



(CASE REPORT)



LONG-TERM VISUAL IMPROVEMENT AFTER SEVERE POST-TRAUMATIC CEREBRAL VISUAL IMPAIRMENT IN A PRESCHOOL CHILD: A CASE REPORT

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ABSTRACT

Background: Long-term outcomes after severe post-traumatic cerebral visual impairment (CVI) in children are rarely described, and evidence for vision rehabilitation in this group is limited to small, uncontrolled reports.

Case presentation: A 3-year-10-month-old child sustained road-traffic polytrauma with neuroimaging findings interpreted as consistent with hypoxic–ischaemic brain injury. Initial eye examination was normal except for significant hypermetropia, and severe bilateral CVI was diagnosed. The child underwent a 336-session, structured vision rehabilitation programme alongside spectacle correction and occupational therapy. At 7.5 years post-injury, binocular Snellen acuity had improved to 6/9, although the right visual field remained non-responsive throughout follow-up; the origin of this field deficit could not be established without formal perimetry or electrophysiology, and concurrent maturation and refractive change limited causal attribution to any single intervention.

Conclusion: Considerable visual recovery can occur even after severe acquired paediatric CVI, and long-term follow-up is needed before a definitive visual prognosis is made, since recovery may continue for years after injury.

Key-words: Cerebral Visual Impairment, Hypoxic-Ischemic Brain Injury, Craniocerebral Trauma, Recovery of Function, Visual Acuity

1 INTRODUCTION

Cerebral visual impairment (CVI) is a distinct form of visual dysfunction that cannot be attributed to ocular pathology or defects in the anterior visual pathways[1]. It is a prevalent cause of childhood vision impairment, and better survival

of extremely preterm children has increased its burden[2,3]. The dorsal visual stream is especially sensitive[1,4]. Frequent causes include prenatal hypoxia, cerebrovascular events, and meningitis; many children largely recover, but seizures are prevalent[5]. Cortical blindness following head injury is usually transient[6], and long-term post-traumatic outcomes are infrequently described; evidence for vision treatment is restricted to tiny, uncontrolled publications[7–9].

Key Messages

- Severe post-traumatic cerebral visual impairment can improve substantially over several years.
- Improvement cannot be attributed to rehabilitation alone, because recovery, maturation and refractive change coincided.
- Prospective studies with standardised measures and formal perimetry are needed.

2 CASE REPORT

A previously healthy and developmentally normal child aged three years and ten months received polytrauma in a road-traffic incident on 11 March 2018 (bilateral femur fractures; lung, liver, duodenal and pancreatic injuries) necessitating critical care. Computed tomography (CT) indicated bilateral posterior parietal subdural hemorrhage. Magnetic resonance imaging (MRI) five days later showed diffusion-weighted hyperintensities in both hemispheres, basal ganglia and splenium, interpreted as severe hypoxic–ischaemic encephalopathy, with haemorrhagic contusion in the left temporal lobe and peritrigonal region; repeat MRI confirmed bilateral parieto-occipital haemorrhagic lesions. Detailed systemic physiological data, including any hypoxic episodes, were not available; hence, the hypoxic–ischaemic process is inferred from imaging. Admission records indicated bilateral peripapillary and disc haemorrhages, a blurred right disc edge, reactive pupils and no relative afferent pupillary impairment. Behavioural perimetry two months after damage revealed missing responses in the right visual field.

Table 1: Structured vision rehabilitation programme (336 sessions of approximately 30–40 minutes, twice weekly, September 2018 to March 2022; 48 sessions per period).

Period (sessions)	Therapy domain	Office-based techniques	Home-based activities / therapeutic objective
1 (1–48)	Vision stimulation	Light ball (multi-coloured, textured) across gaze positions; lightbox with colourful toys; mirror tracking; face-to-face communication	Coloured/textured balls in different gazes. Builds visual curiosity, proprioceptive feedback, socialisation and bonding
2 (49–96)	Vision stimulation + eye-hand coordination	Black-and-white board activities; Marsden ball	Improves contrast discrimination; visual adaptation and eye-object coordination
3 (97–144)	Vision + eye-hand	LEA Playing Cards; LEA Domino Cards; peg board; wooden stacking tasks	Strengthens visual acuity and recognition; fine-motor skills, dynamic visual tasks and early problem-solving
4 (145–192)	Visuomotor + cognition	Tracing on board; colourful rings; nesting bowl; Sanet Vision Integrator / Visionova; LEA 3D puzzle; LEA Rectangle	Visionova (home-based digital vision therapy platform). Targets peripheral awareness, saccades, sensorimotor integration and spatial reasoning
5 (193–240)	Higher-order cognition	LEA Rectangle; LEA Mailbox; LEA Cone Adaptation Test	Shape, size and orientation discrimination; visual adaptation to illumination changes
6 (241–288)	Integration + consolidation	Sanet Vision Integrator / Visionova; balance board; walking rail; hopscotch	Visionova (home-based digital vision therapy platform). Bilateral integration, visual-spatial memory; links visual gains to gross-motor balance and mobility
7 (289–336)	School-readiness consolidation	Combined: vision stimulation + eye-hand coordination + cognition + occupational therapy; low-vision aids trial	All domains reinforced; preparation for a structured learning environment

