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REVIEW

Impact of Acquired Brain Injury on Vision: Patterns, Assessment, and Rehabilitation

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Abstract

Acquired brain injury (ABI), including stroke and traumatic brain injury, is frequently associated with visual system impairments that range from basic sensory deficits to complex perceptual dysfunctions, substantially affecting patient independence, safety, and quality-of-life. This narrative review synthesizes current evidence on the patterns, underlying mechanisms, assessment strategies, and management of vision impairments following ABI, while also highlighting gaps in clinical care and research. A comprehensive literature search was conducted using PubMed, Scopus, and Google Scholar to identify studies addressing post-ABI visual deficits, their pathophysiology, rehabilitation approaches, and outcomes in both adult and pediatric populations. Visual impairments after ABI include visual field defects (e.g., homonymous hemianopia), oculomotor dysfunction, cortical visual impairment, and higher-order visual perceptual disorders such as visual neglect and visual agnosia. Accurate assessment requires interdisciplinary collaboration and the use of tools such as perimetry, visual evoked potentials, neuroimaging, and neurocognitive testing. Rehabilitation strategies encompass compensatory training, prism adaptation, vision therapy, and assistive technologies; however, the strength of evidence supporting these interventions remains variable, and standardized care pathways are lacking. Early screening, coordinated interdisciplinary management, and individualized rehabilitation programs are essential to optimize visual recovery. Further research is needed to establish robust evidence-based interventions and to integrate visual assessment and rehabilitation into comprehensive neurorehabilitation services.

Keywords: Acquired brain injury, visual impairment, cortical visual impairment, visual field loss, traumatic brain injury, stroke rehabilitation, neuro-ophthalmology

Introduction

Acquired brain injury (ABI), encompassing traumatic brain injury (TBI) as well as non-traumatic etiologies such as stroke, hypoxia, infection, and tumors, represents a leading cause of long-term neurological disability worldwide. In addition to cognitive and motor impairments, visual dysfunction is among the most common yet underrecognized sequelae of ABI. The visual system occupies nearly one-third of the human cerebral cortex, rendering it particularly susceptible to both focal and diffuse neural damage. As a result, even localized lesions may disrupt multiple visual pathways, producing a broad spectrum of deficits that substantially affect independence, mobility, and quality-of-life (1).

Epidemiological studies suggest that approximately 50-80% of individuals with ABI experience some degree of visual impairment, ranging from basic sensory deficits, such as visual field loss, to higher-order perceptual disturbances, including visual neglect, visual agnosia, and cortical visual impairment (CVI) (2). These abnormalities frequently coexist with oculomotor dysfunctions—such as strabismus, convergence insufficiency, and saccadic dysmetria—which further compromise binocular vision and reading efficiency. Despite their high prevalence, visual impairments are often overlooked during acute management and rehabilitation, where attention is typically directed toward more apparent motor or language deficits. This underrecognition may delay appropriate intervention and adversely affect functional recovery (3).

Growing recognition within neuro-ophthalmology and vision rehabilitation has underscored the importance of

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integrating systematic visual assessment into multidisciplinary ABI care. Early identification through standardized screening tools, including perimetry, ocular motility assessment, and evaluation of visual perceptual function, allows for timely and targeted interventions that may meaningfully improve patient outcomes. Rehabilitation approaches—such as compensatory scanning training, prism adaptation, vision therapy, and assistive technologies—have demonstrated increasing potential benefit; however, their implementation remains inconsistent across clinical settings (4).

Given the heterogeneity of ABI and the complexity of visual processing, a comprehensive understanding of post-ABI visual dysfunction is essential for the development of effective diagnostic and therapeutic frameworks. Accordingly, this narrative review aims to synthesize current evidence on the mechanisms, clinical manifestations, assessment strategies, and management of visual impairments following ABI, while identifying key gaps in research and clinical practice that must be addressed to optimize patient care.

Epidemiology of Visual Impairments After ABI

Visual dysfunction is among the most prevalent yet frequently underestimated sequelae of ABI. Epidemiological studies consistently report that approximately 50-80% of individuals with ABI experience some form of visual impairment during the acute or chronic phases of recovery (5). However, the true prevalence is likely higher, as subtle sensory deficits and higher-order visual perceptual disturbances may remain undetected in the absence of specialized assessment. Moreover, heterogeneity in study design, visual assessment methods, and patient populations contributes substantially to the wide variability observed in reported prevalence rates.

