Review article

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Injury of the visual analyzer in blast trauma: mechanisms, diagnosis, treatment

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Blast trauma is one of the most challenging problems in modern ophthalmology and neurology, as it is often accompanied by severe injuries of the visual system. According to various authors, ocular involvement accounts for up to 28% of all blast-related injuries. Traumatic brain injury (TBI), which frequently coexists with blast trauma, is complicated by ophthalmic disorders in 84% of cases. This highlights the exceptional vulnerability of the eye to blast-related factors — shock wave, thermal effects, and fragments.

The most common injuries include open globe trauma, intraocular foreign bodies, globe rupture, retinal detachment, and traumatic optic neuropathy. Secondary factors (shrapnel, building debris, soil, metal) markedly increase the risk of severe complications such as endophthalmitis, post-traumatic glaucoma, and retinal neovascularization, which often lead to disability.

Diagnosis requires a comprehensive approach involving ophthalmological methods (ophthalmoscopy, ultrasound, optical coherence tomography), neurophysiological techniques (visual evoked potentials, electroretinography), and neuroimaging (CT/MRI of the orbits and brain). Their combination enables detection of both local ocular damage and central visual pathway impairment.

Treatment includes emergency surgery (globe repair, removal of foreign bodies, vitreoretinal interventions), infection prophylaxis (systemic and local antibiotic therapy), as well as anti-inflammatory and immunomodulatory therapy. Timely prevention of sympathetic ophthalmia is of particular importance. Further rehabilitation involves restorative and functional methods aimed at preserving residual vision and improving patient adaptation.

Thus, blast-related injury of the visual analyzer is characterized by multifactorial mechanisms and a high risk of permanent vision loss. Optimal diagnosis and treatment are possible only through a comprehensive multidisciplinary approach with an emphasis on early intervention and long-term rehabilitation.

Keywords: *blast trauma; visual analyzer; visual analyzer contusion; traumatic optic neuropathy; diagnosis; rehabilitation*

Introduction

In the context of contemporary armed conflicts, the number of patients with blast traumas (BT) accompanied by damage to the organ of vision has been steadily increasing. Given the extraordinary diversity, combined effects, and complexity of blast injury mechanisms, ocular trauma resulting from explosive injuries represents one of the most severe and challenging problems in modern ophthalmology and neurology [1]. Studies have demonstrated that a substantial proportion of blast-related injuries to the visual analyzer are associated with persistent visual impairment or complete vision loss [2]. According to various authors, blast-related ocular injuries account for approximately 28% of the total number of injuries caused by blast wave exposure [3, 4]. During Operations Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF), 10–15% of combat-related injuries involved ocular damage [5]. Traumatic brain injuries (TBI) are frequently accompanied by visual system complications, with a reported incidence of up to 84% [6]. These findings indicate a high vulnerability of the visual analyzer to both direct and indirect effects of blast waves, fragments, and thermal factors.

Explosive devices widely used in modern warfare and terrorist attacks often result in severe damage to the visual analyzer. The most typical injuries associated with such trauma include open globe injury, intraocular foreign bodies, globe rupture, as well as complications such as retinal detachment and traumatic optic neuropathy [7].

Secondary blast factors—such as shrapnel, building debris, soil particles, and metal fragments—pose a particular threat, as they penetrate the structures of the eyeball at high velocity. Even small intraocular foreign bodies may lead to serious complications, including

endophthalmitis, post-traumatic glaucoma, and retinal neovascularization, resulting in a high rate of patient disability [8, 9].

Issues related to timely diagnosis, surgical management, and rehabilitation of patients with blast-related ocular injuries have become increasingly relevant in modern armed conflicts. Early and high-quality diagnosis of visual impairments is critically important for preventing long-term disability in affected patients.

Mechanisms of visual analyzer injury in blast trauma

The eyeball is an exceptionally vulnerable anatomical structure when exposed to blast-related factors, owing to its spherical shape, high fluid content, rich vascularization, and the delicate architecture of its internal tissues [10]. Its exposed anatomical position and the absence of rigid bony protection render the organ of vision particularly susceptible to the mechanical, thermal, and barometric effects of blast waves. Even brief exposure to overpressure or fragment-related injury may result in profound structural damage, ranging from contusion to complete globe rupture [11, 12].

Explosive devices cause four principal types of BT, which differ in their mechanisms of action and in their effects on the visual analyzer [13].

