

Early diagnosis and prognosis of traumatic optic neuropathy

Bilalov E.N.

DSc, Professor, Ophthalmology Department, Tashkent Medical Academy, Uzbekistan

Bahritdinova F.A.

DSc, Professor, Ophthalmology Department, Tashkent Medical Academy, Uzbekistan

Narzikulova K.I.

DSc, Associate Professor, Ophthalmology Department, Tashkent Medical Academy, Uzbekistan

Nazirova S.H.

PhD, Associate Professor, Ophthalmology Department, Tashkent Medical Academy, Uzbekistan

Egamberdieva S.M.

Assistant of Ophthalmology Department, Tashkent Medical Academy, Uzbekistan

Oralov B.A.

PhD, Assistant of Ophthalmology Department, Tashkent Medical Academy, Uzbekistan

Pirnazarov M.Y.

Assistant of Ophthalmology Department, Tashkent Medical Academy, Uzbekistan

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Abstract: Traumatic optic neuropathy (TON) caused by blunt eye injuries represents a significant clinical challenge, frequently leading to irreversible vision loss. Early diagnosis and monitoring of retinal and optic nerve changes are crucial for improving patient outcomes. Optical coherence tomography (OCT) offers unparalleled capabilities in detecting and quantitatively assessing such changes.

Objective. To evaluate the diagnostic potential of OCT in identifying morphological and functional changes in the retina and optic nerve among patients with TON.

Materials and Methods. A total of 40 patients with blunt eye trauma were examined. The diagnostic protocol included OCT, computer perimetry, visual acuity testing, and ophthalmoscopy. Parameters of the RNFL, GCIPL, macula, and optic nerve head were analyzed.

Results. Patients with TON demonstrated significant RNFL thickening in the early stages of trauma, optic nerve head swelling, and GCIPL thinning, which correlated with impaired visual function. OCT proved to be highly effective for early diagnosis and monitoring of these changes.

Conclusion. OCT is a pivotal tool for assessing retinal and optic nerve damage in TON, enabling improved diagnostics, prognostication, and patient management.

Keywords: Traumatic optic neuropathy, blunt eye trauma, optical coherence tomography, RNFL, GCIPL,

diagnostics.

Introduction: Traumatic injuries to the retina and optic nerve, such as traumatic optic neuropathy (TON), impose a significant clinical and social burden, particularly in working-age individuals. These injuries often result in irreversible vision loss, emphasizing the critical importance of early diagnosis and effective treatment to improve outcomes. Advanced imaging techniques, such as optical coherence tomography (OCT), offer new possibilities for the early detection and monitoring of these injuries [1,5,11,12].

Early studies indicate that TON is characterized by degeneration of ganglion cells and their axons, leading to notable morphological changes, including thinning of the retinal nerve fiber layer (RNFL) and the ganglion cell–inner plexiform layer (GCIPL). These changes are strongly associated with visual impairments, such as reduced visual acuity, visual field defects, and impaired color perception. Research suggests that GCIPL is a more sensitive marker for early changes compared to RNFL, making it a critical tool for diagnosis and outcome prediction [2,3,6,8,10].

Moreover, OCT enables the detection of early signs of optic nerve head and peripapillary retinal edema, which are not always visible using conventional examination methods, such as ophthalmoscopy. Numerous studies have reported increased RNFL thickness and macular volume within the first days following eye trauma, underscoring the high sensitivity of this method and its essential role in identifying even minimal pathological changes [4,7,9,13,14].

Another crucial aspect is the ability to differentiate between types of TON, which helps optimize treatment approaches by accounting for the specific characteristics of each injury [4,9,13]. Combining OCT with functional tests, such as computer perimetry, provides a deeper understanding of the mechanisms underlying optic nerve damage, which is particularly relevant in the context of personalized medicine.

Thus, OCT not only enhances the accuracy of diagnosing retinal and optic nerve damage but also plays a vital role in monitoring and predicting outcomes. This allows clinicians to make more informed decisions regarding the management of patients with TON.

Purpose of the study

To evaluate the potential of optical coherence tomography for diagnosing and monitoring retinal and optic nerve changes in patients with traumatic optic neuropathy and to determine the significance of retinal nerve fiber layer and ganglion cell–inner plexiform

layer parameters for early detection and outcome prediction.

METHODS

The study included 40 patients (40 eyes) with contusion eye injuries and clear optical media, who underwent examinations at the Departments of Traumatology and Neurosurgery of the Multidisciplinary Clinic of the Tashkent Medical Academy.

The causes of injuries were diverse: physical altercations (30%), punches (25%), blunt objects (20%), road traffic accidents (15%), and sports-related injuries such as boxing, sparring, and ball impact (10%). The average age of the patients was 40.8 ± 14.5 years, with 28 men (70%) and 12 women (30%). The mean time from injury to optical coherence tomography (OCT) was 3.2 ± 1.7 days. A control group was comprised of 30 healthy individuals (20 men and 10 women), matched for age and gender.

Exclusion criteria included the presence of other ocular diseases affecting the function and structure of the optic nerve, severe systemic comorbidities (such as significant cardiovascular, respiratory, or digestive system disorders), and an age under 18 years.