At the first optometric exam, three months after damage, he could not focus or follow light; acuity, Hiding Heidi contrast sensitivity, LEA Wand visual fields, and cognitive visual functions could not be triggered. Cycloplegic refraction exhibited significant hypermetropia. Dilated fundus examination revealed clear media, connected retina, healthy discs without pallor, and normal macula and vasculature in both eyes, with no haemorrhage, pigmentary change, optic nerve head avulsion or retinal tears; scleral depression was not conducted since he was quite concerned. Severe bilateral CVI owing to post-traumatic brain damage was detected; ocular reasons were judged improbable. Generalised seizures occurred and were treated with antiepileptic medication; electroencephalography (EEG) in 2021 and 2023 indicated persisting generalised and multifocal interictal epileptiform abnormalities.

From September 2018 to March 2022, he attended a 336-session, seven-period vision rehabilitation course, moving from visual stimulation through visuomotor and cognitive training to school readiness (Table 1), accompanied by glasses and occupational therapy. A pediatric optometrist gave all sessions, with caregiver-reinforced home practice. Fixation on and following of light were present by September 2018, and acuity (measured initially with LEA grating paddles at 57 cm and then with Snellen optotypes at 6 m) and contrast sensitivity increased steadily (Table 2). No standardized evaluation was reported between August 2019 and June 2022. The right-sided visual field did not react at any evaluation after baseline, and cognitive visual abilities were clinician-rated as excellent at follow-up. At final follow-up, in September 2025, he knew faces at several metres, read big text at 40 cm and attended a mainstream school with educational help. Key events are presented chronologically in Table 3. Parental signed informed permission was acquired for publishing; ethics clearance was not necessary.

Techniques are listed by therapy domain; home-based activities and the therapeutic objective of each period are summarised in the final column.

Table 2: Longitudinal clinical findings from first assessment to last follow-up.

Date (time since injury)	Visual acuity	Contrast sensitivity	Visual field (behavioural)	Cycloplegic refraction	Fundus / cognitive / sensory findings
Jun 2018 (≈3 mo)	Not fixing/following light	Not demonstrable	Not elicitable	+5.00 DS OU	Dilated fundus: clear media, attached retina, healthy discs without pallor, normal macular appearance and vasculature, no haemorrhage OU. No consistent visual response; cognitive skills not measurable
Sep 2018 (≈6 mo)	Fixing & following light; LEA paddle + spectacles: 0.25 cpd (57 cm)	Hiding Heidi: detected demo plate	Right-sided visual field did not respond	+4.50 DS OU	No formal cognitive visual skills measurable
Feb 2019 (≈11 mo)	LEA paddle + spectacles: 2.0 cpd (57 cm)	Hiding Heidi + spectacles: 25%	Right-sided visual field did not respond	+3.00 DS OU	No formal cognitive visual skills measurable
Aug 2019 (≈17 mo)	LEA paddle + spectacles: 4.0 cpd (57 cm)	Hiding Heidi + spectacles: 1.25% @ 60 cm	Right-sided visual field did not respond	+2.50 DS OU	Facial expression recognition (with spectacles); LEA Mailbox all orientations
Jun 2022 (≈4 y 3 mo)	Snellen 6/120 OU	Pelli-Robson: 0.90 log unit	Right-sided visual field did not respond	+2.00 DS OU	Size/shape/colour recognition good; eye-hand coordination good; LEA puzzle – needed support
Jun 2025 (≈7 y 3 mo)	Snellen OD 6/48; OS 6/48; OU 6/30	Pelli-Robson: 1.35 log unit	Right-sided visual field did not respond	+2.25 DS OD; +1.75 DS OS	All domains good; visual tracing & tracking good; orientation good

Sep 2025 (≈7 y 6 mo)	Snellen OD 6/15; OS 6/12; OU 6/9	Pelli-Robson: 1.50 log unit	Right-sided visual field did not respond	+1.50 DS OU	All domains good; visual tracing & tracking good; orientation good
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Time since injury is calculated from the date of injury (11 March 2018). Visual acuity: LEA grating paddles tested at 57 cm (cycles per degree, cpd) up to August 2019; Snellen acuity tested at 6 m from June 2022 (the two methods differ in task and distance and are not directly comparable). Contrast sensitivity: Hiding Heidi (lowest contrast level detected) and Pelli-Robson (log units; higher is better). Refraction: cycloplegic refraction at every visit; dynamic retinoscopy was not performed. Visual field: behavioural responses. No standardised assessment was documented between August 2019 and June 2022. Acuity and contrast values are binocular unless specified; Hiding Heidi testing distance was 40 cm unless stated. All values of visual acuity and contrast sensitivity were recorded with full refractive correction. cpd, cycles per degree; OU, both eyes; OD, right eye; OS, left eye; DS, dioptre sphere.