Primary BT results from the impact of the shock wave, which generates excessive overpressure propagating through media of varying densities. Organs containing air—such as the lungs, intestines, and tympanic membrane—are the most vulnerable [14]. In the visual system, the shock wave may induce globe contusion, retinal detachment, intraocular hemorrhage, and traumatic optic neuropathy. The shock wave leads to an abrupt rise in intraocular pressure (IOP), posing a significant threat to the integrity of the ocular coats. Peak intraocular pressure during a blast may reach 0.29 MPa (≈ 2175 mmHg) as early as 1.63 ms after shock wave exposure, which is more than twice the normal physiological IOP (~ 15 mmHg) in healthy eyes [15]. Additional factors determining the pattern and severity of blast-related ocular injury include peak overpressure, the duration of blast wave exposure (pressure impulse), distance from the explosion epicenter, head orientation, and gaze direction at the moment of the explosion (i.e., whether the eyes were directed toward the source of the shock wave). These parameters substantially influence intraocular pressure elevation, the degree of globe deformation, and the likelihood of optic nerve and retinal damage [16, 17].

Secondary BT represents the most common mechanism of ocular trauma [18]. It is caused by direct impact of fragments from the explosive device itself or exogenous debris propelled by the blast wave, including shrapnel, glass, metal, and soil particles. Such fragments may result in open penetrating globe injuries and rupture of the cornea or sclera [19]. Damage to superficial ocular structures is also possible, manifesting as closed globe injuries, including superficial foreign bodies, lamellar (partial-thickness) lacerations, or non-penetrating blunt trauma with or without globe rupture. These injuries are frequently associated with combined craniofacial and abdominal trauma [20, 21]. From a physiological standpoint, the primary blast wave reaches the eye significantly earlier than the fragments it carries; however, once fragments strike the eyeball, the intensity and severity of injury increase sharply. Thus, secondary injuries are "secondary" not only in terms of their underlying mechanism but also in the sequence of their occurrence. They rarely occur in isolation and are almost always accompanied by primary blast injuries, resulting in complex and combined trauma patterns of varying severity [22].

Tertiary BT occurs when the "blast wind" propels the injured individual against solid surfaces or objects [23]. This mechanism results in blunt trauma, including closed globe injuries, orbital fractures, compressive injury to the optic nerve, and damage to the extraocular nerves and muscles. Characteristic manifestations include orbital trauma (bone fractures), globe compression, optic nerve injury at the level of the optic canal (traumatic optic neuropathy), ptosis, and paralysis of the extraocular muscles due to cranial nerve damage [24].

Quaternary BT encompasses additional pathophysiological effects not directly related to the blast wave or fragment impact. These include exposure to high temperatures, intense light (flash), and toxic substances. Such injuries comprise thermal burns of the eyelids, cornea, conjunctiva, and facial tissues caused by extreme heat; photochemical retinal damage ("flash blindness"); and chemical burns of the eyes resulting from explosive materials [25, 26]. Quaternary injuries may also be associated with respiratory tract damage, falling debris, leading to craniofacial trauma, including skull base fractures and orbital involvement [27].

A study by Ying-Ying Zou *et al*. (2013) demonstrated that blast wave exposure induces pathological changes in the retina, accompanied by increased expression of proteins involved in inflammation, edema, and apoptotic processes, including vascular endothelial growth factor, aquaporin-4, and glutamate synthetase etc. These changes were detected immediately after blast wave exposure and persisted for up to two weeks. The most pronounced alterations were observed in astrocytes and Müller cells, indicating their critical role in the retinal pathophysiological processes triggered by blast injury [28].

Combined injuries are of particular clinical significance. Numerous studies have demonstrated a close association between blast-related ocular trauma and systemic injuries. In the study by Erdurman *et al*., the incidence of combined injuries was 69.0%; Weichel *et al*. reported 66.0%, while Kalayci *et al*. reported 60.7% [9, 13, 20]. The most common associated injuries involve the maxillofacial region, musculoskeletal system, and thorax. In cases of concussion, visual manifestations are frequently observed due to the extensive integration of the visual system within the cerebral cortex.

Blast trauma is often accompanied by TBI, which may affect higher levels of the visual analyzer, including the optic nerve, optic chiasm, optic tracts, and the visual cortex of the occipital lobe [29]. Such injuries may result in partial or complete vision loss, homonymous hemianopia, visual agnosia, and other visual dysfunctions. TBI is frequently the consequence of diffuse axonal injury caused by rapid acceleration, deceleration, or rotational movements of the head. These mechanical forces lead to abrupt neuronal dysfunction

with massive release of accumulated neurotransmitters, resulting in excessive activation of postsynaptic membranes. This process initiates a pathophysiological cascade characterized by increased energy demand, metabolic crisis, and impaired glucose metabolism in neurons [30, 31].