Global and Regional Prevalence

Globally, the World Health Organization estimates that more than 60 million people live with long-term neurological disability resulting from stroke and TBI combined, a substantial proportion of whom experience visual impairment (6). Among individuals with stroke, visual field defects—such as homonymous hemianopia and quadrantanopia—are reported in approximately 30-50% of cases. Oculomotor abnormalities, including gaze palsy, diplopia, and nystagmus, affect nearly 40% of stroke survivors, while visual neglect occurs in up to 30%, particularly following right hemispheric lesions (7).

TBI, another major contributor to ABI, is associated with an even higher burden of visual sequelae. Recent studies indicate that 60-70% of individuals with moderate-to-severe TBI experience one or more visual dysfunctions, ranging from accommodative and vergence abnormalities to deficits in visual processing (8). Although often considered less severe, mild TBI—commonly related to sports injuries or blast exposure—can also result in subtle yet functionally significant visual symptoms, including photophobia, blurred vision, and impairments in reading and visual attention.

Determinants and Outcomes

The likelihood and severity of visual dysfunction following ABI are influenced by several factors, including lesion location, the extent of diffuse axonal injury, patient age, and the presence of concomitant cognitive deficits. Early identification of visual impairments is frequently impeded by the limited integration of comprehensive vision assessment into routine neurological evaluation and rehabilitation protocols. Consequently, unrecognized visual deficits may contribute to delayed functional recovery, impaired mobility, increased risk of falls, and reduced reintegration into activities of daily living and employment.

Although the epidemiological burden of post-ABI visual impairment has been relatively well characterized in high-income Western countries, data from low- and middle-income regions remain limited. In the context of the rising global incidence of cerebrovascular disease and traumatic injury, enhanced epidemiological surveillance and the implementation of standardized visual screening protocols are essential to accurately define the scope of vision loss secondary to ABI.

Types and Mechanisms of Visual Impairments After ABI

The human visual system relies on the integrated functioning of ocular, cortical, and subcortical structures. ABI—whether caused by ischemic stroke, TBI, or hypoxic insult—can disrupt these networks at multiple levels, resulting in a broad spectrum of visual impairments. The type and severity of deficits depend on lesion location, extent of neural damage, and individual neuroplastic potential.

1. Visual Field Defects

Visual field loss is among the most prevalent visual consequences of ABI, particularly following occipital lobe lesions or posterior cerebral artery strokes. Disorders such as homonymous hemianopia, quadrantanopia, and scotomas arise from injury along the geniculocalcarine pathway, extending from the optic tract to the primary visual cortex. These deficits can significantly impair navigation, reading, and spatial orientation. Although spontaneous partial recovery may occur, persistent field loss often necessitates compensatory strategies, including visual scanning training or prism adaptation. Functional neuroimaging studies suggest that perilesional cortical reorganization may contribute to recovery in selected cases (9).

2. Oculomotor Dysfunction

Oculomotor abnormalities—including impaired saccades, smooth pursuit deficits, nystagmus, and convergence insufficiency—are

common following ABI, particularly in TBI. These deficits result from disruption of cortical-subcortical control circuits involving the frontal eye fields, cerebellum, and brainstem. Affected individuals frequently report diplopia, eye strain, or difficulty with reading. Quantitative assessment tools, such as eye movement recordings and infrared oculography, support accurate diagnosis and guide rehabilitation strategies, including vergence and pursuit training. Persistent oculomotor dysfunction may exacerbate dizziness and postural instability (10).

3. Cortical Visual Impairment

CVI arises from damage to the visual cortex or its associated white matter tracts, leading to deficits in visual perception despite normal ocular health. Although traditionally recognized in pediatric populations, CVI is increasingly identified in adults with ABI. Clinical manifestations include reduced visual acuity, impaired visual attention, and difficulty recognizing faces or objects. Neuroimaging studies indicate functional disconnection among occipital, temporal, and parietal regions. Rehabilitation emphasizes structured visual stimulation, environmental modifications, and targeted perceptual retraining (11).

4. Visual Neglect and Spatial Attention Deficits

Damage to the parietal or temporo-parietal junction can lead to visual neglect, characterized by the failure to attend to one side of space despite intact visual fields. This condition is particularly common after right-hemisphere stroke and is associated with severe disability and safety risks. Visual neglect arises from disrupted attentional control and interhemispheric imbalance rather than primary sensory loss. Interventions such as prism adaptation, scanning therapy, and non-invasive brain stimulation have demonstrated promising, albeit variable, benefits. Early detection using standardized assessments, such as the behavioral inattention test, improves rehabilitation outcomes (12).

