Original article

Visual Pathway Dysfunction in Myasthenia Gravis Assessed by Pattern Reversal Visual Evoked Potentials: A Case–Control Study

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Abstract

Purpose: To evaluate visual pathway function in individuals with myasthenia gravis using pattern-reversal visual evoked potentials (VEP) and to compare P100 latency and amplitude between affected individuals and healthy controls.
Patients and Methods: A case–control design was used, including 10 individuals with myasthenia gravis (20 eyes) and 10 age-matched healthy controls (20 eyes). Pattern-reversal VEP was recorded monocularly under standardized conditions. P100 latency and amplitudes were measured and statistically compared between groups.
Results: Eyes from the myasthenia gravis group demonstrated significantly prolonged P100 latency (113.1 ± 5.74 ms) compared with controls (98.1 ± 3.33 ms; $P < 0.001$). P100 amplitude was markedly reduced in the myasthenia gravis group (2.65 ± 1.22 μV) relative to controls (9.00 ± 2.20 μV; $P < 0.001$).
Conclusion: The results of this study suggest that myasthenia gravis is associated with functional disturbance of the visual pathway, reflected by significant abnormalities in pattern-reversal VEP. Although not diagnostic, VEP may serve as a complementary tool for assessing visual involvement in this condition.

Keywords: Myasthenia Gravis; Visual Evoked Potentials; Visual Pathways; Electrophysiology; P100 Component; Pattern Reversal Stimulation

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Introduction

Neuromuscular junction (NMJ) disorders encompass a group of conditions that ultimately lead to muscle weakness, among which myasthenia gravis (MG) is the most common [1]. MG is an autoimmune disease characterized by antibodies targeting components of the postsynaptic membrane, including acetylcholine receptors, muscle-specific kinase, and lipoprotein-related protein 4, as well as titin and ryanodine receptor antigens [2]. This condition has become a prototype for understanding both autoimmune mechanisms and NMJ physiology [3].

Globally, the prevalence of MG is estimated at 12.4 per 100,000 individuals, highlighting the importance of early diagnosis and appropriate treatment [4]. Because MG impairs neuromuscular transmission, affected individuals experience fluctuating weakness of voluntary muscles, commonly involving the facial, bulbar, limb, and respiratory musculature [5].

Most people with MG initially experience ocular symptoms, and at least one symptom exacerbation during the course of the disease is expected in a large proportion of patients [6,7]. Ptosis and binocular diplopia are two hallmark ocular manifestations, and previous studies suggest that MG can influence visual pathway function [6,7].

Visual evoked potentials (VEP) provide an objective method for assessing visual pathway integrity and may detect abnormalities among MG patients even when structural imaging appears normal. Latency, amplitude, and waveform characteristics of the VEP provide objective information about the integrity of the optic nerve and central visual pathways, parameters widely used in clinical neurophysiology [8]. VEP has also been applied in other immune-mediated conditions affecting the visual system, where treatment-related changes in VEP amplitude have been documented, demonstrating their usefulness as an objective tool for monitoring functional visual changes [9].

The aim of the present study is to evaluate potential changes in VEP parameters, specifically P100 latency and amplitude, in individuals with myasthenia gravis.

Patients and Methods

Study Design and Participants

This case–control study included individuals with clinically confirmed myasthenia gravis and a group of healthy volunteers. The diagnosis of myasthenia gravis was established based on characteristic fluctuating weakness, neurological examination findings, and abnormalities on repetitive nerve stimulation or single-fiber electromyography, consistent with current clinical practice.

Participants with any ocular or retinal disease, optic nerve pathology, history of optic neuritis, or other visual pathway disorders were excluded to avoid confounding effects on VEP measurements.

Healthy controls were volunteers without neurological, ophthalmological, endocrine, or systemic conditions known to affect visual electrophysiology.

Both eyes of each participant were examined separately. This yielded 20 eyes in the myasthenia gravis group and 20 eyes in the control group. All participants had adequate best-corrected visual acuity and no history of amblyopia or significant refractive error.

Ethical Approval and Consent

The study received approval from the

Table 1: Demographic characteristics of case and control groups

Variable	Control Group (10 patients)	Case Group (10 patients)	P value*
Age (years, Mean ± SD)	53.7 ± 5.57	53.2 ± 4.89	0.677
Sex (Male/Female)	3 / 7	3 / 7	1.00

*P value for age was calculated using the Mann–Whitney U test, P value for sex was calculated using the Chi-square test.

institutional ethics committee. All participants were informed about the study procedures, and written informed consent was obtained from each participant before testing. All procedures complied with the Declaration of Helsinki.

VEP Procedure

Pattern reversal VEP was recorded in a dimly lit, quiet room with stable ambient temperature. Participants sat at a fixed distance from the stimulation monitor.

A high-contrast black-and-white checkerboard with alternating pattern reversal was used as the stimulus. The reversal rate, check size, contrast, and luminance were set according to established clinical electrophysiology conventions used in visual pathway assessment. Each eye was tested monocularly, with the fellow eye fully occluded. Participants were instructed to maintain steady fixation on a central target, as fixation instability can influence P100 measurements, particularly in disorders with extraocular muscle fatigability.

Electrode Placement and Signal Acquisition

Scalp electrodes were placed using the international 10-20 system. The active electrode was positioned at Oz, the reference electrode at Fz, and the ground electrode at the midline. Electrode impedance was kept below 5 kΩ.

Signals were amplified and filtered, and repeated stimulus responses were averaged to obtain clean waveform morphology. The N75, P100, and N145 components were identified in each recording. For each eye, P100 latency and amplitudes were recorded.