Acute symptomatology typically persists for 7–10 days and is transient in nature, with gradual functional recovery over several weeks. However, in a subset of patients, residual or persistent symptoms may remain for months or even years [32]. The most common manifestations of post-concussive syndrome include headache, migraine, cognitive impairment, reduced concentration, and prolonged reaction time. In some cases, these conditions may be associated with delayed and long-term neurodegenerative processes [33].

In blast-related injury to the visual analyzer, damage is frequently bilateral. According to the literature, binocular involvement in BT occurs in 3.33–72.91% of cases, whereas in "conventional" causes of ocular injury (domestic, sports-related, or occupational trauma), the incidence of bilateral involvement is only 0–2.13% [2, 13].

Diagnosis

Assessment of the visual analyzer in patients who have sustained BT is a complex multidisciplinary task requiring the involvement of not only an ophthalmologist but also a neuro-ophthalmologist, neurologist, otolaryngologist, neurophysiologist, and, in some cases, a neurosurgeon. The primary objectives include identifying ocular and optic nerve disorders, detecting damage to the central components of the visual analyzer (cortical visual areas), and determining the extent and nature of injury (mechanical, ischemic, compressive, contusive, or combined).

Careful collection of medical history is of critical importance. The evaluation should include a detailed analysis of the circumstances of the explosion, such as distance from the epicenter, intensity of exposure, duration of blast impact, and use of personal protective equipment. Particular attention should be paid to a history of loss of consciousness and to patient-reported symptoms, including decreased visual acuity, diplopia, ocular pain, and tearing [34].

In addition, prior ophthalmic history and the presence of concomitant systemic diseases that may affect visual function or hinder recovery should be assessed.

Clinical examination includes evaluation of the eyelids and conjunctiva, identification of foreign bodies, hematomas, hemorrhages, and orbital deformities. Despite the possible absence of overt external ocular or adnexal injuries in patients with BT, comprehensive ophthalmological examination remains essential. This should include assessment of visual acuity (visometry), tonometry (measurement of intraocular pressure), evaluation of pupillary responses (including relative afferent pupillary defect), analysis of extraocular motility, and slit-lamp examination to detect traumatic lesions of the cornea, anterior chamber, and lens [35].

Cockerham *et al*. emphasize the importance of assessing not only visual acuity using high-contrast charts but also spatial contrast sensitivity and visual fields [36]. Retinal or central nervous system damage may not reduce standard visual acuity yet may be associated with abnormalities in visual fields, contrast sensitivity, or color perception [34].

Perimetry is a method used to examine and evaluate the visual field, defined as the area visible to an individual with a fixed gaze and stationary head position. It is a key test for glaucoma diagnosis as well as for assessing the condition of the optic nerve and retina [37]. The main types of perimetry include kinetic perimetry, which employs a moving light stimulus, and static perimetry, in which stationary stimuli of varying intensities are presented. Standard automated perimetry (SAP) is currently the most widely used technique, utilizing automated static testing to achieve more standardized and reproducible results [38].

Imaging studies may be useful for clarifying the nature and extent of injury. Computed tomography (CT) and magnetic resonance imaging (MRI) of the orbit allow visualization of the optic nerve and optic canal and enable assessment of orbital fractures, bone fragments, or optic nerve sheath hematoma [39].

Ophthalmic imaging modalities, such as ophthalmoscopy and optical coherence tomography (OCT), can detect subtle but clinically significant changes, including optic disc edema or thinning of the retinal nerve fiber layer, as well as the presence of hemorrhages, tears, or retinal detachment, etc [40].

Optical coherence tomography is a modern noninvasive diagnostic technique in ophthalmology that uses the properties of coherent light to obtain high-resolution three-dimensional images of intraocular structures, including the retina, optic nerve, and cornea. This method enables detection of pathological conditions such as degenerative processes, glaucoma, optic nerve disorders, and minute tissue alterations, making OCT a versatile diagnostic tool in ophthalmic practice [41].

The operation of an optical coherence tomograph is based on the principle of light interferometry. Light interference refers to the spatial redistribution of radiant energy resulting from the superposition of two or more light waves.