5. Higher-order Visual Perceptual Disorders

In addition to primary visual deficits, ABI can result in complex perceptual disorders, including visual agnosia, prosopagnosia, and alexia, typically due to damage in the ventral visual stream connecting the occipital and inferotemporal cortices. These disorders often cooccur with cognitive or language deficits, which can complicate recognition and recovery. Management strategies primarily include cognitive-perceptual training and compensatory cueing, although large-scale trials assessing their efficacy remain limited.

Mechanistic Insights and Clinical Implications

Contemporary neuroimaging suggests that visual dysfunction following ABI arises not only from focal damage but also from network-level disconnection and maladaptive neuroplasticity. Injury to white matter tracts and trans-synaptic degeneration contributes to persistent deficits. Rehabilitation strategies that leverage visual neuroplasticity—such as repetitive stimulation and adaptive visual tasks—may facilitate recovery in selected patients. However, variability in injury patterns and the absence of standardized diagnostic criteria continue to limit widespread application.

A clear understanding of the mechanisms underlying visual dysfunction after ABI is essential for developing personalized interventions. The integration of neuro-optometric assessment, neuropsychology, and occupational therapy remains critical for achieving functional improvement and enhancing quality-of-life.

Types and Mechanisms of Visual Impairment in ABI

Visual dysfunction following ABI is diverse and reflects the complexity of the visual system, which involves multiple cortical and subcortical pathways. These impairments may result from direct structural damage to the visual cortex, optic radiations, or visual association areas, as well as secondary factors such as cerebral edema, ischemia, or diffuse axonal injury. The most commonly observed visual sequelae after ABI include visual field loss, oculomotor dysfunction, CVI, and higher-order perceptual disorders.

Visual field defects occur in approximately one-third of patients with stroke or TBI and typically present as homonymous hemianopia or quadrantanopia. These defects generally arise from lesions in the retrochiasmal visual pathways, particularly the optic radiations and occipital cortex. Patients with visual field loss often experience spatial disorientation, difficulty reading, and impaired mobility. Although partial recovery may occur within the first six months, persistent visual field loss requires compensatory strategies, such as visual scanning training or prism adaptation therapy (13).

Oculomotor dysfunction, including convergence insufficiency, saccadic dysmetria, strabismus, and impaired smooth pursuit, is also common after ABI. Lesions in the brainstem, cerebellum, or cortical eye movement centers disrupt binocular coordination, leading to symptoms such as diplopia, blurred vision, and eye strain. These dysfunctions are often underdiagnosed despite their significant impact on balance, mobility, and reading efficiency (14).

CVI represents a distinct form of visual loss resulting from cortical or subcortical injury, despite anatomically normal eyes. Individuals with CVI frequently exhibit fluctuating visual responses, difficulty recognizing complex scenes, and challenges with visual crowding. Functional magnetic resonance imaging (MRI) studies suggest that these symptoms are associated with altered connectivity and compensatory neuroplasticity in occipito-temporal pathways (15).

Higher-order perceptual disorders, including visual neglect, simultanagnosia, prosopagnosia, and visual agnosias, result from lesions affecting the parietal and temporal cortices. Visual neglect, particularly when associated with right parietal lobe damage, reduces awareness of the contralesional visual field and severely impacts daily functioning and spatial attention (16).

Mechanistically, ABI-induced visual deficits arise from both focal and diffuse neural injury. Hypoperfusion, excitotoxicity, inflammation, and axonal shearing contribute to secondary degeneration of interconnected visual networks. Advanced neuroimaging has revealed disrupted connectivity between fronto-parietal and occipito-temporal regions, which underlies persistent dysfunction. Understanding these mechanisms facilitates accurate diagnosis and informs targeted rehabilitation strategies (17).

Assessment of Visual Dysfunction After ABI

Evaluation of visual dysfunction following ABI requires a comprehensive, multidisciplinary approach that integrates neurological, ophthalmological, and optometric perspectives. Because visual deficits can range from basic sensory loss to complex perceptual disorders, no single test can capture the full spectrum of impairments. Early, structured visual assessment is essential to identify functional limitations, guide rehabilitation, and improve quality-of-life.

