Міністерство охорони здоров'я України
Національний медичний університет
імені О. О. Богомольця

Ministry of Health of Ukraine
Bogomolets National Medical University

USMYJ

НАУКОВО-ПРАКТИЧНЕ ВИДАННЯ

УКРАЇНСЬКИЙ НАУКОВО-МЕДИЧНИЙ МОЛОДІЖНИЙ ЖУРНАЛ

THEORETICAL AND PRACTICAL EDITION

UKRAINIAN SCIENTIFIC MEDICAL YOUTH JOURNAL

№4 (158) 2025

ЗМІСТ/CONTENTS

Сторінки/Pages

Medicine/ Медицина

ISSN 2786-6661
eISSN 2786-667X

UDC: 617.7-002.7-073.75:577.112.3:577.175.5
https://doi.org/10.32345/USMYJ.4(158).2025.27-34

Received: July 25, 2025
Accepted: October 27, 2025

Diagnostic and Prognostic Potential of Circulating HSP70 and GRIN2B in Primary Open-Angle Glaucoma

Taras Nedilka[1], Nina Lutsenko[1,2]

[1] Municipal Non-Profit Enterprise "Zaporizhzhia Regional Clinical Hospital" of the Zaporizhzhia Regional Council, Zaporizhzhia, Ukraine.

[2] Zaporizhzhia State Medical and Pharmaceutical University, Educational and Scientific Institute of Postgraduate Education, Zaporizhzhia, Ukraine.

Address for correspondence:

Taras Nedilka

E-mail: hardcoresunlight@gmail.com

Abstract: *Primary open-angle glaucoma is increasingly recognized as a multifactorial neurodegenerative disease in which the progression of optic nerve damage is driven not only by elevated intraocular pressure but also by disturbances in vascular regulation, metabolic imbalance, oxidative stress, and glutamate-mediated excitotoxicity. These mechanisms create the need to identify circulating biomarkers capable of reflecting early and ongoing injury of retinal ganglion cells. The HSP70 is considered an indicator of cellular stress and a potential mediator of endogenous neuroprotection, while the NR2B subunit of the glutamate receptor, encoded by the GRIN2B gene, is involved in excitotoxic neuronal damage. The present study investigated the plasma concentrations of these two markers in individuals with primary open-angle glaucoma and healthy participants, as well as their dynamics after a short course of neuroprotective therapy consisting of reduced glutathione and citicoline. The findings demonstrated a significantly higher level of HSP70 in patients compared with healthy individuals, supporting its role as a marker of oxidative and stress-related neuronal injury. In contrast, the GRIN2B marker did not differ significantly between groups before treatment, although a tendency toward higher values in patients was observed. After ten days of combined therapy, neither marker showed a statistically significant group-level change, which may reflect substantial inter-individual variability, insufficient treatment duration, or the need for larger sample sizes to detect subtle neurobiological effects. Despite the lack of a significant difference in baseline values, the GRIN2B marker exhibited a strong prognostic ability in identifying individuals who demonstrated a favorable biochemical response to treatment, suggesting potential utility in patient stratification and monitoring early therapeutic effects. HSP70 demonstrated limited predictive value over the short therapeutic interval, which may reflect the complexity of its regulation and the need for longer observation windows. Overall, the results indicate that these circulating proteins may complement existing clinical tools for assessing disease mechanisms in primary open-angle glaucoma. HSP70 may serve as an indicator of oxidative stress and retinal ganglion cell injury, while the GRIN2B marker may hold promise as an early indicator of treatment response. Further research is required to determine their stability over time, relevance in different disease stages, and suitability for integration into clinical decision-making.*

Key words: Apoptosis, Glaucoma, Glutamate Receptor, HSP70 Heat-Shock Proteins, Oxidative Stress, Nerve Neuroprotection, Degeneration.

ISSN 2786-6661
eISSN 2786-667X

Introduction

Glaucoma is one of the leading causes of irreversible blindness, with primary open-angle glaucoma (POAG) being its most common form in older adults. Although elevated intraocular pressure remains a key factor, current evidence indicates that the progression of glaucomatous neuropathy is also driven by vascular, metabolic, oxidative, and excitotoxic mechanisms [1, 2]. This has increased interest in systemic biomarkers of neurodegeneration for assessing the condition of retinal ganglion cells and the effectiveness of neuroprotection.

