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Spasm of the Near Reflex After Traumatic Brain Injury: A Case Report

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Abstract

Spasm of the near reflex (SNR) is an uncommon disorder in which accommodation, convergence, and miosis are inappropriately sustained, and it may mimic neurological conditions. We report the case of a 14-year-old girl who developed diplopia at near, reduced near visual acuity, a large-angle esotropia, and a bilateral abduction deficit after a frontal traumatic brain injury (TBI), initially suggesting bilateral abducens nerve palsy. Neuroimaging and neurological evaluation were unremarkable. Monocular ductions were full, and the oculocephalic reflex was normal, which excluded a sixth nerve lesion. Autorefraction showed high myopia (approximately -8.00 D), which shifted to +2.00 D after cycloplegia. The esotropia and abduction deficit persisted under cyclopentolate and after one week of atropine. With +5.00 D spherical lenses, ocular motility and orthotropia were fully restored. A +3.00 D near addition, later incorporated into progressive spectacles, led to marked symptomatic improvement. At 18 months, she remained orthotropic and asymptomatic with progressive spectacles, but the esotropia persisted without correction. SNR with accommodative and convergence components can follow TBI and simulate bilateral sixth nerve palsy. Cycloplegic refraction and monocular ductions allow the diagnosis, and plus lenses may control the convergence component when cycloplegia fails.

Categories: Ophthalmology
Keywords: accommodative spasm, convergence spasm, cycloplegic refraction, esotropia, pseudo-abducens palsy, pseudomyopia, spasm of the near reflex, traumatic brain injury

Introduction

The near reflex is a physiological response that allows clear and single vision of near targets. It comprises the triad of accommodation (an increase in the refractive power of the lens to focus near objects), convergence (inward rotation of both eyes to maintain binocular fixation), and miosis (pupillary constriction, which increases the depth of field) [1]. These three components are normally activated together because they share a common supranuclear command. When a near target is fixated, cortical signals reach near-response neurons in the mesencephalon, close to the oculomotor nucleus. These neurons activate the medial rectus motoneurons, which produce convergence, and the parasympathetic neurons of the Edinger-Westphal nucleus, whose fibers synapse in the ciliary ganglion and contract the ciliary muscle and the iris sphincter, producing accommodation and miosis [2].

Spasm of the near reflex (SNR) is a disorder in which this response is inappropriately sustained, even when the patient is not fixating a near target [1,3,4]. SNR is rare, and in a case series of 22 patients published in 2021, 19 were female and the median age was 10 years [3]. The sustained activation is attributed to excessive parasympathetic activity [3,4]. In most patients, this excessive activity has a functional or psychogenic origin, but organic causes have also been described, including traumatic brain injury (TBI) and other central nervous system disorders [1,4].

Post-traumatic SNR has been reported mainly as isolated cases and small series. Its mechanism is uncertain, but trauma may either activate the mesencephalic centers that control the near response or damage the supranuclear pathways that normally inhibit them [5]. Chan and Trobe described six men with accommodative spasm after severe closed head injury, which persisted for at least seven years [5], and similar cases of persistent accommodative spasm have been reported [6,7]. Complete SNR has been described after head injury in an 84-year-old patient [8], and convergence spasm without limitation of abduction has been reported in an 18-year-old woman [9]. These reports are heterogeneous, and most describe isolated accommodative spasm in adults after severe head injury. In single cases, a functional component cannot always be excluded.

Regardless of its cause, the clinical presentation of SNR is variable, and isolated accommodative spasm is more common than simultaneous involvement of all three components of the triad [4]. Accommodative spasm causes pseudomyopia, convergence spasm causes a variable esotropia, and complete SNR combines both with miosis [1,4]. Patients typically report fluctuating or blurred vision, diplopia, and asthenopia [4].

The diagnosis is clinical and relies on cycloplegic refraction [4]. SNR must be distinguished from abducens

nerve palsy, convergence excess, and basic esotropia [3,4]. Convergence excess is an esotropia larger at near than at distance, whereas basic esotropia is a deviation of similar size at distance and near [10]. Convergence spasm can mimic abducens nerve palsy, because on attempted lateral gaze, the convergence drive keeps the abducting eye in adduction [1,11]. Unlike in a true palsy, abduction is full on monocular ductions and with the oculocephalic maneuver, and miosis usually accompanies the apparent limitation [1,2,11]. This distinction is particularly difficult after head trauma, both because trauma is a recognized cause of sixth nerve palsy and of supranuclear gaze disorders [2,12], and because blurred vision is easily attributed to the brain injury when cycloplegic refraction is not performed in the acute setting [5].

