed as Ménière's disease, benign paroxysmal positional vertigo, or functional dizziness [10–12]. From a pathophysiological perspective, vestibular migraine is believed to arise from complex interactions between central sensory processing, vestibular pathways, and migraine-related mechanisms. These include cortical spreading depolarization, altered brainstem excitability, and dysfunctional multisensory integration [13–15]. Neuroimaging and neurophysiological studies have demonstrated abnormalities within vestibulo-thalamo-cortical networks, supporting the concept of VM as a disorder of central vestibular processing rather than a purely peripheral dysfunction [16–18]. Nevertheless, the extent to which peripheral vestibular involvement contributes to the clinical phenotype remains debated. Objective vestibular testing in patients with vestibular migraine has produced heterogeneous and often conflicting results. Conventional vestibular assessments such as caloric testing and video head impulse testing (vHIT) are frequently normal or show non-specific abnormalities, limiting their diagnostic utility [19,20]. Similarly, vestibular evoked myogenic potentials (VEMPs) and subjective visual vertical (SVV) testing may demonstrate subtle alterations, yet these findings lack sufficient sensitivity and specificity to be considered reliable biomarkers of the disease [21–24]. As a result, VM continues to be regarded largely as a diagnosis of exclusion. In recent years, increasing attention has been directed toward subtle oculomotor abnormalities observed in patients with vestibular migraine. Several studies have reported spontaneous or positional nystagmus during both ictal and interictal phases, suggesting the presence of underlying central vestibular dysfunction [25,26]. Among these signs, pupillary nystagmus, characterized by rhythmic oscillations of pupil diameter, has been proposed as a potential indicator of vestibular imbalance and brainstem autonomic instability [27]. Recent advances in pupillometry have further expanded the physiological interpretation of these phenomena. Spontaneous oscillations of pupil diameter, commonly referred to as pupillary hippus, reflect the dynamic interaction between sympathetic and parasympathetic control mechanisms and higher-order central regulatory networks. These oscillations typically occur within a frequency range between approximately 0.04 and 2 Hz and are modulated by central autonomic and arousal-related mechanisms; pupillary dynamics are widely considered to reflect activity within brainstem noradrenergic systems, including the locus coeruleus [28]. The pupil therefore represents an accessible physiological window into central autonomic and neuromodulatory processes integrating arousal, sensory processing, and cognitive control. Within this framework, pupillary oscillations may provide indirect information about the functional stability of central vestibulo-autonomic networks. Vestibular migraine has increasingly been conceptualized as a disorder involving altered interactions between vestibular nuclei, brainstem arousal systems, and cortical sensory integration pathways. Dysregulation within these circuits could therefore manifest as abnormal oscillatory dynamics within pupillomotor pathways, potentially explaining the occurrence of pupillary nystagmus in some VM patients. Parallel to these developments, advances in functional vestibular testing have led to the introduction of modified head impulse paradigms such as the functional head impulse test (fHIT). Unlike conventional vHIT, which primarily measures vestibulo-ocular reflex gain, fHIT evaluates vestibular function under cognitively demanding visual conditions and therefore probes the functional integrity of vestibulo-ocular pathways during tasks that more closely resemble real-life sensory environments [29,30]. Preliminary evidence suggests that fHIT may reveal subtle vestibular dysfunction in central vestibular disorders, including vestibular migraine, even when traditional gain-based measurements remain within normal limits [31–33]. Taken together, these developments suggest that integrating advanced functional vestibular testing with physiological markers derived from pupillometry may provide new insights into the neuro-otological mechanisms of vestibular migraine. Pupillary oscillations, particularly pupillary nystagmus, may represent a non-invasive indicator of instability within vestibulo-autonomic-arousal networks, complementing established vestibular tests and potentially improving diagnostic accuracy in complex clinical scenarios. The aim of the present study is therefore to provide a comprehensive

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clinical and instrumental analysis of patients with vestibular migraine, with particular focus on pupillary nystagmus and functional head impulse testing. By integrating conventional vestibular assessments with advanced oculomotor and functional testing, this study seeks to identify objective patterns that may enhance the diagnostic framework of vestibular migraine and improve the understanding of its underlying neuro-otological mechanisms.