In recent years, compelling evidence has accumulated regarding the role of oxidative stress in the pathogenesis of glaucoma. Meta-analyses and systematic reviews have demonstrated an imbalance between pro-oxidant and antioxidant systems in glaucoma patients—characterized by elevated oxidative markers and reduced activity of antioxidant enzymes and reduced glutathione [3, 4].

Heat shock proteins, particularly HSP70, are considered universal molecular chaperones that are induced in response to various forms of cellular stress (ischemia, oxidative injury, inflammation). They may reflect both the degree of tissue damage and the activation of endogenous neuroprotective mechanisms. A recent literature review demonstrated that HSP70 expression increases in ocular tissues across a broad spectrum of pathologies, including glaucoma, and that most experimental studies show protective effects of HSP70 on the retina and optic nerve [5]. In experimental ischemia–reperfusion models, pharmacological induction of HSP70 is accompanied by reduced apoptosis of retinal ganglion cells and decreased glial activation, confirming its key role in neuroprotection [6]. Several reports have shown that alterations in HSP70 expression in blood and aqueous humor are associated with the risk of POAG development and progression, although these findings remain fragmented and require confirmation in larger cohort studies [7]. Therefore, HSP70 may serve as a circulating marker of stress-induced damage to the visual pathway in glaucoma; however, its clinical relevance in POAG remains insufficiently defined.

In parallel with oxidative stress, glutamate-mediated excitotoxicity via NMDA receptor activation plays a crucial role in retinal ganglion cell death. The NR2B subunit of this receptor is encoded by the GRIN2B gene and is critical for calcium-dependent signaling that triggers apoptosis and neurodegeneration [8]. During acute ischemic injury of neural tissues, degradation fragments of NR2 (the so-called NR2 peptide), as well as autoantibodies to NR2A/NR2B, can be detected in the blood and are considered specific biomarkers of excitotoxic neuronal injury. Contemporary reviews on biomarkers of stroke and transient ischemic attacks identify the NR2 peptide and NR2A/NR2B autoantibodies as among the most promising markers of acute ischemic brain damage, demonstrating high sensitivity and specificity [9].

Despite the clear pathophysiological parallels between glaucomatous optic neuropathy and other chronic neurodegenerative or ischemic CNS disorders, systemic markers of NMDA-receptor-related excitotoxicity (NR2/GRIN2B) remain virtually unexplored in POAG patients. Existing studies largely focus on genetic variants of GRIN2B and their role in psychiatric and cognitive disorders rather than on the measurement of circulating proteins or their fragments as indicators of glutamate-mediated neuronal damage [10]. Data on circulating GRIN2B/NR2 levels in glaucoma patients, their association with clinical characteristics of the disease, and their dynamics during treatment are currently lacking, creating a substantial gap in current knowledge.

A distinct direction in modern glaucoma research is the development of effective neuroprotective strategies aimed at preserving retinal ganglion cells, independently of intraocular pressure. Citicoline, a precursor of phosphatidylcholine and acetylcholine, is considered one of the key neuroprotective agents. Recent randomized trials have shown that oral citicoline (as part of combination regimens) improves pattern electroretinogram parameters in patients with open-angle glaucoma with well-controlled IOP [11]. Additionally, recent studies on oxidative stress in POAG highlight reduced levels of reduced glutathione, providing

ISSN 2786-6661
eISSN 2786-667X

a rationale for the use of glutathione-containing agents as components of antioxidant therapy [4].

Thus, on the one hand, the search for reliable circulating biomarkers reflecting oxidative stress (HSP70) and glutamate-mediated excitotoxicity (GRIN2B/NR2) in POAG patients remains highly relevant; on the other hand, evaluating their dynamics during combined neuroprotective therapy with citicoline and glutathione represents an important research objective.

Aim

To investigate the levels of HSP70 and GRIN2B in patients with POAG and to evaluate the dynamics of changes in these biomarkers following a course of reduced glutathione and citicoline.