On OCT images, retinal layers are differentiated according to their reflectivity. A standard color scale is used for image reconstruction: highly reflective structures are displayed in red and white tones, whereas weakly reflective structures appear in dark colors (black, blue, or dark green) [42, 43].

Retinal thickness is defined as the distance from the inner retinal surface to the level of the retinal pigment epithelium (RPE). However, an important methodological consideration should be noted: some OCT devices measure thickness to the inner boundary of the RPE, whereas others measure to its outer boundary. Consequently, discrepancies may arise, complicating comparisons of retinal thickness obtained using different OCT systems [44].

To obtain tomographic images of the optic nerve head, both longitudinal and circular linear scanning are employed. OCT allows assessment of optic disc diameter, excavation size and depth, cup-to-disc ratio (area-based as well as horizontal and vertical meridians), and retinal nerve fiber layer thickness in the peripapillary region [45].

An important technical feature of optical coherence tomography is the use of an infrared light beam with an

average wavelength of approximately 830 nm, which enables layer-by-layer retinal imaging, quantitative assessment of retinal thickness, and evaluation of the extent and distribution of pathological changes [43].

Functional magnetic resonance imaging (fMRI) is a neuroimaging technique that enables assessment of brain functional activity based on changes in cerebral blood flow (blood oxygen level–dependent, BOLD, signal). In studies of the visual analyzer, fMRI is used to map the visual cortex, analyze functional organization and neuroplasticity following traumatic injury, and investigate cortical responses to visual stimuli [46]. This method is particularly valuable in comprehensive diagnostics, as it complements structural neuroimaging data and allows correlations between morphological alterations and functional activity to be established [47].

In addition to clinical ophthalmological examination and neuroimaging techniques, neurophysiological methods are also employed in the diagnosis of visual analyzer injury. The most widely used include visual evoked potentials (VEPs), which provide an objective assessment of visual signal conduction along the visual pathways from the retina to the cerebral cortex, and electroretinography, which is used to evaluate retinal function and electrophysiological activity [48]. These methods have considerable potential for comprehensive diagnosis and patient monitoring and will be discussed in greater detail in subsequent publications.

The principal diagnostic methods for assessing visual analyzer injury in BT are presented in ***Table 1***.

Therapeutic Approaches and Rehabilitation

Treatment of visual analyzer injury is often complex and prolonged. Depending on the nature of the injury, patients may require different therapeutic modalities, including conservative management, pharmacological therapy, surgical intervention, or a combination thereof. Primary care consists of applying a sterile dressing and transporting the patient to a medical facility as rapidly as possible. Hospitalization is frequently required, with multiple surgical procedures aimed at preserving globe integrity and restoring visual function. Appropriate surgical techniques are selected on an individual basis according to the clinical scenario [49]. At the initial stage, the most common interventions include globe repair, enucleation with implantation of an ocular prosthesis, and removal of intraocular and orbital foreign bodies. The second stage (planned procedures) may involve enucleation and orbital reconstruction, pars plana vitrectomy, including potential use of silicone oil to maintain globe configuration and normal intraocular pressure [50, 51]. Following surgery, patients with significant retinal fibrosis or scarring are at increased risk of tractional retinal detachment or may experience difficulty in achieving retinal reattachment, necessitating prolonged retention of silicone oil within the eye.

Proliferative vitreoretinopathy is a process of intraocular scarring characterized by the growth and contraction of cellular membranes within the vitreous cavity, on both surfaces of the retina, as well as intraretinal fibrosis [52, 53]. It typically develops after retinal breaks, allowing retinal pigment epithelial cells to migrate into the vitreous space. Currently, no pharmacological agents have been proven to prevent or modulate the development of proliferative vitreoretinopathy. Vitrectomy remains the only effective treatment; however, there is no consensus regarding patient selection or the optimal timing of surgical intervention [54, 55].