All patients underwent a comprehensive examination, including visual acuity testing, ophthalmoscopy, standard perimetry, and central visual field testing using the Humphrey 24/2 program for computer perimetry. Additionally, magnetic resonance imaging (MRI) of the brain was performed to rule out associated injuries.

OCT was conducted using the Huvitz HOCT-1F/1 device (South Korea). The device features a resolution of 5–8 μm , a scanning speed of 26,000 A-scans per second, and a laser wavelength of 840 nm. The following parameters were assessed: retinal nerve fiber layer (RNFL) thickness, macular volume, optic nerve head (ONH) volume and area, and ganglion cell–inner plexiform layer (GCIPL). Standardized protocols such as “RNFL Thickness Average” and “Retinal Thickness/Volume Tabular” were applied.

Morphometric data of the injured eye were compared with those of the paired healthy eye and the control group using the Student's t-test. Differences were considered statistically significant at $p < 0.05$. The data obtained were used to evaluate the degree of morphological changes in the retina and optic nerve and to identify correlations with functional impairments.

RESULTS

The study revealed significant morphological changes

in the retina and optic nerve in all patients with TON caused by blunt trauma. These changes affected the RNFL, GCIPL, macular region, and optic nerve head. The findings are detailed below.

The average RNFL thickness in injured eyes was $114.2 \pm 11.1 \mu\text{m}$, which was significantly higher than that in the healthy paired eyes ($106.5 \pm 8.9 \mu\text{m}$, $p < 0.01$). The increase in RNFL thickness during the acute phase was associated with peripapillary edema triggered by traumatic impact. The most pronounced thickening was observed in the superior and inferior quadrants of the peripapillary area, where RNFL thickness reached $120.5 \pm 10.2 \mu\text{m}$ (superior quadrant) and $118.7 \pm 9.8 \mu\text{m}$ (inferior quadrant), compared to the corresponding values in healthy eyes ($112.3 \pm 8.7 \mu\text{m}$ and $110.4 \pm 9.1 \mu\text{m}$, respectively).

In the temporal quadrant, 42% of patients exhibited pallor of the optic nerve head, accompanied by a reduction in RNFL thickness in this region. The average RNFL thickness in the temporal quadrant of the injured eye was $88.5 \pm 6.9 \mu\text{m}$, which was below the normative value in the control group ($93.7 \pm 5.8 \mu\text{m}$, $p < 0.05$).

Early degenerative changes in the GCIPL manifested as thinning, even in the presence of RNFL edema. The average GCIPL thickness in the injured eye was $70.8 \pm 9.7 \mu\text{m}$, significantly lower than in the paired healthy eye ($78.2 \pm 7.1 \mu\text{m}$, $p < 0.05$). These changes suggest initial axonal degeneration caused by optic nerve trauma. The most significant thinning of the GCIPL was observed in the central and paracentral regions of the macula, correlating with functional impairments, such as reduced sensitivity in the central visual field.

Morphometric changes in the optic nerve head (ONH) included an increase in its volume and area, linked to trauma-induced edema and inflammation. The average ONH volume in the injured eye was $0.47 \pm 0.14 \text{ mm}^3$, significantly exceeding that in the healthy eye ($0.33 \pm 0.09 \text{ mm}^3$, $p < 0.001$). An increase in the neuroretinal rim area ($1.71 \pm 0.11 \text{ mm}^2$ versus $1.26 \pm 0.08 \text{ mm}^2$ in the healthy eye, $p < 0.01$) was accompanied by a reduction in the excavation area ($0.07 \pm 0.02 \text{ mm}^2$ versus $0.12 \pm 0.03 \text{ mm}^2$ in the healthy eye, $p < 0.05$).

In 32.5% of patients with blunt eye trauma, macular edema was observed, accompanied by an increase in macular volume. The average macular volume in the injured eye was $8.9 \pm 0.6 \text{ mm}^3$, compared to $7.5 \pm 0.4 \text{ mm}^3$ in the healthy eye ($p < 0.01$). Macular edema was more frequently seen in patients who sustained injuries from road traffic accidents and blunt object impacts.

To validate the significance of the changes in morphometric parameters, a comparative analysis was conducted with a control group of 30 healthy individuals. The data revealed statistically significant

differences in all key metrics for patients with TON. These included an 8–10% increase in RNFL thickness during the acute phase, a 9–12% reduction in GCIPL thickness, a 40% increase in ONH volume, and an 18% increase in macular volume.

In 40% of patients, pallor of the temporal half of the optic nerve head was observed, correlating with localized thinning of the RNFL. Additionally, 78% of patients showed changes in the central and paracentral macular zones, confirmed by computer perimetry results.

Morphological changes associated with traumatic optic neuropathy included peripapillary edema, an increase in optic nerve head volume, thinning of the GCIPL, and localized macular alterations. These findings underscore the high diagnostic value of OCT for early detection and monitoring of retinal and optic nerve changes in patients with blunt eye trauma.