Materials and methods

The study included 47 individuals aged 50 to 77 years, comprising 22 women and 25 men. The control group (group 1) consisted of 9 healthy participants with no ophthalmic or systemic pathology. The main group (group 2) included 38 patients with POAG and compensated intraocular pressure on standard hypotensive therapy, without concomitant neurological diseases or other conditions that could potentially affect metabolic or neurodegenerative biomarkers. Exclusion criteria for both groups included the use of medications capable of modulating oxidative stress or neuroprotective mechanisms (excluding ocular hypotensive therapy and the study-assigned treatment). Among the main group, 15 patients received pharmacological therapy for 10 days, including reduced glutathione at a dose of 250 mg/day and citicoline at a dose of 500 mg/day. All procedures complied with the Declaration of Helsinki, and the study received approval from the local ethics committee. All participants voluntarily signed informed consent.

Venous blood samples were collected from all participants in the morning, after an overnight fast, under standard conditions. The control group and POAG patients underwent baseline sampling at the beginning of the study. The second sampling was performed after 10 days only in the 15 patients who received pharmacological therapy. Peripheral blood was centrifuged according to standard protocols, and the resulting plasma was stored at –80 °C until analysis. Biomarker concentrations were determined using enzyme-linked immunosorbent assay (ELISA) with certified commercial kits. The markers measured included HSP70 (Heat Shock Protein 70) and Human GRIN2B (glutamate receptor, NR2B subunit). Results were expressed in ng/mL. All laboratory procedures were performed in accordance with Good Laboratory Practice (GLP).

Statistical analysis was conducted using Statistica 10.0 (Statsoft, USA). Descriptive statistics were presented as mean ± standard deviation (M ± SD). An independent samples t-test was used to compare the control group and POAG patients before treatment, while the paired t-test was used to evaluate changes after therapy. Diagnostic informativeness of HSP70 and GRIN2B was assessed through intergroup comparisons. Clinical significance of changes was evaluated using Cohen's d. Relationships between pre- and post-treatment values were analyzed using linear regression with calculation of β and R^2. The prognostic capability of the markers was assessed using ROC analysis, with calculation of AUC and optimal threshold based on Youden's index (sensitivity, specificity). A p-value < 0.05 was considered statistically significant.

Results

To assess the diagnostic informativeness of the HSP70 and GRIN2B markers, their levels were compared between the patient group before treatment and healthy individuals (Fig. 1).

The mean GRIN2B level in patients before treatment was 1.95 ng/mL, whereas in the control group it was 1.48 ng/mL. Although patients demonstrated a tendency toward higher GRIN2B values, the difference was not statistically significant ($p = 0.483$). In contrast to GRIN2B, HSP70 showed clearly pronounced differences. The mean HSP70 level in patients before treatment was 12.35 ng/mL, which significantly exceeded the control group value of 5.08 ng/mL, with a statistically significant difference ($p = 0.032$).

A total of 15 paired measurements of heat shock protein (HSP70) levels before and after treatment were analyzed. In most cases, post-

ISSN 2786-6661
eISSN 2786-667X

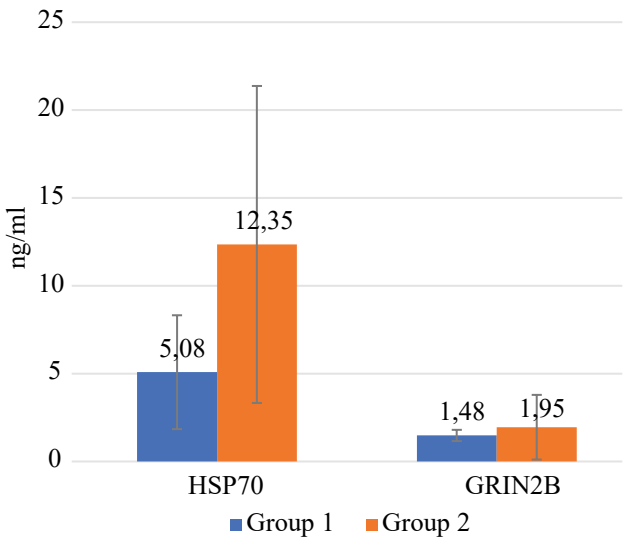

Fig. 1. Levels of HSP70 and GRIN2B in control group and patients with POAG

treatment HSP70 levels exceeded baseline values. The mean difference was 4.72 ± 16.74; however, no statistically significant changes were detected ($p = 0.29$). Cohen's d indicated a small effect size ($d = 0.28$), suggesting high inter-individual variability in treatment response. Linear regression demonstrated a weak and statistically non-significant association between baseline and post-treatment HSP70 levels ($\beta = 0.41$; $p = 0.13$; $R^2 = 0.17$), indicating limited prognostic value of this marker (Fig. 2).