Once the diagnosis is established, treatment usually combines cycloplegia and plus lenses [4,13,14]. We report a case of post-traumatic SNR with accommodative and convergence components that mimicked bilateral abducens nerve palsy in an adolescent, a presentation that differs from most published post-traumatic cases. It illustrates the clinical findings that allow the diagnosis and the role of plus lenses in the management of the convergence component.

Case Presentation

A previously healthy 14-year-old girl sustained a frontal TBI after a fall with direct impact onto a hard wooden surface. She was initially asymptomatic, but approximately one hour later, she developed a near-syncopal episode followed by headache, nausea, imbalance, diplopia at near, and decreased near vision. In the emergency department, she presented with ocular motility abnormalities, decreased muscle strength (more pronounced on the right side), and an unsteady gait. Ophthalmological assessment revealed a bilateral abduction deficit, suggesting a non-isolated bilateral sixth nerve palsy. Non-contrast head computed tomography (CT) was unremarkable.

She was transferred to our tertiary care center and admitted to the pediatric ward, where she was assessed at the bedside by the ophthalmology team. Pupils were round and reactive, with no relative afferent pupillary defect. There was no miosis or ptosis. She had a large-angle esotropia of 30 prism diopters (PD) in primary position at near (Figure *1E*). Near visual acuity was worse than 20/100, the largest optotype available on the smartphone-based near visual acuity test used at the bedside. Versions showed a bilateral abduction deficit, with neither eye crossing the midline (Figure *1D*, F). The right eye was also limited in dextroelevation and dextrodepression (Figure *1A*, G), and the left eye in levoelevation and levodepression (Figure *1C*, I). Pupil size during attempted lateral gaze was not recorded.

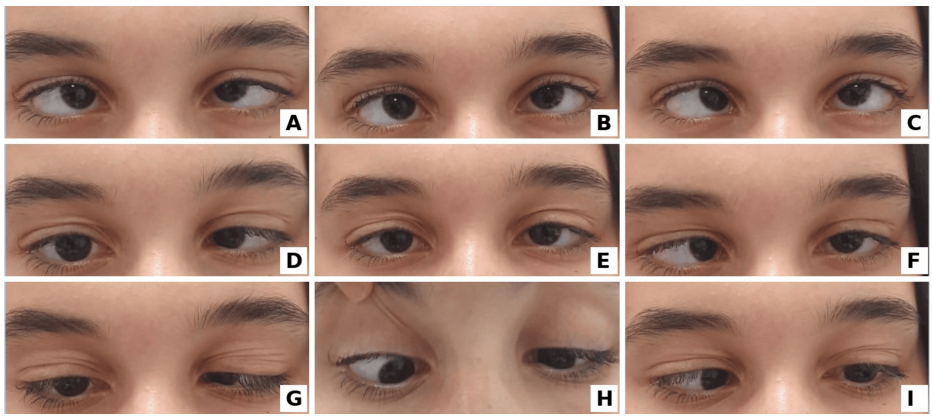


FIGURE 1: Nine-position gaze photographs at presentation.

Photographs are arranged in the standard nine-position layout and shown from the examiner's view (the patient's right eye is on the left of each panel). (E) Esotropia in primary position. (D, F) Bilateral abduction deficit on dextroversion and levoversion, with neither eye crossing the midline. (A, G) Limitation of the right eye in dextroelevation and dextrodepression, and (C, I) limitation of the left eye in levoelevation and levodepression, corresponding to the abduction component of these oblique positions rather than to a true vertical limitation. (B) Supraversion. (H) Infraversion.

Monocular ductions showed full abduction in both eyes (Figure *2*), and the oculocephalic (doll's head) maneuver elicited a normal response bilaterally. These findings indicated that the abducens nerves and lateral rectus muscles were functionally intact. Anterior segment biomicroscopy and fundoscopy were unremarkable, with pink optic discs with sharp margins, a normal macula, and an attached retina without lesions.