2. Materials and Methods

This case–control study aimed to quantitatively evaluate the diagnostic performance of the clinical sign defined as "pupillary nystagmus" in distinguishing patients with vestibular migraine from non-migraine subjects. The study included 69 participants (14 males and 55 females) aged between 18 and 75 years (mean age 47.1 years, standard deviation ≈ 17.3 years). Patients were included if they met the diagnostic criteria for vestibular migraine according to the Bárány Society. Patients with otological or neurological diseases were excluded from the study, with the exception of migraine with or without aura. A control group of healthy subjects was also included. This group consisted of 13 individuals (4 males and 9 females), aged between 18 and 75 years (mean age 40.7 years, standard deviation ≈ 14.8 years). These subjects had no history of symptoms or clinical signs attributable to migraine. Subjects with a history of otological or neurological disorders or previous head trauma were excluded from the control group.

All participants underwent a comprehensive clinical and instrumental otoneurological evaluation. Clinical assessment included detailed medical history, otoscopy, neurological examination with cerebellar testing and evaluation of cranial nerves, pure-tone audiometry, and the assessment of spontaneous, gaze-evoked, and positional nystagmus using infrared video-oculography goggles, as well as the head-shaking test. Instrumental vestibular testing included video head impulse testing (vHIT), functional video head impulse testing (fHIT), bithermal caloric testing performed according to the Fitzgerald–Hallpike protocol, with responses analyzed using Jongkees' formulas based on the slow-phase angular velocity of nystagmus, and cervical and ocular vestibular evoked myogenic potentials (cVEMPs and oVEMPs). Patients diagnosed with vestibular migraine also underwent contrast-enhanced magnetic resonance imaging (MRI) to exclude structural disorders of the central nervous system.

Pupillary activity was evaluated using a dedicated infrared pupillometer (Neurolight, Figure 1) specifically designed for the assessment of pupillary oscillatory activity.

Figure 1. Infrared digital pupillometer used for high-frequency recording of spontaneous pupillary oscillations.

The device measures spontaneous fluctuations in pupil diameter under ambient light conditions and calculates the Pupillary Unrest Activity Level (PUAL) using the Larson–Neice algorithm. The Larson–Neice algorithm consists of a standardized multi-step signal processing pipeline, including artifact correction (blink replacement using the last valid sample), removal of slow drifts, and spectral decomposition via Fast Fourier Transform (FFT). The PUAL is calculated as the sum of spectral amplitudes within a defined frequency band (typically 0.2–2 Hz), representing spontaneous

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pupillary oscillatory activity. PUAL represents the spontaneous oscillatory activity of the pupil around its mean diameter and reflects parasympathetic tone originating from the Edinger–Westphal nucleus. Higher PUAL values correspond to greater amplitude and variability of pupillary oscillations, reflecting increased instability of autonomic pupillomotor control. Measurements were obtained under standardized illumination conditions (approximately 100 lux). The recording consisted of 512 samples acquired at 60 Hz (8.53 seconds), with frequency analysis performed within the 0.2–2.5 Hz band. Pupillary data were processed using dedicated software implementing the Larson–Neice method, which applies artifact correction, removal of slow drifts, and Fast Fourier Transform (FFT) analysis to quantify oscillatory activity. The magnitude of pupillary nystagmus was therefore expressed as a single numerical value (PUAL). Two datasets of PUAL values were obtained, corresponding to the vestibular migraine group and the control group, and were subsequently compared using statistical analysis.

Statistical analyses were performed using dedicated software programs: Jamovi (Version 2.3, 2024), PSPP (PSPP Development Team, 2025; GNU Project, Boston, MA, USA), and RStudio Integrated Development Environment for R (Posit Software, PBC, Boston, MA, USA, 2025).
A preliminary assessment of data distribution was carried out using the Kolmogorov–Smirnov and Shapiro–Wilk tests. Since one of the two distributions, namely that of the migraine patients, was not normally distributed, comparisons between groups were performed using non-parametric methods, specifically the Wilcoxon test.