The GRIN2B marker demonstrated a different pattern. In a subset of patients, GRIN2B decreased after treatment, which was interpreted as a positive therapeutic response. The mean difference was –0.02 ± 1.09, and Cohen's d was close to zero ($d = –0.02$), indicating an overall absence of a group-level effect.

Linear regression analysis for GRIN2B before and after treatment showed a weak and statistically non-significant association ($\beta = 0.05$; $R^2 = 0.01$), indicating that baseline levels did not predict post-treatment values (Fig. 2).

In the initial analysis, treatment effectiveness was evaluated based on an increase in HSP70 after therapy. The AUC was only 0.20, indicating no prognostic ability of HSP70 (Fig. 3). Even when using a ≥20% increase in HSP70 as the response criterion, diagnostic accuracy remained low.

After defining a positive response as a decrease in GRIN2B following treatment, ROC analysis demonstrated a substantial improvement in the prognostic ability of this marker. The AUC reached 0.812, indicating good diagnostic accuracy (Fig. 3).

Using Youden's index, the optimal pre-treatment GRIN2B cutoff was determined as 1.88. This threshold provided a sensitivity of 0.71 and a specificity of 1.00, making it a promising cutoff for patient stratification.

Discussion

In light of current evidence confirming the multifactorial nature of POAG — highlighting not only intraocular pressure but also vascular, metabolic, oxidative, and excitotoxic mechanisms — the search for circulating biomarkers capable of reflecting retinal ganglion cell neurodegeneration remains highly relevant. In particular, increased expression of

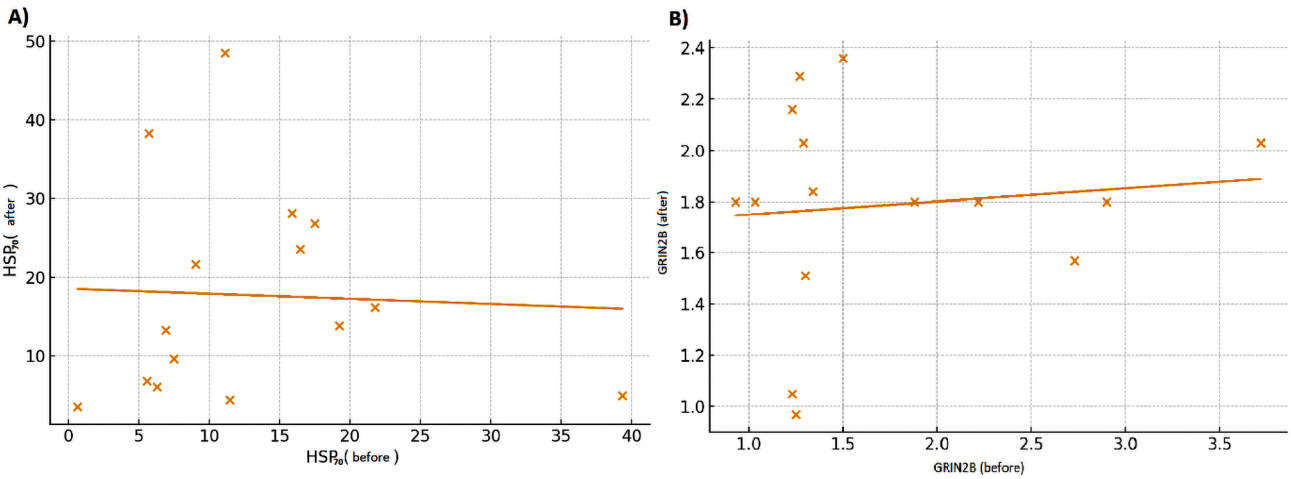

Fig. 2. Linear regression for (A) HSP70 and (B) GRIN2B before and after treatment

