Waheeda-Azwa H[1,2,*], Wan Julyatee WY[1,2], Nor Abibas CR[1,2] Shahidatul-Adha M[1,2], Wan-Hazabbah WH[1,2], Aiman-Mardhiyyah MY[1,2,*]

[1]Department of Ophthalmology and Visual Sciences, School of Medical Sciences, Universiti Sains Malaysia, 16150, Kubang Kerian, Kelantan, Malaysia

[2]Hospital Pakar Universiti Sains Malaysia, 16150, Kubang Kerian, Kelantan, Malaysia

Received 24 October 2025
Revised 28 January 2026
Accepted 8 February 2026
Published Online
1 March 2026

*Corresponding author
Waheeda Azwa Hussein
waheedaazwa@usm.my
Aiman Mardhiyyah Mohd Yazid
dhiyazid@usm.my

Management of Vertical Diplopia Secondary to Inferior Rectus Paresis Following Traumatic Brain Injury Using Prism Correction: A Case Report

Abstract – Traumatic brain injury (TBI) is an acquired brain insult caused by external mechanical forces and may result in a wide range of neurological and ophthalmic sequelae. Diplopia is a common but often under-recognized complication that can significantly impair functional ability and quality of life. We report the case of a previously healthy 21-year-old male who developed persistent binocular vertical diplopia following severe TBI sustained in a motor vehicle accident. Ophthalmic evaluation revealed right hypertropia measuring 10 prism diopters (Δ) base down with a small 2Δ base-in exotropia, consistent with right inferior rectus paresis. Prism adaptation testing demonstrated optimal symptomatic relief with 4Δ base-down prisms in both eyes. The patient was managed conservatively with ground-in prism glasses and followed up over a two-month period. At follow-up, best-aided visual acuity improved to 6/7.5 in the right eye and 6/9 in the left eye, with complete resolution of diplopia in primary gaze and daily activities. Surgical intervention was offered but declined due to satisfactory symptom control with prism correction. This case highlights the effectiveness of prism glasses as a safe, non-invasive treatment option for post-traumatic vertical diplopia and underscores the importance of early ophthalmic assessment in patients with TBI to optimize functional recovery and quality of life.

Keywords*: Traumatic brain injury, prism glasses, vertical diplopia*

1 INTRODUCTION

Traumatic brain injury (TBI) represents a significant challenge in modern medicine and is defined as an alteration in brain function or other evidence of brain pathology caused by an external mechanical force (1,2). The clinical consequences of TBI range from transient neurological impairment to permanent disability, with many patients experiencing persistent cognitive, emotional, and sensory sequelae long after the initial injury (3,4). Visual dysfunction is a well-recognized but often underappreciated complication of TBI and can substantially impair daily functioning and quality of life (5,6,7).

Among the spectrum of post-traumatic visual disturbances, ocular motility disorders are particularly common and frequently present as diplopia. These disorders may arise from damage to the extraocular muscles, disruption of neuromuscular transmission, or injury to the cranial nerves responsible for eye movements. Cranial nerves III (oculomotor), IV (trochlear), and VI (abducens) are especially vulnerable to traumatic injury due to their long intracranial courses, proximity to rigid bony and dural structures, and susceptibility to acceleration–deceleration and rotational shear forces (7,9). Such forces may result in nerve stretching, ischemia, contusion, or focal brainstem injury, leading to paresis or paralysis of the innervated extraocular muscles.

The trochlear nerve (cranial nerve IV) is particularly susceptible to trauma because of its thin calibre, long intracranial course, and dorsal exit from the brainstem, making it the most commonly affected cranial nerve in traumatic ocular motility disorders (7,13). Injury to the oculomotor nerve or its fascicles can similarly produce selective weakness of individual extraocular muscles, resulting in complex patterns of vertical or combined ocular misalignment. In addition to direct neural injury, secondary mechanisms such as orbital trauma, microvascular compromise, and disruption of supranuclear ocular motor pathways may further contribute to post-traumatic diplopia (5,7).

Vertical diplopia presents a distinct diagnostic challenge, as its differential diagnosis includes cranial nerve palsies, orbital restrictive disease, skew deviation, myasthenia gravis, thyroid eye disease, and decompensated pre-existing strabismus (12,13). Accurate diagnosis requires careful clinical evaluation, detailed ocular motility assessment, prism measurements, and appropriate neuroimaging or laboratory investigations to exclude alternative or coexisting etiologies.