Statistical Analysis

Mean P100 latency and amplitude values were compared between the myasthenia gravis and control group. The distribution of each variable was assessed, and statistical tests were selected accordingly. A significance level of $P < 0.05$ was applied. Data were analyzed using IBM SPSS Statistics for Windows, Version 24.0 (IBM Corp., Armonk, NY, USA)

Results

A total of 10 individuals with myasthenia gravis (20 eyes) and 10 healthy controls (20 eyes) were included in the study. All participants completed the full pattern-reversal VEP protocol without technical artifacts or unusable recordings.

Demographic Characteristics

The case and control groups were well matched for age and sex. There were no statistically significant differences between the groups, indicating adequate comparability

Table 2: Pattern-reversal VEP findings in case and control groups

VEP Parameter	n (eyes)	Control (Mean ± SD)	Case (Mean ± SD)	P value*
P100 Amplitude (μV)	20	9.00 ± 2.20	2.65 ± 1.22	< 0.001
P100 Latency (ms)	20	98.1 ± 3.33	113.1 ± 5.74	< 0.001

*P values were calculated using the Mann–Whitney U test.

for electrophysiological analysis.

VEP Findings

Quantitative analysis of the pattern-reversal VEP recordings demonstrated clear abnormalities in the myasthenia gravis group. P100 latency was significantly prolonged, and P100 amplitude was markedly reduced when compared with controls. These findings are summarized in table 2.

The primary abnormality observed in the myasthenia gravis group was substantially reduced P100 amplitude, reflecting diminished cortical responsiveness to patterned visual stimulation.

Also prolongation of P100 latency indicates slower conduction along the anterior visual pathway.

These electrophysiological alterations support the presence of functional visual pathway disturbance in patients with myasthenia gravis, consistent with previously reported findings.

Discussion

This case-control study compared pattern-reversal visual evoked potential (VEP) parameters between individuals with myasthenia gravis and age-matched healthy controls. The MG group demonstrated clear abnormalities in P100 parameters, including prolonged latency and reduced amplitude, indicating functional involvement of the visual pathway.

These results are consistent with previous investigations. Dziadkowiak et al., [10] examined 42 patients with MG and similarly reported delayed P100 latency, supporting the presence of visual pathway disturbances in this condition. Earlier work by Fotiou et al., [11] also described both prolonged latency and reduced amplitude of the P100 component in MG patients, further reinforcing the electrophysiological evidence of visual system dysfunction.

In addition to these visual electrophysiological findings, multimodal studies have demonstrated that MG can produce abnormalities in brainstem auditory and somatosensory evoked potentials, indicating broader involvement of afferent pathways [10]. These broader electrophysiological disturbances support the interpretation that the VEP abnormalities observed here may reflect part of a wider pattern of sensory pathway involvement rather than an isolated visual pathway change [10].

The fluctuating and often progressive nature of myasthenia gravis, including its tendency for symptom exacerbations over time, reinforces the relevance of sensitive electrophysiological markers when evaluating functional involvement of systems such as the visual pathway [12]. Given that early clinical manifestations of MG are frequently ocular and may initially be subtle, the presence of VEP abnormalities in our patients complements

the broader understanding that early MG can benefit from objective neurophysiological assessment to detect functional deficits not evident on routine examination [12].

The prolonged P100 latency observed in our cohort is consistent with the established role of VEP as a sensitive measure of conduction delay within the visual pathways, particularly in conditions involving impaired central conduction [8]. Likewise, the reduction in P100 amplitude corresponds with reports that amplitude attenuation may reflect axonal or functional compromise of the visual system, complementing the latency findings [8]. Comparable VEP methodologies have also been applied in other immune-mediated disorders affecting visual function, where amplitude changes rather than latency shifts were shown to be the primary marker of treatment-related functional variation [9]. Additionally, prior work demonstrated that increased intraindividual variability in VEP parameters may signal subtle functional disturbances of the visual pathways, a concept compatible with the electrophysiological alterations observed in our cohort [9].

In summary our findings align with the existing literature and suggest that VEP abnormalities may reflect impaired conduction or synaptic transmission along the visual pathway in myasthenia gravis. The consistency across studies highlights the potential value of VEP as a complementary tool for evaluating visual involvement in MG.

Several limitations should be considered when interpreting these findings. The sample size was small, which limits the statistical power and generalizability of the results. All patients were examined at a single center, and variation in disease severity, duration, or treatment status could not be fully controlled. The cross-sectional nature of the study also prevents assessment of longitudinal changes in VEP parameters or their relationship to clinical fluctuations. Furthermore, only pattern-reversal VEPs were analyzed, and other electrophysiological modalities that might provide complementary information were not included.

Future research should aim to include larger and more diverse patient cohorts to better characterize the spectrum of visual pathway involvement in MG. Longitudinal designs would allow evaluation of how VEP parameters change over time and respond to therapeutic interventions. Incorporating additional electrophysiological measures, such as multifocal VEPs or optical coherence tomography, may also help clarify structure–function relationships within the visual system. Comparative analyses with other conditions that affect visual processing could further define the specificity and diagnostic value of VEP abnormalities in myasthenia gravis.

Conclusion

The results of this study suggest that myasthenia gravis is associated with functional disturbance of the visual pathway, reflected by significant abnormalities in pattern-reversal VEP. Although not diagnostic, VEP may serve as a complementary tool for assessing visual involvement in this condition.

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Footnotes and Financial Disclosures

Conflict of interest:

The authors have no conflict of interest with the subject matter of the present manuscript.