Clinical screening typically begins with a standard ophthalmic assessment, including visual acuity, refraction, and ocular health evaluation, to exclude preexisting ocular pathology. Visual field testing—performed using automated or manual perimetry—remains the cornerstone for detecting hemianopia, quadrantanopia, or scotomas. Goldmann and Humphrey perimetry can precisely delineate the extent and pattern of field loss, providing critical information for both diagnosis and rehabilitation planning (18). In acute settings where formal perimetry is impractical, bedside confrontation tests may serve as an initial screening tool.

Oculomotor assessment is equally important, as dysfunctions in vergence, saccades, and pursuit movements are common after ABI. Objective techniques, such as eye-tracking or video-oculography, can detect subtle abnormalities that routine clinical examination might miss. Specific assessments, including the developmental eye movement test and the King-Devick test, are useful for evaluating reading-related eye movements and can indicate underlying oculomotor inefficiencies (19). Additionally, pupillary responses and near point of convergence testing provide further insight into cranial nerve and brainstem function.

Assessment of visual attention, neglect, and higher-order perceptual deficits often requires neuropsychological evaluation. Standardized tests, such as the behavioral inattention test and the Bells test, are commonly used to detect unilateral neglect, whereas object and face recognition tasks can identify agnosias or prosopagnosia (20). Functional visual assessment, including observation of reading, navigation, and visually guided reaching, provides ecological validity to formal test results.

Neuroimaging techniques, particularly MRI and diffusion tensor imaging (DTI), are invaluable for identifying lesions within visual pathways and associated networks. These modalities can correlate structural damage with clinical symptoms and monitor recovery over time (21). Electrophysiological assessments, including visual evoked potentials, offer objective evidence of postchiasmal dysfunction and are particularly useful when behavioral responses are unreliable, such as in pediatric or severely impaired patients (22).

Given the complex interplay between visual, cognitive, and motor domains, interdisciplinary collaboration is essential. Optometrists, ophthalmologists, neurologists, and neuropsychologists should work together to ensure comprehensive evaluation and integrated management. The implementation of standardized vision screening protocols in neurorehabilitation programs has been shown to improve detection rates and facilitate timely intervention (23). Emerging digital technologies, including virtual reality (VR)-based visual field mapping and mobile vision assessment platforms, further enhance accessibility and accuracy in post-ABI visual evaluation (24).

Rehabilitation and Management Approaches in Visual Dysfunction After ABI

Rehabilitation of visual dysfunction following ABI aims to restore visual performance, enhance compensatory mechanisms, and improve functional independence. The complexity of visual processing and the heterogeneity of impairments necessitate a multimodal, interdisciplinary approach that integrates optometric, neurological, and occupational rehabilitation strategies.

Management begins with a comprehensive assessment of the type and severity of visual impairment, followed by individualized therapy plans. For patients with visual field deficits, compensatory techniques such as visual scanning training, systematic eye movement exercises, and reading retraining are commonly employed. Scanning therapy promotes systematic exploration of the blind hemifield, facilitating adaptation and improving detection of peripheral stimuli. Prism adaptation therapy, using yoked or sectoral prisms, has demonstrated efficacy in shifting the visual field and enhancing awareness of the impaired field (25,26). Recently, VR-based rehabilitation platforms have emerged as effective adjuncts, providing immersive environments for repetitive, feedback-based training (27).

Restorative approaches aim to enhance neural plasticity and residual visual field function through visual restitution therapy (VRT) and perceptual learning. These interventions involve repetitive visual stimulation near the border of the visual field defect to strengthen synaptic activity and cortical representation. Although the evidence remains mixed, some studies report measurable improvements in detection sensitivity and functional outcomes following sustained training (28,29).

Oculomotor rehabilitation targets common deficits after traumatic or ischemic brain injury, including convergence insufficiency, saccadic dysmetria, and pursuit impairments. Techniques such as vergence exercises, accommodative therapy, and dynamic saccadic training can restore binocular control and improve reading fluency. Computer-assisted oculomotor training and neuro-optometric rehabilitation have demonstrated promising results in enhancing fixation stability and visual endurance (30). Furthermore, integrating vestibular and balance training can further support overall recovery, particularly in patients experiencing postural instability or dizziness.