Management of post-traumatic diplopia is individualized and depends on the underlying cause, severity, and stability of ocular misalignment. While surgical intervention may be indicated in selected cases, conservative approaches such as prism correction play an important role, particularly in patients with stable deviations or those who decline surgery. Prism glasses realign the visual axes by altering the path of incoming light and have been shown to effectively reduce diplopia and improve functional outcomes in patients with TBI-related ocular motility disorders (8,9,10,11). Early recognition and targeted ophthalmic intervention are therefore essential to optimize visual rehabilitation and overall quality of life following TBI.

2. CASE REPORT

A 21-year-old Malay male with no prior ocular or systemic illness presented with persistent binocular vertical diplopia following a severe traumatic brain injury sustained in a motor vehicle accident in June 2022. The patient experienced loss of consciousness at the scene and required endotracheal intubation for airway protection. He was admitted for two weeks to a district hospital, where he was managed conservatively, before being referred to Hospital Pakar Universiti Sains Malaysia, a tertiary care center, for further ophthalmic evaluation.

At his initial ophthalmology clinic visit, approximately two months post-injury, the patient reported constant vertical diplopia that was most noticeable in primary gaze and interfered significantly with daily activities, including reading, driving, and academic work. The diplopia resolved with monocular occlusion, confirming its binocular nature. The patient expressed significant distress due to visual symptoms and concern about his ability to continue university studies.

A comprehensive ophthalmic assessment was performed at the Ophthalmology Clinic, Hospital Pakar Universiti Sains Malaysia, with findings summarized in Table I. At the first visit, unaided visual acuity (VA) was 6/120 pinhole (PH) 6/18 in the right eye (RE) and 6/120 PH 6/18 in the left eye (LE). Best-corrected VA improved to 6/18 PH 6/12 (RE) and 6/15 PH 6/9 (LE) with a spectacle correction of −3.50/−0.50 × 170 in both eyes. Pupillary examination revealed no relative afferent pupillary defect. Anterior segment and dilated fundus examinations were unremarkable bilaterally.

Table 1. Assessment of first visit

Assessment	Right Eye	Left Eye
Visual Acuity (unaided)	6/120 PH 6/18	6/120 PH 6/18
Visual Acuity (aided)	6/18 PH 6/12	6/15 PH 6/9
Spectacle Power	-3.50/-0.50 x 170	-3.50/-0.50 x 170
Pupil	RAPD negative	
EOM	Right levoduction -1	
Hirschberg	Right Eye hypertropia	
Cover Test	Right Eye hypertropia with small exotropia	
Hess Test	Right Eye Superior Oblique overaction	
Diplopia Test	Diplopia at primary gaze, elevation and depression	
Slit Lamp examination	Anterior and Posterior segment normal	
Motor examination	Power 5/5 Normal tone	

Extraocular motility assessment demonstrated a mild limitation of right eye levoduction (−1). Hirschberg and cover testing revealed a right hypertropia associated with a small exotropia. Diplopia was elicited in primary gaze, elevation, and depression. Hess charting demonstrated apparent superior oblique overaction; however, this finding was interpreted cautiously in the context of post-traumatic ocular misalignment. The initial Hess chart findings are shown in Figure 1A. Neurological motor examination showed normal tone and full power (5/5) in all limbs.

At presentation, a provisional diagnosis of left fourth cranial nerve palsy was considered based on the presence of vertical diplopia following

trauma. This initial impression was later revised following detailed ocular motility assessment, prism measurements, and follow-up evaluation, which demonstrated findings more consistent with right inferior rectus paresis.

Prism assessment revealed a vertical deviation measuring 10 prism diopters (Δ) base-down hypertropia with a small 2Δ base-in exotropia. No clinical signs suggestive of restrictive or mechanical pathology, such as orbital floor entrapment, were present. There was no variability of symptoms or fatigability to suggest myasthenia gravis. Relevant imaging findings were reviewed and are described in the Case Report. Imaging did not show orbital fractures, extraocular muscle entrapment, or compressive intracranial lesions, helping to exclude mechanical or structural causes of the vertical diplopia. As no abnormal findings were identified, representative images were not included.

