

Photophobia in Migraine: Clinical and Neurophysiological Insights into Ocular Versus Central Mechanisms

Basim Abdulelah Ali Al- Janahi

Al Diwanyia Teaching Hospital/Neurology Center, Mbchb, F.I.C.M.S Neurology

Annotation: Background: Photophobia is one of the most disabling symptoms of migraine, affecting more than 80% of patients. Although traditionally attributed to ocular hypersensitivity, evidence suggests a central neural mechanism involving the thalamus and visual cortex.

Objective: To evaluate the relative contribution of ocular versus central mechanisms to photophobia in migraine patients.

Methods: Case-control study, 60 migraine patients and 40 healthy controls, with ophthalmic examination, photophobia threshold testing, visual evoked potentials (VEP), and validated questionnaires.

Results: Migraine patients had significantly lower photophobia thresholds ($2,800 \pm 320$ lux) than controls ($4,600 \pm 410$ lux; $p < 0.001$). VEP recordings revealed shorter P100 latencies and higher amplitudes in migraineurs ($p < 0.01$). Ocular health parameters showed no significant differences.

Conclusion: Photophobia in migraine is predominantly mediated by central neural mechanisms. Management should target cortical hyperexcitability and thalamic sensitization.

Keywords: Migraine, Photophobia, Visual Evoked Potentials, Cortical Hyperexcitability, Thalamus.

Introduction

Migraine is a highly prevalent neurological disorder, affecting approximately 15% of the global population, characterized by recurrent headaches and sensory hypersensitivities. Photophobia, an abnormal intolerance to light, is one of the most disabling migraine symptoms, occurring during attacks and often persisting between them.

Previously considered a peripheral phenomenon related to retinal hypersensitivity, photophobia is now recognized as involving central mechanisms, particularly hyperexcitability of the visual cortex and sensitization of the posterior thalamus. Intrinsically photosensitive retinal ganglion cells (ipRGCs) play a role in transmitting light information to both visual and pain-processing centers, explaining photophobia even in some blind individuals.

This study aims to assess ocular versus central contributions to photophobia in migraine through clinical, ophthalmological, and neurophysiological methods.

Literature Review

Photophobia affects over 80% of migraineurs and significantly impacts quality of life. ipRGCs in the retina transmit signals to the thalamus, visual cortex, and pain centers. Peripheral mechanisms include increased retinal sensitivity, but central mechanisms dominate, involving posterior thalamic activation and cortical hyperexcitability.

Studies in blind migraineurs with intact ipRGCs confirm a non-visual pathway for photophobia. Clinical assessment tools include the Visual Light Sensitivity Questionnaire (VLSQ), photophobia threshold testing, and VEP recordings. Treatments include tinted lenses, migraine prophylaxis, and emerging neuromodulation approaches.