3. Results

Normality of the data distributions was first evaluated using the Kolmogorov–Smirnov and Shapiro–Wilk tests.In the control group, the Kolmogorov–Smirnov test showed a p-value of 0.7306, indicating no significant deviation from normality. The Shapiro–Wilk test confirmed this finding ($p = 0.487$), supporting a Gaussian distribution of the values. In contrast, the vestibular migraine group showed a clear deviation from normality. The Kolmogorov–Smirnov test yielded a p-value of 0.003234, while the Shapiro–Wilk test resulted in $p < 0.001$, indicating a statistically significant non-normal distribution. Because one of the two distributions was not normally distributed, non-parametric statistical methods were applied. Group comparisons were therefore performed using the Wilcoxon rank-sum test. The Wilcoxon test demonstrated a highly significant difference between vestibular migraine patients and healthy controls ($W = 308.5$, $p = 2.265 \times 10^{-11}$), allowing rejection of the null hypothesis that the two samples originate from the same population. These results indicate a clear statistical separation between the two groups with respect to Pupillary Unrest Activity Level (PUAL) values. Graphical representations of the data, including histograms and boxplot distributions (Figures 2 and 3), further illustrated the marked difference between vestibular migraine patients and control subjects.

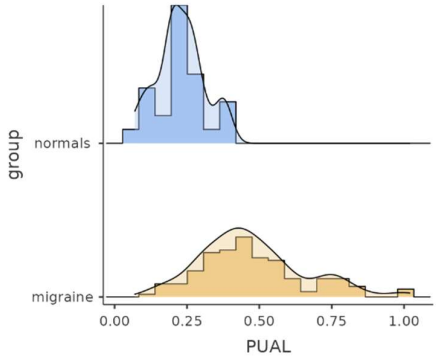

Figure 2. Boxplot distribution of Pupillary Unrest Activity Level (PUAL) values in vestibular migraine patients and healthy controls. A clear separation between the two groups is observed.

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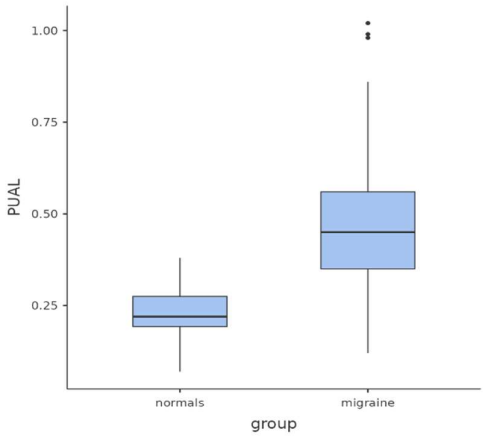

Figure 3. Histogram showing the distribution of PUAL values in vestibular migraine patients and controls. Control values follow a Gaussian distribution, whereas vestibular migraine patients show a shifted and non-normal distribution.

To define a potential diagnostic threshold, descriptive statistics of the control group were used. Since the distribution of the control values followed a Gaussian pattern, a cut-off value was estimated using twice the standard deviation of the control population. This approach yielded an upper normal limit of 0.393 for the PUAL index. Accordingly, PUAL values exceeding 0.393 may suggest the presence of vestibular migraine, indicating that pupillographic analysis could provide a quantitative parameter capable of distinguishing migraine patients from healthy subjects.

4. Discussion

The diagnosis of vestibular migraine (VM) remains predominantly clinical and relies on detailed anamnesis, with diagnostic criteria requiring the coexistence of migraine features and vestibular symptoms according to the Bárány Society and ICHD-3 classification [5,7]. However, in everyday clinical practice, diagnostic uncertainty is frequent, as patients often have difficulty accurately describing their symptoms or present with overlapping vestibular disorders [8–12]. In this context, the identification of objective or semi-objective clinical signs becomes particularly valuable. Traditionally, VM lacks pathognomonic instrumental markers, and conventional vestibular testing often yields normal or non-specific findings [19–24]. Therefore, the search for reliable clinical indicators that may support diagnosis has long been of interest. Among these, pupillary oscillatory phenomena—commonly referred to as hippus and here interpreted as "pupillary nystagmus"—have recently gained attention. The Pupillary Unrest Activity Level (PUAL) provides a quantitative representation of spontaneous pupillary oscillations (hippus), which in the clinical setting may be perceived as pupillary nystagmus. In this context, PUAL may serve as an objective and reproducible biomarker of altered vestibulo–autonomic dynamics in vestibular migraine. Previous studies have demonstrated a high prevalence of this sign in VM patients, with reported sensitivity and specificity exceeding 90%, suggesting that its presence may represent a clinically meaningful indicator even during the interictal phase [27]. Importantly, this sign is easily detectable during bedside examination under stable lighting conditions, making it particularly attractive in routine clinical practice.