Prism adaptation testing was performed to determine the optimal prismatic correction (Table II). Initial refraction at this stage was −4.00/−0.50 × 180 (RE) with VA of 6/12, and −3.75/−0.75 × 180 (LE) with VA of 6/9. Across four sequential trials conducted at 15-minute intervals, the best symptomatic relief and optimal visual acuity were achieved with 4Δ base-down prisms in both eyes. This prescription was finalized as −4.00/−0.50 × 180 (6/15) with 4Δ base-down for the right eye and −3.75/−0.75 × 180 (6/12) with 4Δ base-down for the left eye, providing near VA of N5 at 40 cm.

Table 2. Prism adaptation tests

Assessments	Right Eye	Left Eye
Refraction	-4.00/ -0.50 X 180 (6/12)	-3.75/ -0.75 X 180 (6/9)
Prism Test	Left Eye 10Δ BD Hypertropia, 2Δ BI Exotropia	
Prism Adaptation Test (First visit)	First trial	
	4Δ BD VA 6/18	4Δ BD + 2Δ BI VA 6/15
	Near VA Both Eye N5 30 cm	
	Second trial	
	4Δ BD VA 6/12	4Δ BD + 2Δ BI VA 6/12
	Near VA BE N5 30 cm	
	Third trial	
	4Δ BD VA 6/12	2Δ BD VA 6/12
	Near VA BE N5 30 cm	
	Fourth trial	
	4Δ BD VA 6/12	4Δ BD VA 6/12
Prism Glass Prescription	4.00 / -0.50 X 180 (6/15) 4Δ BD	-3.75 / -0.75 X 180 (6/12) 4Δ BD
	Near VA Both Eye N5 40 cm	

VA= Visual Acuity, BD= Base Down, BI= Base In, Δ = Prism

At the two-month follow-up, the patient reported minimal residual diplopia and was able to drive both a car and a motorcycle without difficulty. Repeat Hess charting at follow-up demonstrated improved symmetry (Figure 1B). Best-aided VA improved to 6/12 PH 6/7.5 in the right eye and 6/12 PH 6/9 in the left eye. Subjective refraction yielded −4.00/−0.50 × 180 (6/9−2) in the right eye and −4.25/−1.25 × 170 (6/9−2) in the left eye, with stable prismatic correction of 4Δ base-down in both eyes.

Park's three-step test was performed during follow-up but yielded inconclusive results (Table III). While this did not definitively exclude trochlear nerve palsy, the overall clinical findings including prism measurements, absence of gaze-dependent worsening typical of fourth cranial nerve palsy, lack of significant torsional symptoms, and stable response to prism correction, favoured a diagnosis of right hypertropia secondary to right inferior rectus paresis.

Surgical management in the form of a right superior rectus Faden procedure was discussed with the patient as a potential option should symptoms persist or worsen. However, given the satisfactory resolution of diplopia with prism correction and the patient's preference to avoid surgery, conservative management with prism glasses was continued.

3. DISCUSSION

Vertical diplopia is a recognized and functionally disabling visual sequela of traumatic brain injury (TBI), frequently arising from disruption of ocular motor control involving the extraocular muscles, their innervating cranial nerves, or supranuclear pathways (12,13). This case highlights the diagnostic complexity of post-traumatic vertical diplopia and emphasizes the importance of careful clinical reasoning when differentiating inferior rectus paresis from fourth cranial nerve palsy, two conditions with overlapping but distinct clinical characteristics.

Trauma is the most common acquired cause of trochlear nerve palsy due to the nerve's long intracranial course, dorsal exit from the brainstem, and susceptibility to acceleration–deceleration and rotational shear forces (13). Clinically, fourth cranial nerve palsy is characterized by hypertropia that worsens in contralateral gaze and ipsilateral head tilt, often accompanied by torsional diplopia and compensatory head posture, particularly during downgaze activities such as reading or descending stairs (12,13).

In contrast, inferior rectus paresis typically produces vertical misalignment that may be most apparent in primary gaze or downgaze, without prominent torsional symptoms.

Table 3. Assessment of second visit

Assessments	Right Eye	Left Eye
Symptom	Patient reported minimal diplopia and was able to drive his car and motorcycle comfortably	
VA Aided	6/12 PH 6/7.5	6/12 PH 6/9
Subjective Refraction	-4.00/ -0.50 x 180 ($6/9^{-2}$)	-4.25/ -1.25 x 170 ($6/9^{-2}$)
Prism Assessment:	4Δ BD	4Δ BD
Park 3 step	Inconclusive	
Diagnosis	Right hypertropia secondary to Right inferior rectus paresis	
Management	Offer for Right Eye superior rectus Faden procedure but patient was not keen for an operation. He is comfortable with his current glasses.	