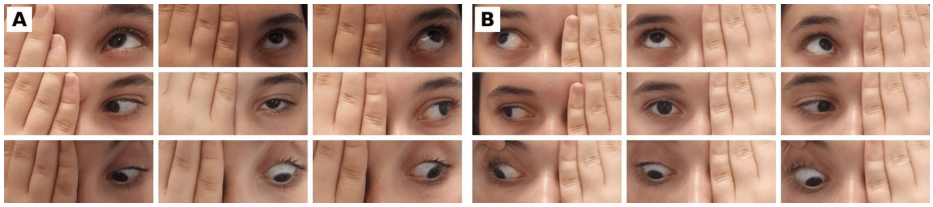


FIGURE 2: Monocular ductions.

Photographs are arranged in the nine-position layout and shown from the examiner's view. (A) Left eye, with the right eye occluded. (B) Right eye, with the left eye occluded. Abduction is full in each eye (middle row, right panel in A and left panel in B), in contrast with the abduction deficit on versions (Figure 1D, F).

Brain and orbital magnetic resonance imaging (MRI) was normal, excluding structural lesions (Figure 3). Neurosurgical consultation concluded that a post-concussive syndrome was the most likely diagnosis. At discharge, the ocular motility findings were attributed to a supranuclear lesion of the horizontal gaze pathways (possibly involving the frontal cortex or the paramedian pontine reticular formation) in the setting of concussion. During the one-week admission, she was also assessed by child psychiatry and psychology. The motor deficit and gait disturbance had resolved by discharge, and no further neurological or neurosurgical follow-up was required. No ophthalmological treatment was prescribed, and outpatient ophthalmology follow-up was arranged.

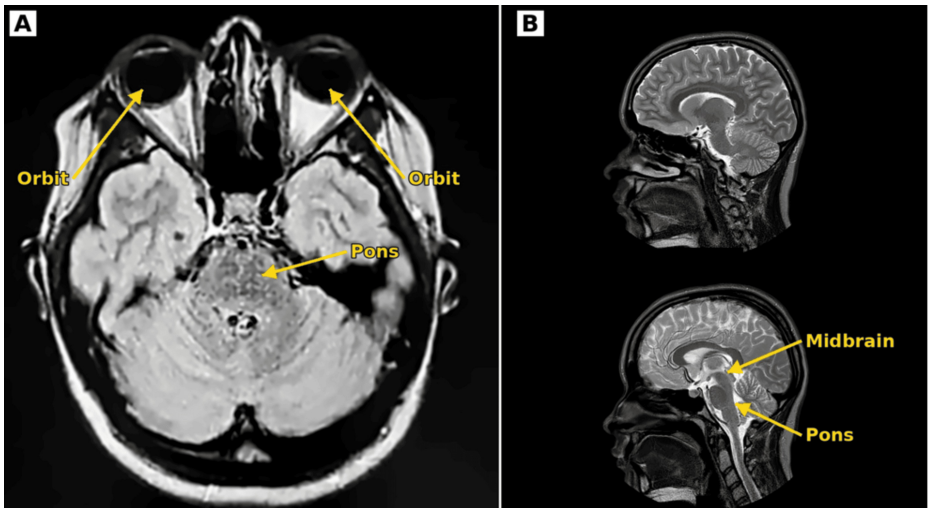


FIGURE 3: Brain and orbital magnetic resonance imaging.

(A) Axial image at the level of the pons and orbits. (B) Sagittal T2-weighted images. No structural abnormality is seen in the pons (which contains the abducens nuclei), in the midbrain (which contains the near-response neurons and the oculomotor nuclei), or in the orbits (arrows).

At the neuro-ophthalmology outpatient visit, one week after discharge, the patient still reported reduced near vision and diplopia at near. Autorefraction revealed high myopic values (right eye −7.50 −0.25 × 165°, left eye −8.00 −0.75 × 15°). However, best-corrected distance visual acuity on the Snellen chart was 20/20 in both eyes with only the astigmatic correction (right eye −0.25 × 165°, left eye −0.75 × 15°). The limitation of ocular movements persisted, and prism measurement showed an esotropia of 20 PD at distance and 30 PD at near. The accommodative convergence to accommodation (AC/A) ratio, calculated with the heterophoria method from these measurements and an interpupillary distance of 63 mm, was approximately 9.6 PD/D. The near point of convergence and the amplitude of accommodation could not be reliably measured because diplopia was present at the starting test position and no consistent subjective endpoint for blur could be identified. After cycloplegia with cyclopentolate, autorefraction shifted to hyperopic values (right eye +2.00 −0.25 × 165°, left eye +2.00 −0.75 × 15°), while the abduction deficit persisted and the esotropia appeared larger and was so variable that it could not be reliably quantified with prisms. However, with +5.00 D spherical trial lenses in both eyes, still under cyclopentolate cycloplegia, the motility limitation resolved completely and the patient became orthotropic in primary position (Figure 4). Orthotropia was confirmed with the cover test at distance and near. Lower plus powers were also tested, and +5.00 D was the lowest power that neutralized the deviation under cycloplegia.