ISSN 2786-6661
eISSN 2786-667X

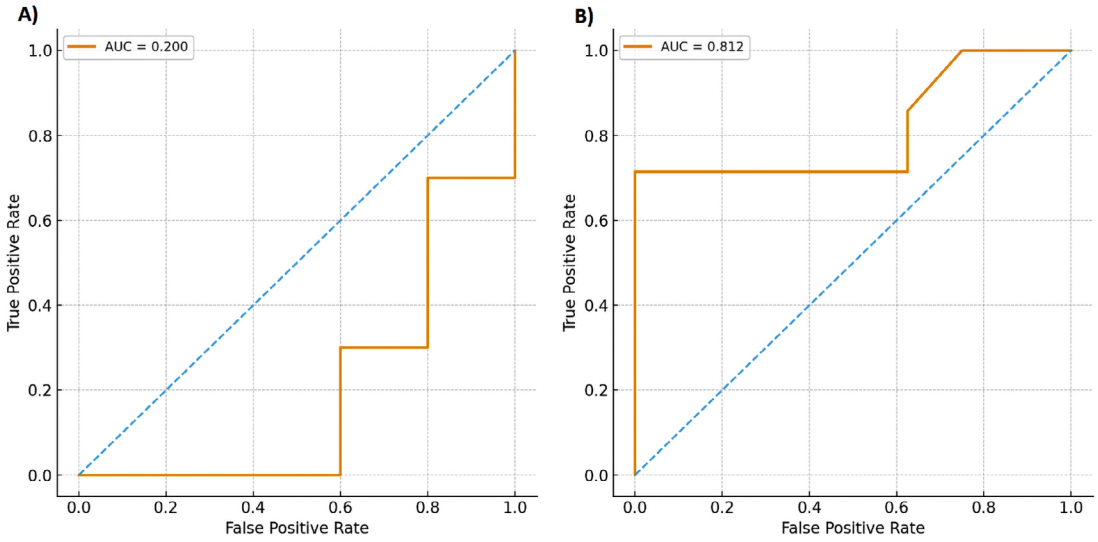

Fig 3. ROC analysis for (A) HSP70 and (B) GRIN2B.

HSP70 and the involvement of GRIN2B gene in the pathogenesis of neuronal injury provide a rational basis for our investigation. Notably, oxidative stress has been shown to play a key role in glaucomatous neuropathy through mitochondrial dysfunction and activation of reactive oxygen species [12, 13]. Additionally, NMDA-receptor activation and glutamate excess induce calcium-dependent retinal ganglion cell death [14].

In our study, we found a significant elevation of plasma HSP70 levels in POAG patients compared with healthy controls (12.35 ± 9.02 ng/mL vs. 5.08 ± 3.23 ng/mL; $p = 0.032$). This is consistent with evidence of enhanced cellular stress-response activation in glaucoma and a possible upregulation of endogenous neuroprotection via HSP70 [15]. Thus, HSP70 may serve as a potential marker of oxidative/stress-related injury within the visual pathway. However, our attempt to evaluate HSP70 dynamics after 10 days of treatment (glutathione + citicoline) revealed only a slight, statistically insignificant increase ($p = 0.29$; $d = 0.28$) and a weak prognostic association ($\beta = 0.41$; $R^2 = 0.17$). This indicates high inter-individual variability in treatment response and limited short-term prognostic value of HSP70.

Regarding the NR2/GRIN2B marker, although a tendency toward elevation before treatment was observed (1.95 ± 2.13 ng/mL in patients vs. 1.48 ± 0.35 ng/mL in controls; $p = 0.483$), the difference did not reach statistical significance. Following treatment, the mean change was minimal (–0.02 ± 1.09; $d = –0.02$), with no correlation between baseline and post-treatment levels ($\beta = 0.05$; $R^2 = 0.01$). However, a noteworthy finding emerged from the ROC analysis: AUC = 0.812, and the optimal cutoff of 1.88 ng/mL provided a sensitivity of 0.71 and a specificity of 1.00. This suggests that plasma NR2/GRIN2B may serve as a marker of therapeutic response or patient stratification, despite the absence of a statistically significant group shift. Given the limited number of studies assessing circulating NR2/GRIN2B biomarkers in glaucoma, our findings address an important knowledge gap.