VA= Visual Acuity

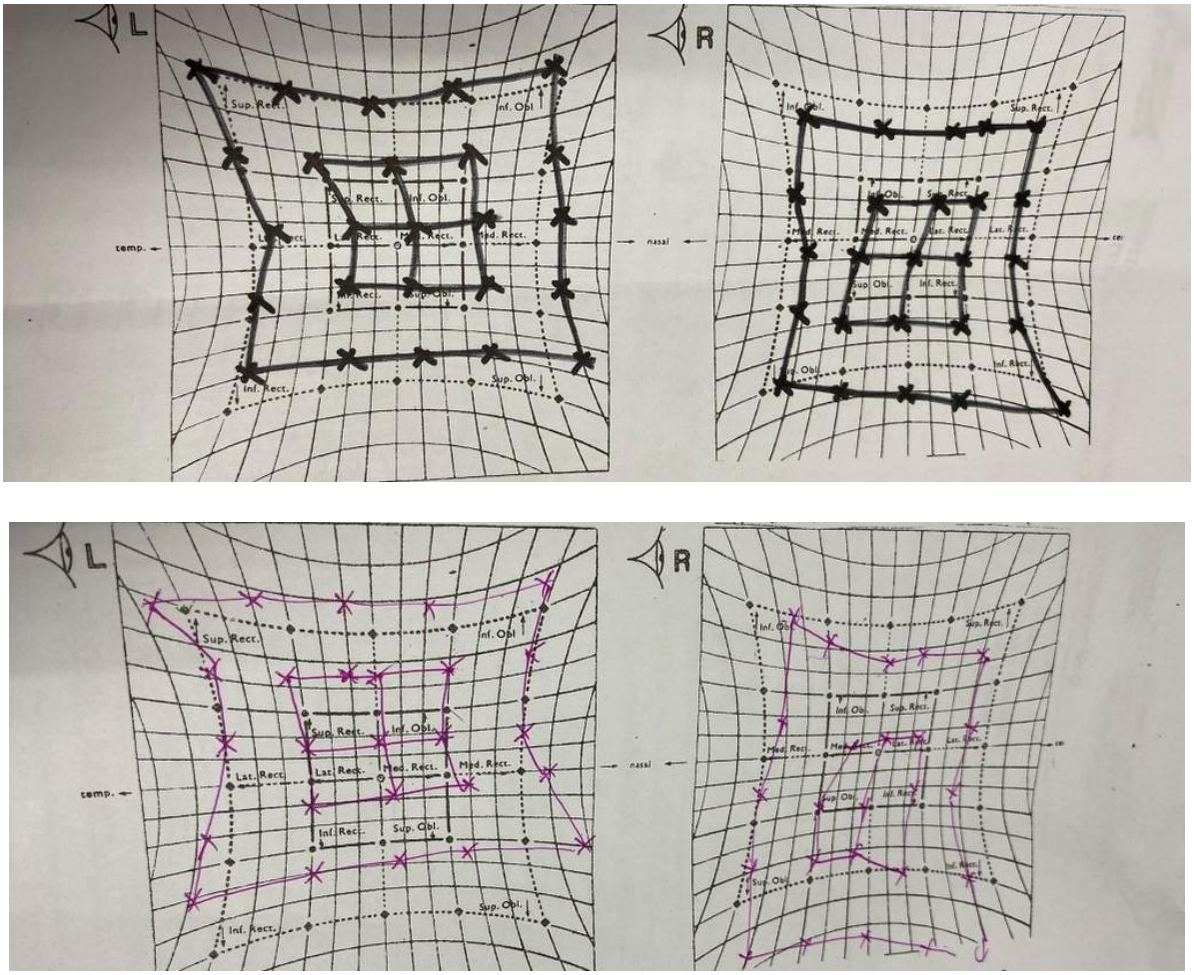

Figure 1. Hess chart findings before and after treatment.
(A) Initial Hess chart demonstrating asymmetry with reduced excursion in downgaze, consistent with weakness of the right inferior rectus muscle.
(B) Follow-up Hess chart showing improved symmetry and expansion of the affected field, indicating functional improvement following prism correction.