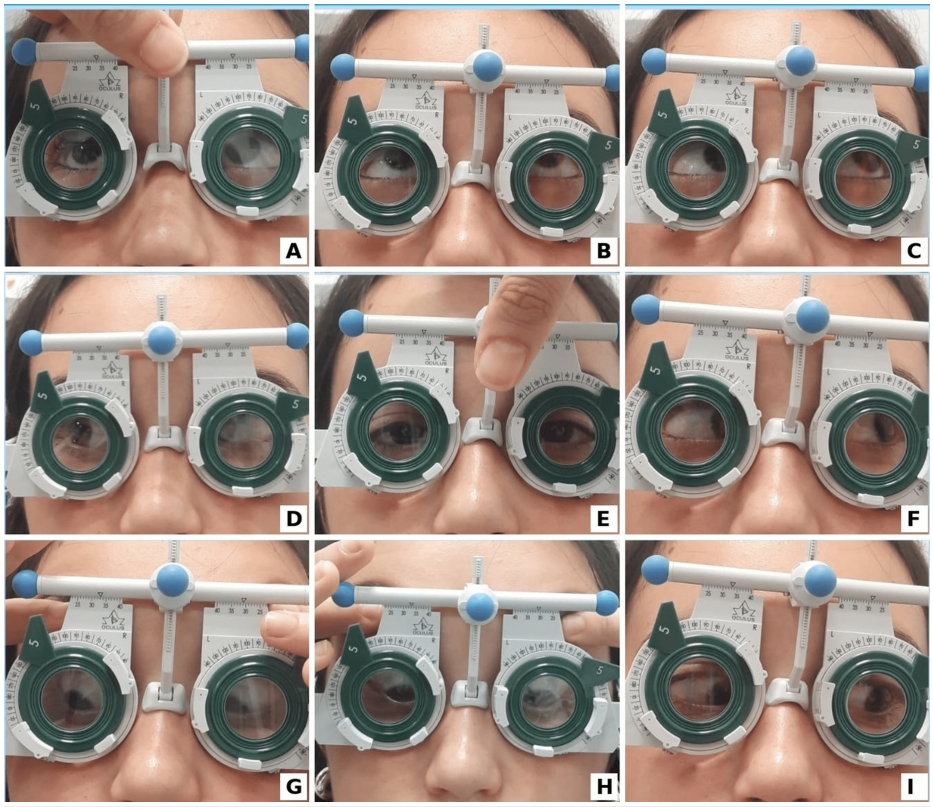


FIGURE 4: Nine-position gaze photographs under cycloplegia and with plus lenses.

Photographs taken under cyclopentolate cycloplegia at the first outpatient visit, with +5.00 D spherical trial lenses in both eyes, arranged in the standard nine-position layout and shown from the examiner's view. Versions are full in all positions, including abduction on dextroversion (D) and levoversion (F), and the patient is orthotropic in primary position (E), in contrast with Figure 1. (A) Dextroelevation, (B) supraversion, (C) levoelevation, (G) dextrodepression, (H) infraversion, and (I) levodepression. The same findings were observed with +5.00 D lenses under atropine at the reassessment.

Atropine 1% eye drops were prescribed twice daily in both eyes for one week. At reassessment, with nonreactive mydriasis confirming cycloplegia, the symptoms were unchanged and the patient reported poor tolerance to cycloplegia. Autorefraction showed hyperopic values similar to the previous examination, and the esotropia and motility limitation persisted. Both were again neutralized with +5.00 D lenses, while she was still under atropine. Atropine was therefore discontinued. Without cycloplegia, +3.00 D lenses were sufficient to neutralize the deviation, and a +3.00 D near addition was prescribed. The patient tolerated this correction well and reported marked improvement in diplopia and near vision.