Table 1. Instrumental diagnostic methods for visual analyzer injury in blast trauma

Method	Level of injury assessed	Typical findings in BT
Ophtalmoscopy	Retina, optic disc	Hemorrhages, retinal detachment, secondary optic nerve atrophy
Optical Coherence Tomography (OCT)	Retinal nerve fiber layer, ganglion cell layer	Thinning of the retinal nerve fiber layer, signs of optic nerve atrophy
Perimetry	Function of visual pathways from the retina to the cortex	Scotomas, visual field defects (asymmetric, often with non-classical topography)
ERG	Function of photoreceptors and inner retinal layers	Reduced amplitudes or localized response defects in contusion-related retinopathy
VEP, (conventional)	Conduction from the retina to the cortex (optic nerve, chiasm, optic tracts)	Reduced P100 amplitude, prolonged latency, interhemispheric asymmetry, absence of response in severe injury
mfVEP	Local assessment of conduction within different sectors of the visual field	Focal defects corresponding to localized nerve fiber damage (partial involvement of the optic nerve, chiasm, or optic tract)
CT/MRI of the orbits and brain	Orbits, optic nerves, chiasm, optic tracts, occipital cortex	Orbital fractures, hematomas, optic nerve compression or rupture, diffuse axonal injury
fMRI	Occipital cortex	Reduced or absent activation of the primary visual cortex during stimulation of the affected eye

Note. ERG — electroretinography; mfVEP — multifocal visual evoked potentials.

In addition, many patients require oculoplastic, corneal, or glaucoma surgery, while a smaller proportion need consultation with a uveitis specialist, underscoring the importance of a multidisciplinary approach.

One of the most serious complications is sympathetic ophthalmia, a granulomatous autoimmune uveitis that occurs in 0.06–0.19% of cases and may lead to vision loss in the contralateral eye [56]. Historically, severely injured eyes were believed to require enucleation within two weeks after trauma to prevent sympathetic ophthalmia; however, neither the timing nor the effectiveness of this practice has been supported by robust evidence. Prompt initiation of corticosteroid therapy or immunosuppressive agents (cyclosporine, azathioprine) in cases of sympathetic ophthalmia can significantly improve treatment outcomes [57].

Therefore, available evidence supports the principle that even in cases of severe open globe injury, every effort should be made to preserve and reconstruct the eye whenever clinically feasible.

Endophthalmitis ranks second after open globe trauma and affects approximately 16.5% of patients. In this context, systemic antibiotic prophylaxis has limited evidence of efficacy, whereas intravitreal antibiotic injections, although used less frequently, are supported by stronger evidence [58, 59].

Prognosis

The prognosis of blast-related visual analyzer injury remains variable and depends on multiple factors, including the severity of the primary injury, involvement of the optic nerve, timeliness and effectiveness of therapeutic interventions, individual compensatory capacity, and response to treatment. In some patients, substantial recovery of visual function is possible, whereas others experience persistent and disabling visual impairment. Importantly, prognosis improves significantly with early diagnosis and initiation of treatment within the first hours to days following injury.

Conclusions

Combat-related blast trauma (BT) is a leading cause of combined damage to the visual analyzer. The pathophysiological mechanisms of such trauma are complex and multifactorial, and the absence of visible ocular damage does not preclude injury. Assessment of the visual analyzer in patients after BT requires a comprehensive approach, including the evaluation of both structural and functional characteristics of the eye and visual pathways. Blast injury may result in direct mechanical damage to the eye as well as secondary changes caused by the effects of the shock wave, exposure to high temperatures, infrared radiation, toxic substances, and other factors.

The examination of these patients should be comprehensive and multidisciplinary, involving an ophthalmologist, neurologist, neurosurgeon, and, when necessary, a plastic surgeon. Careful collection of the history of the blast event, when feasible, enables initial risk stratification and helps in selecting optimal diagnostic tools. Blast-related ocular injuries may present with a wide spectrum of symptoms, ranging from minimal discomfort to severe pain or complete loss of vision.

A basic ophthalmological examination (visometry, tonometry, pupillary reactions, slit-lamp examination, assessment of ocular motility, and perimetry) is mandatory even in the absence of obvious external injuries. It is essential to perform not only an initial but also a dynamic assessment, as a number of pathological changes (e.g., optic nerve atrophy) may become apparent in the delayed period.

Neuroimaging and neurophysiological methods play an important role in the comprehensive diagnosis and monitoring of patients with visual analyzer injuries. These techniques allow not only for the precise determination of the localization and nature of structural damage, but also for objective assessment of the functional state of the visual pathways and retina, which is crucial for determining prognosis and selecting an appropriate treatment strategy.

To improve long-term outcomes, regular ophthalmological and neurophysiological monitoring is required, along with active patient involvement in visual rehabilitation programs, training in compensatory strategies, and the use of a multidisciplinary approach. Even in cases with unfavorable functional recovery, comprehensive support and rehabilitation can significantly improve patients' quality of life.

Disclosure

Conflict of Interest

The authors declare no conflict of interest.

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