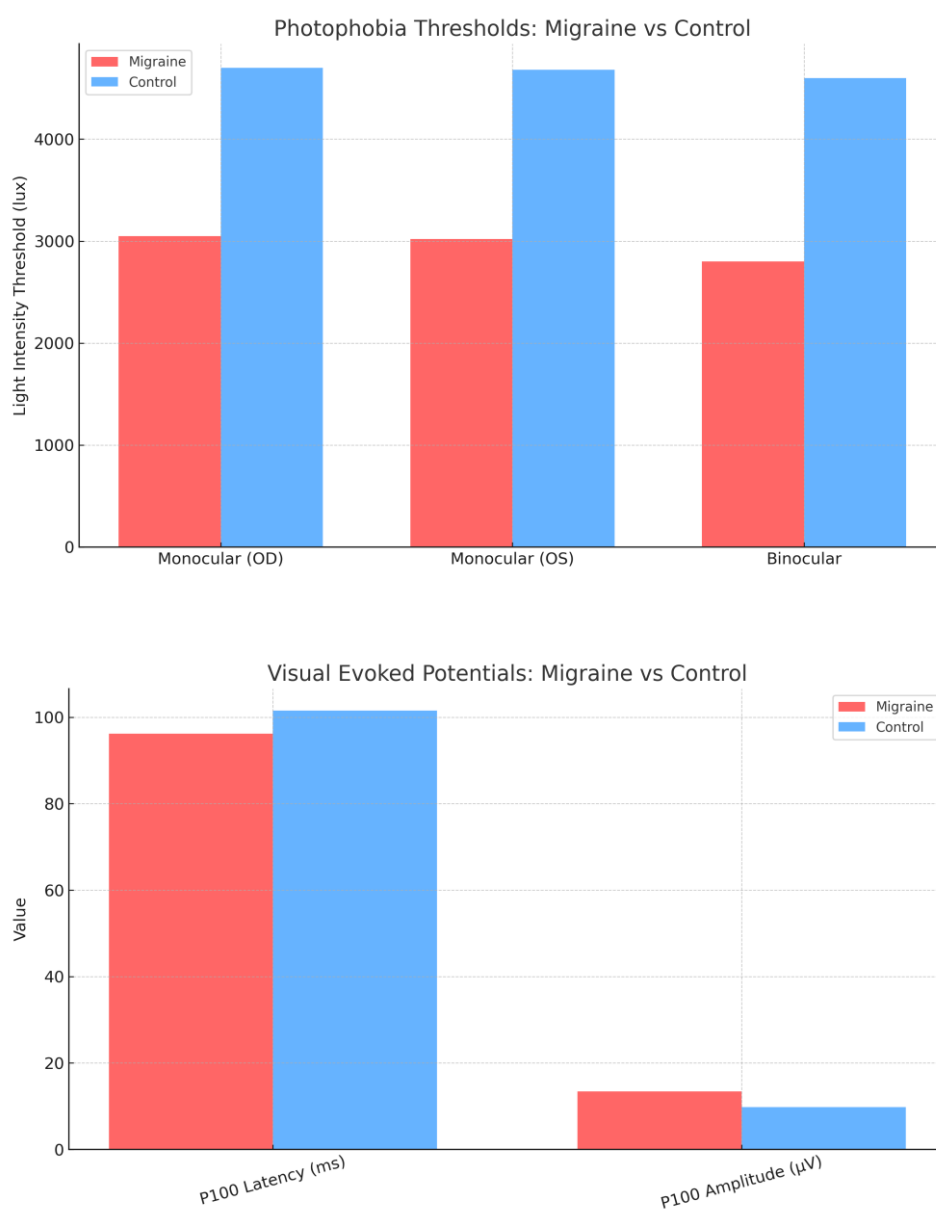
Methodology

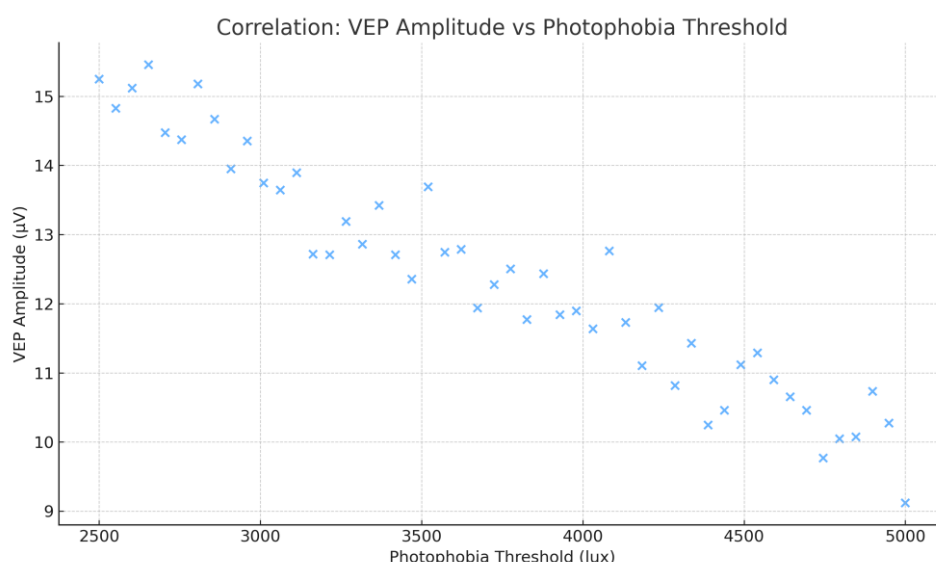
Design: Hospital-based case-control study at Al-Diwaniya Teaching Hospital (Jan 2019 – Dec 2020).
Participants: 60 migraine patients (ICHD-3 criteria) and 40 matched controls.
Assessments: Ophthalmological examination, photophobia threshold testing, VEP recordings, VLSQ and MIDAS questionnaires.

Data Analysis: SPSS v26; t-tests, chi-square, correlation, and regression analyses; $p < 0.05$ significant.

Results

Photophobia thresholds were significantly lower in migraineurs. VEP results showed cortical hyperexcitability. Ocular parameters did not differ significantly.





Discussion

Findings confirm central mechanisms dominate in migraine photophobia, consistent with prior research. VEP amplitude correlated with photophobia severity, while ocular health remained normal. This suggests management should focus on reducing cortical hyperexcitability rather than treating the eyes.

Conclusion

Photophobia in migraine is primarily centrally mediated. Targeted migraine prophylaxis and sensory modulation strategies are recommended.

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