One month later, she returned with asthenopia. Distance visual acuity had decreased to 20/25 in the right eye and 20/32 in the left eye, improving to 20/20 with a +0.50 D spherical correction. Progressive spectacles (+0.50 D sphere with a +3.00 D near addition) were prescribed.

At the child psychiatry follow-up, one month after discharge, no psychiatric disorder was identified, and she was discharged from psychiatric care. At the last follow-up, 18 months after the injury, she was orthotropic at distance and near and asymptomatic with progressive spectacles, and she used progressive contact lenses for sports. Without correction, the esotropia (20 PD at distance and 30 PD at near) and the abduction deficit persisted, and the manifest and cycloplegic refraction were similar to those at the first outpatient visit.

These findings were consistent with SNR with accommodative and convergence components secondary to TBI. The diagnosis was based on four findings: an abduction deficit present on versions but absent on monocular ductions and with the oculocephalic maneuver, a myopic shift of approximately 10 D between manifest and cycloplegic refraction, neutralization of the esotropia and motility limitation with plus lenses, and normal neuroimaging. The convergence component was inferred from the motility pattern and from the response to plus lenses, as it was not measured directly.

Discussion

We describe a case of post-traumatic SNR with accommodative and convergence components that initially

mimicked bilateral abducens nerve palsy. The convergence component persisted under cycloplegia and was neutralized only with plus lenses.

SNR with a convergence component and bilateral abducens nerve palsy share esotropia, a bilateral abduction deficit, and diplopia [3,11]. After a head injury, sixth nerve palsy was a reasonable first hypothesis. It is a recognized complication of closed head injury, attributed to stretching of the nerve along its long intracranial course or to injury at the petrous apex [12]. In our patient, however, abduction was limited on versions but full on monocular ductions, and the oculocephalic reflex was normal. These findings exclude a lesion of the abducens nucleus or nerve [2,11]. In summary, an abduction deficit limited to versions, with full monocular ductions and a normal oculocephalic response, a large myopic shift between manifest and cycloplegic refraction, and neutralization of the esotropia with plus lenses distinguish SNR from abducens nerve palsy [1,2,11]. The dissociation between versions and ductions points to a supranuclear disorder, which explains why a lesion of the horizontal gaze pathways, from the frontal eye fields to the paramedian pontine reticular formation, was considered during hospitalization [2]. SNR is itself a supranuclear disorder of the near response [2], but a refractive assessment was not performed at that stage. Visual acuity had been measured only at near, and its reduction was attributed to the neurological picture. Neuroimaging was warranted, given the focal motor deficit and the mechanism of injury. Once structural lesions had been excluded, however, distance visual acuity and refraction would have suggested the diagnosis earlier.

The difference of approximately 10 D between manifest and cycloplegic refraction established the accommodative spasm [3,4]. Because the esotropia was fully corrected with plus lenses and the calculated AC/A ratio was high, a decompensated accommodative esotropia with a high AC/A ratio was also considered. We consider it unlikely, as the patient had no previous strabismus or refractive error, the onset was acute and followed the trauma, and neither the pseudomyopia nor the dissociation between versions and ductions is expected in accommodative esotropia [1,4]. In addition, the heterophoria method assumes that accommodation is relaxed at distance and matches the dioptric demand at near. In the presence of accommodative spasm, neither assumption holds, and the calculated value may reflect the spasm rather than the true AC/A ratio. This alternative cannot, however, be completely excluded. Pupil size during attempted lateral gaze was also not assessed. Miosis accompanying the abduction deficit is a characteristic sign of convergence spasm [1,4], and its documentation would have strengthened the diagnosis. In its absence, the convergence component was inferred from the motility pattern and the response to plus lenses.