Management of CVI and higher-order perceptual disorders primarily emphasizes compensatory strategies and environmental modifications. Simplifying visual scenes, enhancing contrast, and providing structured routines can reduce visual crowding and cognitive load. For patients with visual neglect, interventions such as prism adaptation, optokinetic stimulation, and non-invasive brain stimulation techniques—including transcranial direct current stimulation—are under investigation for their potential to improve spatial awareness (31,32).

Assistive technologies are playing an increasingly important role in vision rehabilitation. Electronic magnifiers, head-mounted display systems, and augmented reality (AR) devices facilitate reading and mobility. Mobile applications offering gaze-tracking, text-to-speech, and scene interpretation have enhanced accessibility for individuals with visual-perceptual deficits (33). Emerging evidence also supports the integration of artificial intelligence-based adaptive vision aids, which can adjust display and contrast parameters in real time according to user needs (34).

Ultimately, successful rehabilitation depends on individualized goal setting, patient engagement, and early initiation of therapy. Interdisciplinary coordination among ophthalmologists, optometrists, neuropsychologists, and occupational therapists ensures comprehensive care. Despite advances, gaps remain in the standardization of rehabilitation protocols and the measurement of long-term outcomes, highlighting the need for high-quality, controlled trials to establish evidence-based best practices (35).

Discussion and Future Directions

Despite growing recognition of visual dysfunction following ABI, significant gaps remain in understanding its mechanisms, diagnosis, and management. The heterogeneity of ABI—including stroke, TBI, hypoxic injury, and intracranial hemorrhage—contributes to variability in visual outcomes and complicates the development of standardized rehabilitation approaches. Recent advances in neuroimaging, digital technologies, and neurorehabilitation have opened promising avenues for personalized interventions; however, integrating these approaches into routine clinical practice remains challenging (36,37).

Current evidence highlights the critical role of neuroplasticity in postinjury visual recovery. Functional MRI and DTI studies have demonstrated cortical reorganization within the occipital and parietal regions following targeted rehabilitation, particularly through VRT and perceptual learning paradigms (38). The extent of cortical plasticity, however, appears to depend on lesion location, size, and chronicity. This variability underscores the potential benefit of tailoring rehabilitation strategies to individual neural profiles, using imaging biomarkers as predictive tools to optimize outcomes (39).

Technological innovations—particularly VR, AR, and telerehabilitation—offer unprecedented opportunities for visual training. These tools create immersive, adaptive, and feedback-rich environments that can enhance patient engagement and facilitate home-based rehabilitation (40). Artificial intelligence powered gaze-tracking systems and machine-learning algorithms can further personalize therapy intensity and objectively monitor progress. Nevertheless, accessibility, cost, and the need for rigorous clinical validation remain significant barriers, especially in low-resource settings (41).

Multidisciplinary collaboration is another key determinant of successful outcomes. Integrated care models involving neuro-ophthalmologists, optometrists, occupational therapists, and neuropsychologists ensure that visual, cognitive, and perceptual deficits are addressed holistically (42). Despite this, vision rehabilitation remains underrepresented in many neurorehabilitation programs, often overshadowed by motor and language therapies. Incorporating vision screening protocols into early post-stroke and post-TBI care pathways can substantially improve detection rates and recovery potential (43).

Future research should prioritize three key areas. First, large-scale randomized controlled trials are necessary to establish evidence-based protocols for specific interventions, including prism adaptation, visual scanning, and non-invasive brain stimulation. Second, long-term follow-up studies should assess sustained functional gains and quality-of-life outcomes rather than focusing solely on short-term visual metrics. Third,

interdisciplinary and patient-centered research frameworks should incorporate patient-reported outcomes to address the psychosocial and occupational impacts of visual dysfunction (44).

In conclusion, although substantial progress has been made in understanding and managing visual impairments following ABI, the field remains in an evolving state. Bridging the gap between neuroscience, technology, and rehabilitation practice will be critical to achieving meaningful visual recovery and enhancing life participation among affected individuals (45).

Conclusion

Visual dysfunction following ABI remains a significant yet frequently underrecognized contributor to long-term disability. Early screening and targeted rehabilitation can substantially enhance functional recovery and quality-of-life. Incorporating visual assessment into standard neurorehabilitation programs is essential for comprehensive care. A coordinated, multidisciplinary approach—augmented by advancing technologies such as VR and telerehabilitation—offers promising opportunities for improving visual outcomes. Ongoing research and the standardization of evidence-based practices will be critical to ensuring that vision rehabilitation becomes an integral component of brain injury recovery globally.

Footnotes

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