When comparing our results with existing literature, it should be noted that most studies focus on oxidative stress while few investigate circulating protein biomarkers of neurodegeneration in glaucoma. For example, a recent review emphasized oxidative stress as a central mechanism of glaucomatous neuropathy and a potential therapeutic target [16]. Meanwhile, the role of NMDA-mediated excitotoxicity in glaucoma is discussed mainly in experimental models, and clinical data on NR2B protein or its autoantibodies remain fragmented. Our findings support the concept that NR2/GRIN2B may be a relevant biomarker — although larger studies with longer therapeutic interventions and inclusion of clinical endpoints are required.

ISSN 2786-6661
eISSN 2786-667X

From a practical perspective, the results obtained have several implications. First, elevated HSP70 in POAG reinforces the rationale for incorporating antioxidant and neuroprotective strategies alongside intraocular pressure control. Second, NR2/GRIN2B may serve as a tool for patient stratification or monitoring treatment response to neuroprotective agents, though its clinical use requires further validation. At the same time, the short 10-day treatment period did not allow for conclusive HSP70 changes, indicating the need for longer observation and larger samples.

In summary, our study demonstrates that HSP70 and NR2/GRIN2B hold potential as circulating biomarkers in POAG: HSP70 as an indicator of stress-related neuroinjury, and NR2/GRIN2B as a possible marker of therapeutic response. However, further research involving larger cohorts and longer treatment durations is necessary before their implementation in clinical practice.

Conclusions

In patients with primary open-angle glaucoma, a significantly elevated level of HSP70 was detected compared with healthy individuals, confirming its role as a marker of oxidative stress and neuronal injury. In contrast, GRIN2B/NR2 did not demonstrate statistically significant intergroup differences but showed good prognostic capability in the ROC analysis for identifying patients who may potentially respond to neuroprotective therapy. The short 10-day course of glutathione and citicoline treatment did not produce significant changes in the levels of the studied biomarkers, which may indicate the need for a longer therapeutic intervention or larger sample sizes to detect meaningful effects. The results obtained highlight the potential of HSP70 as a diagnostic indicator and GRIN2B as a marker of therapeutic response, emphasizing the need for further studies to clarify their clinical significance in the management of glaucomatous neuropathy.

Financing

This study did not receive external funding.

Conflict of interest

The authors declare that there is no conflict of interest and no financial interest in the preparation of this article.

Consent to publication

All authors have read and approved the final version of the manuscript. All authors have agreed to publish this manuscript. AI tools were not used in preparing this manuscript.

Ethical Considerations

Ethical approval №9 from 09.11.2020

Author Contributions (CRediT taxonomy)

Conceptualization: Nedilka Taras (ORCID: 0009-0003-9185-561X);
Methodology: Lutsenko Nina (ORCID: 0000-0001-9433-7568);
Software: Nedilka Taras;
Validation: Lutsenko Nina;
Formal analysis: Nedilka Taras;
Investigation: Nedilka Taras;
Resources: Nedilka Taras;
Data curation: Nedilka Taras;
Writing – original draft: Nedilka Taras;
Writing – review & editing: Lutsenko Nina;
Visualization: Nedilka Taras;
Supervision: Lutsenko Nina;
Project administration: Lutsenko Nina;
Funding acquisition: Nedilka Taras.

REFERENCES

1. Wang X, Wang M, Liu H, Mercieca K, Prinz J, Feng Y, Prokosch V. The Association between Vascular Abnormalities and Glaucoma-What Comes First? Int J Mol Sci. 2023 Aug 25;24(17):13211. DOI 10.3390/ijms241713211.

2. Kang EY, Liu PK, Wen YT, Quinn PMJ, Levi SR, Wang NK, Tsai RK. Role of Oxidative Stress in Ocular Diseases Associated with Retinal Ganglion Cells Degeneration. Antioxidants (Basel). 2021 Dec 5;10(12):1948. DOI 10.3390/antiox10121948.