From a pathophysiological perspective, pupillary oscillations reflect the dynamic balance between sympathetic and parasympathetic activity within central autonomic networks. Altered hippus dynamics have been linked to autonomic regulation and proposed as candidate biomarkers in neurological and clinical contexts [28], while in vestibular migraine the clinical relevance of pupillary nystagmus has already been suggested by previous bedside observations [27]. However, a major limitation of pupillary nystagmus has historically been its qualitative nature, due to the absence of standardized quantitative metrics and defined thresholds of normality. In the present

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study, we addressed this limitation by applying infrared pupillometry to obtain continuous recordings of pupil diameter and to derive a numerical parameter, the Pupillary Unrest Activity Level (PUAL), reflecting the magnitude of pupillary oscillations. This approach enabled direct comparison between vestibular migraine patients and healthy controls and demonstrated a statistically significant separation between the two groups, thereby providing objective validation of a previously descriptive clinical sign. Notably, our findings do not negate the clinical value of bedside observation. On the contrary, pupillary oscillations are often clearly appreciable even without instrumentation, and their detection remains a rapid and accessible component of the otoneurological examination. Quantitative pupillometry should therefore be considered complementary, providing a means to standardize and validate clinical impressions, particularly in diagnostically uncertain cases.

These interpretations are consistent with the emerging concept of oculomics and oculometrics, which propose that eye movements and pupillary dynamics can serve as non-invasive, objective biomarkers of neurological function [34]. In this paradigm, the pupil represents a dynamic interface reflecting the activity of distributed neural networks, including vestibular, autonomic, and arousal systems. Accordingly, the presence and quantitative characterization of pupillary nystagmus in VM may be interpreted as a measurable expression of instability within vestibulo–autonomic–arousal networks rather than a purely phenomenological observation. The integration of quantitative pupillometry with clinical examination therefore represents a promising step toward bridging the gap between subjective symptom-based diagnosis and objective biomarker-based assessment. Such an approach may improve diagnostic confidence, particularly in patients who do not fully meet established criteria or present with atypical features. Nevertheless, some limitations must be acknowledged. Although our findings demonstrate a clear statistical separation between groups, larger studies are required to validate the proposed cut-off values and to assess the influence of potential confounding factors such as age, medications, and comorbidities. Furthermore, the variability of pupillary oscillations across physiological states necessitates standardized recording conditions to ensure reproducibility

5. Conclusions

Pupillary nystagmus appears to represent a reliable clinical sign supporting the diagnosis of vestibular migraine, with previous studies demonstrating high sensitivity and specificity and confirming its value even during the interictal phase [27]. Although its presence can be quantitatively assessed using dedicated instrumental methods, it retains its role as a simple and reproducible bedside test in routine clinical practice. The analysis of pupillary nystagmus through digital pupillometry emerges as a promising neuro-otological tool for the functional assessment of central vestibular disorders, particularly vestibular migraine. Preliminary findings suggest that micro-oscillations of pupil diameter—reflecting the dynamic activity of vestibulo–autonomic networks—may mirror alterations in central modulation of the vestibulo-ocular reflex and in the sympathetic–parasympathetic balance, supporting their potential role as objective biomarkers of central dysfunction [34]. The integration of qualitative clinical evaluation with quantitative pupillometric analysis may enhance diagnostic accuracy and facilitate the identification of disease-specific patterns, contributing to a more refined differential diagnosis between vestibular migraine and other causes of vertigo. Future studies based on larger cohorts and standardized protocols will be necessary to validate pupillometric parameters as reliable physiological biomarkers of vestibular migraine.

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Institutional Review Board Statement: This study was conducted in accordance with the Declaration of Helsinki. Ethical review and approval were waived for this study. Due to its retrospective nature, it was not set up as part of a research project. Furthermore, the study does not include new experimental diagnostic protocols, and the patients included in the study were diagnosed according to national guidelines.

Informed Consent Statement: Written informed consent has been obtained from the patient to publish this paper.

Data Availability Statement: The original contributions presented in this study are included in the article. Further inquiries can be directed to the corresponding author.

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