In the present case, an initial provisional diagnosis of fourth cranial nerve palsy was reasonable given the history of severe TBI and vertical diplopia. However, several clinical features prompted diagnostic reconsideration. The diplopia was most symptomatic in primary gaze rather than downgaze, torsional symptoms were absent, and no consistent compensatory head tilt was observed, features atypical for isolated trochlear nerve palsy. These findings necessitated a broader diagnostic evaluation.

Park's three-step test was performed but yielded inconclusive results. Although this test is a useful clinical tool for localizing paretic extraocular muscles, it has recognized limitations, particularly in post-traumatic settings where partial involvement of multiple muscles or supranuclear pathways may coexist (12). An inconclusive Park's three-step test does not definitively exclude fourth cranial nerve palsy and should be interpreted in conjunction with other clinical findings. In this case, the overall pattern of ocular misalignment, prism measurements, and symptom distribution favoured inferior rectus paresis as the more likely diagnosis.

The diagnosis of right inferior rectus paresis was further supported by the pattern of hypertropia, stable prism measurements, and the patient's consistent response to prism adaptation testing. Mechanical or restrictive causes, such as orbital floor fracture with inferior rectus entrapment, were considered but deemed unlikely due to the absence of downgaze restriction, globe retraction, or radiological evidence of orbital trauma. Additionally, the lack of symptom variability or fatigability made myasthenia gravis improbable (12,13). Neuroimaging did not demonstrate orbital fractures or compressive lesions, further supporting a neurogenic aetiology.

Selective involvement of the inferior rectus muscle following TBI, with preservation of other cranial nerve functions, is uncommon but possible. Traumatic forces may result in focal ischemia, contusion, or stretch injury affecting specific oculomotor nerve fascicles or their peripheral branches, or disruption of supranuclear vertical gaze pathways (13). Although the precise mechanism cannot be definitively established in this case, the clinical presentation and course are consistent with localized inferior rectus dysfunction rather than generalized oculomotor nerve involvement.

Management of post-traumatic vertical diplopia must be individualized based on the severity, stability, and functional impact of ocular misalignment. While surgical intervention may be indicated for stable deviations persisting beyond 6–12 months or for cases refractory to conservative measures, prism correction represents an effective first-line, non-invasive option in selected patients (14,15). Prism lenses realign the visual axes by altering the path of incoming light and have been shown to significantly reduce diplopia and improve functional outcomes in patients with TBI-related ocular motility disorders (14,15).

In this patient, prism adaptation testing identified 4Δ base-down prisms in both eyes as the optimal correction, resulting in complete resolution of diplopia in primary gaze and daily activities. The patient reported meaningful functional improvement, including the ability to resume driving and academic activities, reflecting an improvement in quality of life. Although prism correction does not address the underlying muscle weakness and may be inconvenient during activities without spectacles such as showering or upon waking, the patient expressed high satisfaction with this conservative approach and declined surgical intervention.

The novelty of this case lies in the successful non-surgical management of inferior rectus paresis following TBI using prism correction alone. Reports specifically describing inferior rectus involvement after traumatic brain injury remain limited. This case therefore contributes to the existing literature by demonstrating that careful diagnostic evaluation and patient-centered conservative management can achieve excellent functional outcomes in selected patients (16).

4 CONCLUSION

Vertical diplopia following traumatic brain injury presents significant diagnostic and management challenges. This case highlights inferior rectus paresis as an important differential diagnosis in post-traumatic vertical misalignment, particularly when clinical features are atypical and Park's three-step test is inconclusive. Careful clinical evaluation and interpretation of ocular motility findings are essential for accurate diagnosis.

Prism correction provided effective, non-invasive management in this patient, resulting in complete resolution of diplopia and meaningful functional improvement. This case supports prism therapy as a valuable first-line treatment option for selected patients with post-traumatic vertical diplopia and underscores the importance of

individualized, patient-centered care in optimizing visual and quality-of-life outcomes.

ACKNOWLEDGEMENTS

The authors wish to express their sincere appreciation to the staffs of the Department of Ophthalmology and Visual Sciences, School of Medical Sciences and Ophthalmology Clinic, Hospital Pakar Universiti Sains Malaysia (HPUSM), for their invaluable contributions to the clinical assessment, management, and follow-up of this patient. We are also grateful to the hospital administration for facilitating access to clinical facilities and resources. Finally, we extend our gratitude to the patient for his cooperation and for granting permission to share his case for educational and research purposes.