3. Benoist d'Azy C, Pereira B, Chiambaretta F, Dutheil F. Oxidative and Anti-Oxidative Stress Markers in Chronic Glaucoma: A Systematic Review and Meta-Analysis. PLoS One. 2016 Dec 1;11(12):e0166915. DOI 10.1371/journal.pone.0166915

4. Seven E, Tekin S. Comparative Analysis of Oxidative Stress Biomarkers in 50 Primary Open-Angle Glaucoma Patients. Med Sci Monit. 2025 Jun 21;31:e948665. DOI 10.12659/MSM.948665.

ISSN 2786-6661
eISSN 2786-667X

5. Modrzejewska M, Zdanowska O. The Role of Heat Shock Protein 70 (HSP70) in the Pathogenesis of Ocular Diseases-Current Literature Review. J Clin Med. 2024 Jun 30;13(13):3851. DOI 10.3390/jcm13133851.

6. Zhang L, Xue K, Fan P, Chen C, Hu J, Huang J, Lu W, Xu J, Xu S, Ran J, Zhu S, Gan S. Geranylgeranylacetone-induced heat shock protein70 expression reduces retinal ischemia-reperfusion injury through PI3K/AKT/mTOR signaling. Exp Eye Res. 2023 Apr;229:109416. DOI 10.1016/j.exer.2023.109416.

7. Nowak A, Rozpędek W, Cuchra M, Wojtczak R, Siwak M, Szymanek K, Szaflik M, Szaflik J, Szaflik J, Majsterek I. Association of the expression level of the neurodegeneration-related proteins with the risk of development and progression of primary open-angle glaucoma. Acta Ophthalmol. 2018 Feb;96(1):e97-e98. DOI 10.1111/aos.13479.

8. Xie C, Kessi M, Liu F, He F, Peng J. GRIN2B-related neurodevelopmental disorders: genotype-phenotype correlations and therapeutic implications. Orphanet J Rare Dis. 2025 Oct 27;20(1):541. DOI 10.1186/s13023-025-04055-x.

9. di Biase L, Bonura A, Pecoraro PM, Carbone SP, Di Lazzaro V. Unlocking the Potential of Stroke Blood Biomarkers: Early Diagnosis, Ischemic vs. Haemorrhagic Differentiation and Haemorrhagic Transformation Risk: A Comprehensive Review. Int J Mol Sci. 2023 Jul 17;24(14):11545. DOI 10.3390/ijms241411545.

10. Chum PP, Bishara MA, Solis SR, Behringer EJ. Cerebrovascular miRNAs Track Early Development of Alzheimer's Disease and Target Molecular Markers of Angiogenesis and Blood Flow Regulation. J Alzheimers Dis. 2024;99(s2):S187-S234. DOI 10.3233/JAD-230300.

11. Rossi GCM, Rolle T, De Silvestri A, Sisto D, Mavilio A, Mirabile AV, Paviglianiti A, Strano B, Picasso E, Pasinetti GM, Milano G, Ruberto G. Multicenter, Prospective, Randomized, Single Blind, Cross-Over Study on the Effect of a Fixed Combination of Citicoline 500 mg Plus Homotaurine 50 mg on Pattern Electroretinogram (PERG) in Patients With Open Angle Glaucoma on Well Controlled Intraocular Pressure. Front Med (Lausanne). 2022 Apr 29;9:882335. DOI 10.3389/fmed.2022.882335.

12. Kimura A, Namekata K, Guo X, Noro T, Harada C, Harada T. Targeting Oxidative Stress for Treatment of Glaucoma and Optic Neuritis. Oxid Med Cell Longev. 2017;2017:2817252. DOI 10.1155/2017/2817252.

13. Trivli A, Koliarakis I, Terzidou C, Goulielmos GN, Siganos CS, Spandidos DA, Dalianis G, Detorakis ET. Normal-tension glaucoma: Pathogenesis and genetics. Exp Ther Med. 2019 Jan;17(1):563-574. DOI 10.3892/etm.2018.7011.

14. Ding RF, Yu Q, Liu K, Du J, Yin HJ, Ji ZL. Gene network analyses unveil possible molecular basis underlying drug-induced glaucoma. BMC Med Genomics. 2021 Apr 19;14(1):109. DOI 10.1186/s12920-021-00960-9.