Table 3: Timeline of key clinical events from injury to last follow-up.

Date (time since injury)	Event
11 Mar 2018 (age 3 y 10 mo)	Road-traffic polytrauma requiring intensive care; CT: bilateral posterior parietal subdural haemorrhage
Mar 2018 (five days after injury)	MRI: diffusion-weighted hyperintensities (severe hypoxic–ischaemic encephalopathy) and left temporal and peritrigonal haemorrhagic contusion; repeat MRI (date not recorded): bilateral parieto-occipital haemorrhagic lesions
May 2018 (≈2 mo)	Behavioural perimetry: absent right visual field responses suggested
Jun 2018 (≈3 mo)	First optometric assessment; severe bilateral CVI diagnosed (Table 2)
Sep 2018 – Mar 2022	Vision rehabilitation programme (Table 1) with spectacles and occupational therapy
Interval not recorded	Generalised seizures, managed with antiepileptic medication
2021 and 2023	EEG: persistent generalised and multifocal interictal epileptiform abnormalities
Jun 2022 (≈4 y 3 mo)	Post-programme assessment (Table 2)
Jun 2025 (≈7 y 3 mo)	Follow-up assessment (Table 2)
Sep 2025 (≈7 y 6 mo)	Last follow-up (Table 2); attends mainstream school with educational support

CT, computed tomography; CVI, cerebral visual impairment; EEG, electroencephalography; MRI, magnetic resonance imaging.

3 DISCUSSION

This case is notable for three reasons: it documents profound bilateral visual loss after road-traffic polytrauma with imaging-inferred hypoxic–ischaemic injury, a mechanism less common than perinatal hypoxia in large clinic series[5]; it records visual improvement over 7.5 years despite persistent epileptiform EEG abnormalities, whereas in one small series the child with uncontrolled seizures improved least[7]; and it highlights the difficulty of attributing improvement to any single intervention.

Healthy discs without pallor, normal macula and vasculature, no relative afferent pupillary defect, and severe posterior brain damage suggested a cerebral rather than ocular etiology, in agreement with the recognized CVI criteria[1]. Retinal function was not evaluated; therefore, a modest retinal influence cannot be ignored. The continuous right-sided field non-response was congruent with the observed left-sided posterior brain lesions. However, whether this indicates a real homonymous field impairment, visual inattention, or faulty behavioural testing could not be verified without thorough perimetry or electrophysiological testing. Likewise, since thorough systemic physiological data were missing, hypoxia episodes could be neither verified nor eliminated.

Improvement in CVI is widely documented and may represent brain plasticity, maturity, therapy, and enhanced testability[3,5]. The programme's organized, multimodal stimulation suited to functional level mirrors prior limited findings[7–9]. Attribution is problematic since rehabilitation, spontaneous recovery, maturation, refractive change, increased testability, and antiepileptic medication coincided, and the initial benefits (fixation and light following) came around the start of the training. Acuity and contrast sensitivity were tested with separate tests that varied in task and distance; Changes between August 2019 and June 2022 cannot be interpreted as a definite decline because the assessment instruments and task demands changed. The latter rise in Snellen acuity happened after the treatment finished, and the decline in hypermetropia is in accord with emmetropisation.

Limitations include the single-patient design, no pre-rehabilitation observation period, concurrent interventions, changing instruments, unreliable early behavioural testing, unmasked clinician-rated assessments, and no formal perimetry, electrophysiology, optical coherence tomography (OCT) or validated CVI-specific measures. Prospective studies employing standardized metrics, such as the CVI Range[10], are required.

4 CONCLUSIONS

This case demonstrates that substantial visual recovery can occur even after severe post-traumatic paediatric CVI, extending over several years and continuing well beyond the active rehabilitation period. Because rehabilitation, spontaneous recovery, maturation and refractive change coincided, improvement cannot be attributed to any single intervention. Clinically, the findings support sustained long-term follow-up before finalising a visual prognosis in children with severe acquired CVI, and highlight the need for prospective studies using standardised, validated CVI-specific outcome measures.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of conflict of interest

The authors report no conflicts of interest.

Statement of informed consent

Parental written informed consent was obtained for publication of this case, including clinical details and history; formal institutional ethics committee approval was not required for a single-patient case report at our institution.

AI assistance

No AI tools were used in the preparation of this manuscript.

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