AUTHOR CONTRIBUTIONS

WAH: investigation, writing-original draft; WJWY, NACR, SAM: investigation; WHWH: Supervision, Conceptualization; AMMY: investigation, writing-review and editing.

AUTHORSHIP CLARIFICATION

All authors meet the ICMJE criteria for authorship and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethics approval was not required for this case report. Written informed consent was obtained from the patient.

CONSENT FOR PUBLICATION

Not applicable

CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

REFERENCES

[1] Maas AIR, Menon DK, Adelson PD, Andelic N, Bell MJ, et al. Traumatic brain injury: integrated approaches to improve prevention, clinical care, and research. Lancet Neurol. 2017;16(12):987–1048. doi:10.1016/S1474-4422(17)30371-X

[2] Sen N. An insight into the vision impairment following traumatic brain injury. Neurochem Int. 2017;111:103–107. doi:10.1016/j.neuint.2017.01.019

[3] Konrad C, Geburek AJ, Rist F, Blumenroth H, Fischer B, et al. Long-term cognitive and emotional consequences of mild traumatic brain injury. Psychol Med. 2011;41(6):1197–1211. doi:10.1017/S0033291710001728

[4] Johnson VE, Stewart W, Smith DH. Traumatic brain injury and amyloid-β pathology: a link to Alzheimer's disease? Nat Rev Neurosci. 2010;11(5):361–370. doi:10.1038/nrn2808

[5] Kapoor N, Ciuffreda KJ. Vision disturbances following traumatic brain injury. Curr Treat Options Neurol. 2002;4(4):271–280. doi:10.1007/s11940-002-0027-z

[6] Capó-Aponte JE, Urosevich TG, Temme LA, Tarbett AK, Sanghera NK. Visual dysfunctions and symptoms during the subacute stage of blast-induced mild traumatic brain injury. Mil Med. 2012;177(7):804–813. doi:10.7205/MILMED-D-12-00061

[7] Rauchman SH, Zubair A, Jacob B, Rauchman D, Pinkhasov A. Traumatic brain injury: mechanisms, manifestations, and visual sequelae. Front Neurosci. 2023;17:1090672. doi:10.3389/fnins.2023.1090672

[8] Anilkumar SE, Narendran K. Prisms in the treatment of diplopia with strabismus of various etiologies. Indian J Ophthalmol. 2022;70(2):609–612. doi:10.4103/ijo.IJO_939_21

[9] Barton JJS, Ranalli PJ. Vision therapy: occlusion, prisms, filters, and vestibular exercises for mild traumatic brain injury. Surv Ophthalmol. 2021;66(3):346–353. doi:10.1016/j.survophthal.2020.08.001

[10] Fox SM, Koons P, Dang SH. Vision rehabilitation after traumatic brain injury. Phys Med Rehabil Clin N Am. 2019;30(1):171–188. doi:10.1016/j.pmr.2018.09.001

[11] Ciuffreda KJ, Ludlam DP, Yadav NK, Thiagarajan P. Traumatic brain injury. Adv Ophthalmol Optom. 2016;1(1):307–333. doi:10.4172/2155-9562.1000401

[12] Doble JE, Feinberg DL, Rosner MS, Rosner AJ. Identification of binocular vision dysfunction (vertical heterophoria) in traumatic brain injury patients and effects of individualized prismatic spectacle lenses in the treatment of postconcussive symptoms: a retrospective analysis. PM R. 2010;2(4):244–253. doi:10.1016/j.pmrj.2010.01.011

[13] Jain S. Diplopia: diagnosis and management. Clin Med (Lond). 2022;22(2):104–106. doi:10.7861/clinmed.2022-0045

[14] Portela-Camino JA, Sanchez I, Gutierrez C, Martín-González S. Successful treatment of diplopia using prism correction combined with vision therapy/orthoptics improves health-related quality of life. PeerJ. 2024;12:e17315. doi:10.7717/peerj.17315

[15] Kapoor N, Ciuffreda KJ, Han Y. Oculomotor rehabilitation in acquired brain injury: a case series. Arch Phys Med Rehabil. 2004;85(10):1667–1678. doi:10.1016/j.apmr.2003.12.044

[16] Ponsford J, Sloan S, Snow P. Traumatic brain injury: rehabilitation for everyday adaptive living. 2nd ed. London: Psychology Press; 2012.