15. Tsai T, Grotegut P, Reinehr S, Joachim SC. Role of Heat Shock Proteins in Glaucoma. Int J Mol Sci. 2019 Oct 18;20(20):5160. DOI 10.3390/ijms20205160.

16. Cáceres-Vélez PR, Hui F, Hercus J, Bui B, Jusuf PR. Restoring the oxidative balance in age-related diseases - An approach in glaucoma. Ageing Res Rev. 2022 Mar;75:101572. DOI 10.1016/j.arr.2022.101572.

Діагностичний та прогностичний потенціал циркулюючих HSP70 та GRIN2B при первинній відкритокутовій глаукомі

Тарас Неділька[1], Ніна Луценко[1,2]

[1] Комунальне некомерційне підприємство «Запорізька обласна клінічна лікарня» Запорізької обласної ради, м. Запоріжжя, Україна

[2] Запорізький державний медико-фармацевтичний університет, Навчально-науковий інститут післядипломної освіти, м. Запоріжжя, Україна

Address for correspondence:

Taras Nedilka

E-mail: hardcoresunlight@gmail.com

Анотація: *Первинна відкритокутова глаукома дедалі частіше розглядається як мультифакторне нейродегенеративне захворювання, прогресування якого зумовлене не лише підвищеним внутрішньоочним тиском, а й порушеннями судинної регуляції, метаболічним дисбалансом, оксидативним стресом та глутамат-опосередкованою ексайтотоксичністю. Ці механізми формують потребу у виявленні циркулюючих біомаркерів, здатних відображати*

ISSN 2786-6661
eISSN 2786-667X

раннє та поточне ураження гангліозних клітин сітківки. HSP70 вважають індикатором клітинного стресу та потенційним медіатором ендогенного нейропротекційного захисту, тоді як субодиниця рецептора глутамату NR2B, кодована геном GRIN2B, бере участь в ексайтотоксичному ушкодженні нейронів. У цьому дослідженні було проаналізовано концентрації цих двох маркерів у плазмі крові в осіб з первинною відкритокутовою глаукомою та у здорових учасників, а також їхню динаміку після короткого курсу нейропротективної терапії, що включала відновлений глутатіон і цитиколін. Отримані результати показали значно вищий рівень HSP70 у пацієнтів порівняно зі здоровими особами, що підтверджує його роль як маркера оксидативного та стрес-асоційованого нейронального ушкодження. На відміну від цього, рівень маркера GRIN2B достовірно не відрізнявся між групами до лікування, хоча спостерігалася тенденція до вищих значень у пацієнтів. Після десятиденного комбінованого лікування жоден із маркерів не продемонстрував статистично значущої зміни на груповому рівні, що може відображати значну міжіндивідуальну варіабельність, недостатню тривалість терапії або потребу у збільшенні вибірки для виявлення тонких нейробіологічних ефектів. Незважаючи на відсутність суттєвої різниці у вихідних значеннях, маркер GRIN2B продемонстрував високу прогностичну здатність щодо ідентифікації осіб, які показали сприятливу біохімічну відповідь на лікування, що свідчить про його потенційну користь для стратифікації пацієнтів і моніторингу ранніх терапевтичних ефектів. HSP70 продемонстрував обмежену прогностичну цінність у короткий терапевтичний інтервал, що може бути зумовлено складністю його регуляції та необхідністю тривалішого періоду спостереження. Загалом результати свідчать, що ці циркулюючі білки можуть доповнювати наявні клінічні інструменти для оцінки механізмів розвитку первинної відкритокутової глаукоми. HSP70 може слугувати індикатором оксидативного стресу та ушкодження гангліозних клітин сітківки, тоді як маркер GRIN2B може мати перспективу як ранній індикатор відповіді на лікування. Подальші дослідження необхідні для визначення їх рівнів в динаміці, значення на різних стадіях захворювання та можливості інтеграції у процес клінічного прийняття рішень.

Ключові слова: Апоптоз, Глаукома, Рецептор Глутамату, Дегенерація, HSP70 Білки теплового шоку, Нейропротекція, Оксидативний стрес.

Copyright: © 2025 by the authors; licensee USMYJ, Kyiv, Ukraine. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0/).