Our patient matches the usual demographic profile of SNR, an adolescent girl [3], but the etiology is less common. Unlike most published post-traumatic cases, which described isolated accommodative spasm [5-7], often in adults after severe head injury [5,7], our patient had a TBI with normal neuroimaging and a combined accommodative and convergence spasm, with a pseudomyopia of approximately 10 D. Accommodative and vergence dysfunctions are frequent after acquired brain injury [15]. In our patient, the acute onset immediately after TBI, the absence of psychiatric history, and a normal psychiatric evaluation are consistent with an organic rather than a functional cause [3,5]. However, a temporal association does not establish causality, and a normal psychiatric evaluation does not exclude a functional component. We hypothesize that the trauma disrupted or disinhibited the supranuclear centers that coordinate the near triad. Monteiro et al. found traumatic cerebellar and supratentorial lesions, but no midbrain abnormality, in post-traumatic accommodative spasm, which supports a mechanism of disinhibition [7]. Young patients may be more susceptible because of their large accommodative amplitude [5], which is consistent with the age of our patient. This mechanism cannot be demonstrated, as neuroimaging was normal. Functional SNR is usually associated with psychological stressors or psychiatric history [3,4]. The persistence of the findings at 18 months is consistent with an organic etiology, but it does not exclude a functional cause by itself, as functional SNR may also be chronic or recurrent [16]. Post-traumatic SNR has been described as resolving within one to two years [6], but persistence for several years has been reported in other cases [5,7]. Our patient therefore remains under follow-up.

Atropine, used without optical correction, relaxed the ciliary muscle but did not change the esotropia, whereas +5.00 D lenses neutralized it on both occasions they were tested. Under cyclopentolate without correction, the esotropia even increased and became more variable. Cycloplegic agents block the muscarinic receptors of the ciliary muscle and abolish accommodation [14], but they do not act on the central near-reflex drive. Under cycloplegia without correction, near targets remain blurred, and the effort to accommodate may persist or even increase. Because accommodative convergence depends on accommodative effort rather than on the actual change in lens power, convergence may be maintained despite paralysis of the ciliary muscle [17]. In healthy subjects, cycloplegia induces an esophoric shift at near and an increase in the AC/A ratio, attributed to increased central accommodative effort, and this shift is largely eliminated by plus lenses [17]. We hypothesize, although this remains speculative, that this explains the persistence of the esotropia under atropine. The +5.00 D lenses correspond approximately to the cycloplegic hyperopia (+2.00 D) plus a near addition of +3.00 D. By removing the accommodative demand, they may have reduced the stimulus to accommodative convergence. At distance, they also act as an optical fog, a strategy described in the management of accommodative spasm [13]. The combination of cycloplegia and plus lenses is considered first-line treatment [4,14]. In this case, the convergence component responded to plus lenses alone. Because atropine was used for only one week, without correction and with poor tolerance, this observation does not show that cycloplegia is ineffective. It suggests, however, that plus lenses should be tested early when the convergence component predominates. The later need for a +0.50 D

distance correction is difficult to explain. One possibility, which remains speculative, is partial relaxation of the spasm, unmasking part of the latent hyperopia.

This report has the limitations of a single case, and its observations cannot be generalized. The convergence component was inferred from the motility pattern and the response to plus lenses, and it was not measured directly. Accommodation was not measured objectively, for example with dynamic retinoscopy, and the near point of convergence and the amplitude of accommodation could not be reliably quantified. Pupil size during attempted abduction was not recorded. The AC/A ratio was calculated with the heterophoria method, which is unreliable in the presence of accommodative spasm, and the deviation could not be quantified with prisms under cycloplegia without correction. The atropine trial was short, without optical correction, and poorly tolerated. Finally, a functional component cannot be completely excluded.

Conclusions

SNR can mimic other neuro-ophthalmological conditions, including bilateral abducens nerve palsy after TBI. Once structural lesions have been excluded with neuroimaging, cycloplegic refraction, monocular ductions, and the oculocephalic reflex are useful to distinguish this supranuclear disorder of the near response from peripheral nerve palsies. Recognizing SNR in this setting may reduce repeated testing and inappropriate treatment. In this case, the convergence component was inferred from the motility pattern and the response to plus lenses, and an accommodative esotropia with a high AC/A ratio, although unlikely, could not be formally excluded because the AC/A ratio could not be reliably measured. The association with trauma is supported by the timing of onset but remains unproven. Post-traumatic SNR may persist for a long time, so prolonged follow-up is advisable. Plus lenses controlled the convergence component in this patient after a short trial of cycloplegia had been unsuccessful, an observation that requires further study.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Vera Sá Araújo, Rui Silva, Cristina Freitas

Acquisition, analysis, or interpretation of data: Vera Sá Araújo, Daniel Abilheira, Cristina Almeida

Drafting of the manuscript: Vera Sá Araújo

Critical review of the manuscript for important intellectual content: Vera Sá Araújo, Daniel Abilheira, Rui Silva, Cristina Freitas, Cristina Almeida

Supervision: Cristina Almeida

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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