Significant functional impairments of the visual system were detected in all patients with TON caused by blunt trauma. These impairments included decreased visual acuity, disruptions in peripheral and central visual fields, and reduced sensitivity in central and paracentral macular zones.

The majority of patients experienced reduced visual acuity in the injured eye. The average visual acuity was 0.5 ± 0.3 (Snellen scale), significantly lower than that of the paired healthy eye (1.0 ± 0.1 , $p < 0.001$). In 42% of patients, visual acuity ranged from 0.3 to 0.5, indicating moderate visual impairment. In severe cases, especially those caused by road traffic accidents or blunt object impacts, visual acuity ranged from 0.1 to 0.2, indicating significant damage to the optic nerve and retina.

Computer perimetry revealed a significant reduction in sensitivity in the central and paracentral zones in 82% of patients. The mean deviation in central field sensitivity ranged from -3.2 to -17.4 dB , depending on the severity of the trauma. Peripheral field constriction was noted in 65% of patients, predominantly in the superotemporal and inferotemporal sectors. Additionally, 30% of cases exhibited isolated color perception deficits, particularly in red and green, further confirming optic nerve fiber damage.

Central vision impairment was observed in 78% of patients. Humphrey 24/2 perimetry revealed reduced sensitivity in central and paracentral retinal areas, correlating with GCIPL thinning. In patients with the most severe central vision loss, GCIPL thickness decreased to $65.8 \pm 7.3 \mu\text{m}$, significantly lower than in patients with moderate impairments ($73.1 \pm 8.5 \mu\text{m}$, $p < 0.01$).

Relative afferent pupillary defects (RAPD) were noted

in 58% of patients in the injured eye, indicating substantial damage to optic nerve fibers responsible for transmitting visual signals.

Functional changes were closely correlated with the morphological parameters identified through OCT. Patients with pronounced reductions in visual acuity and visual fields demonstrated more severe thinning of RNFL and GCIPL, confirming a pathogenetic relationship between structural damage and functional impairments.

To benchmark functional impairments, visual function metrics were compared with the control group. Healthy individuals had an average visual acuity of 1.0 ± 0.0 , visual fields within age-appropriate norms, and central zone sensitivity ranging from -0.5 to 0.5 dB. These results confirm that the functional impairments in patients with TON were exclusively trauma-induced.

Functional impairments in traumatic optic neuropathy include reduced visual acuity, disruptions in peripheral and central visual fields, and afferent pupillary defects. These changes strongly correlate with morphological findings from OCT and highlight the importance of an integrated diagnostic approach that combines structural and functional analysis. Early detection of functional impairments allows for the prediction of optic nerve damage severity and timely initiation of therapy.

CONCLUSION

Traumatic optic neuropathy (TON) caused by blunt injuries leads to significant morphological and functional changes in the retina and optic nerve. This study demonstrated that optical coherence tomography (OCT) is a highly informative tool for diagnosing and monitoring these changes. In the early stages of trauma, OCT detects retinal nerve fiber layer (RNFL) and optic nerve head (ONH) edema, as well as initial degenerative changes in the ganglion cell–inner plexiform layer (GCIPL), which are critical markers of optic nerve damage.

Functional impairments, such as reduced visual acuity, disruption of central and peripheral visual fields, and relative afferent pupillary defects (RAPD), strongly correlate with morphological findings. This highlights the pathogenetic link between structural damage and functional deficits. Early detection of these changes using OCT not only allows for an objective assessment of injury severity but also facilitates outcome prediction, ensuring timely treatment and rehabilitation.

The findings confirm that OCT can serve as a gold standard for diagnosing and monitoring traumatic injuries to the retina and optic nerve. The method

provides detailed evaluations of RNFL thickness, GCIPL metrics, and macular parameters, paving the way for a personalized approach to managing TON patients. Further research with longer follow-up periods and larger patient cohorts is essential to refine diagnostic and therapeutic strategies for traumatic optic neuropathy.

Key findings

Patients with TON caused by blunt injuries exhibited significant alterations in the retina and optic nerve. During the acute phase, RNFL and ONH edema were observed, along with GCIPL thinning, indicative of early neurodegeneration.

Trauma resulted in reduced visual acuity, disruptions in peripheral and central visual fields, and relative afferent pupillary defects. These functional deficits strongly correlated with the identified structural changes, confirming their interrelationship.

OCT demonstrated high diagnostic accuracy in assessing traumatic optic nerve and retinal injuries. It provided precise measurements of RNFL thickness, GCIPL, ONH, and macular parameters, establishing its indispensability for early detection and monitoring of changes.

Using OCT during the acute phase of trauma enables the detection of pathological changes at stages when traditional diagnostic methods are insufficient. This facilitates timely therapy initiation and outcome prediction.

The results emphasize the need to integrate OCT into standard diagnostic protocols for patients with traumatic injuries to the eye and optic nerve. The method offers promising opportunities for a personalized approach to treatment and rehabilitation in TON patients.

To deepen the understanding of TON pathogenesis and enhance therapeutic strategies, further studies with larger patient cohorts and extended follow-up periods are needed. These investigations will help optimize diagnostic and treatment approaches, ultimately improving outcomes for patients with traumatic optic neuropathy.

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