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Optimizing diagnose for visual disturbances after head trauma in school children

*[1]**Victoria Verejan,** MD, PhD Applicant; [1]**Eugen Bendelic,** MD, PhD, Professor;
[2]**Jana Bernic,** MD, PhD, Professor

[1]Department of Ophthalmology, [2]Departmant of Pediatric Surgery, Traumatology and Orthopedics
Nicolae Testemitsanu State University of Medicine and Pharmacy, Chisinau, the Republic of Moldova

*Corresponding author: verejan_victoria@yahoo.com
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Abstract

Background: Despite the unprecedented evolution of medical science in recent decades, trauma, especially traumatic brain injury, is one of the most common and widespread injuries worldwide. WHO reports annually over 1 million deaths worldwide, which estimates a doubling of the rate of trauma during childhood, particularly those aged 7 to 18 years. Far from being addressed, the associated complications remain, such as disorders of the visual analyzer, other organs and systems. For this reason, the lesions of the visual analyzer are often a critical condition and a determining factor for the health of the future adult and for these reasons have an increased interest in the field of ophthalmology therapy. The diagnostic behavior is different, if we refer to children compared to the adult population, which requires a personalized, but also an objective, approach in order to assess the visual deficiencies.
Conclusions: The present paper considers the clinical aspects, corroborated with those specially selected in the certain diagnosis and treatment. The study reveals objective data, specially selected for the evaluation of a set of instrumental and laboratory investigations relevant to the age of 7-18 years. Determining an algorithm for diagnosing visual disorders that occur as a result of cranio-cerebral trauma in children will allow a relevant and objective examination to assess the treatment and behavioral tactics.

Key words: traumatic brain injury, visual disorders in children, ophthalmoscopy.

Introduction

The severity of traumatic brain injury (TBI) differs greatly depending on age. The child is not a mere reduced model of the adult. In children, brain trauma occurs on an immature brain. Anatomical differences: the larger head in relation to body mass, the absence of development of the cervical muscles and so on, facilitates the transmission of kinetic force to the brain. This aspect affects the child's response to trauma, as well as the prognosis. TBI causes a cascade of metabolic and inflammatory reactions with local and systemic consequences that lead to global cerebral ischemia with the development of cerebral edema [1]. Due to a vicious pathophysiological circle, as well as the fact that much of the brain structures are involved in the process of vision, they remain a problem far from being solved, testifies Singman EL [2]. Contusions or so-called cranio-cerebral traumas, as a rule, can induce both acute and chronic seizures [1, 3, 4]. This can be explained by the fact that a large part of the brain structures is involved in the process of vision [2]. The visual deficiency can be explained in several ways [5-7]. The mechanism of the visual changes is related to that of the trauma, which in turn can be produced by direct or indirect forces on the brain, affecting the white substance, according to the data of Cockerham GC et al. [8]. The white matter lesion occurs at the cellular and subcellular levels. Therefore, hemorrhage or organic lesions cannot be diagnosed even by the method of computed tomography or nuclear magnetic resonance, Suchoff IB et al. noted [9]. Brad P. Barnett et al. mentions that the age of the child plays an important role in the association of long-term sequelae, because the brain in its evolution is considered to be much more vulnerable, due to the sensitive blood-brain barriers, the elastic properties and the degree of myelination of the nervous tissue [10, 11].

Due to some peculiarities of the regulation of cerebral blood flow, the pediatric age group is subject to the development of intracranial hypertension (ICH), the cause of the development of which is the congestion (expansion, swelling) of the brain. Here the water content inside the nerve cell is increased as opposed to cerebral edema, where the water content is increased in the interstitial space. The incidence of brain congestion in pediatric patients is twice as high as in the adult population, resulting in a mortality of 46-53%. Diffuse cerebral edema occurs in over 35% of children aged 0-4 years and in over 40% of patients aged 4-16 years [1].

In children, the overall cerebral blood flow has a higher value (>100ml/100g/min) compared to that of the adult (30-40ml/100g/min), as well as the cerebral blood flow under physiological conditions in the child, which gives the brain better protection [1].

The treatment of post-traumatic visual impairments involves the use of visual rehabilitation therapies aimed at improving ocular convergence, accommodation and motility. Press L.J. et al. reports that 90% of patients mentioned improvement in visual symptomatology after applying visual therapy [12]. Similarly, there was an improvement, both clinical and statistical, of the accommodation, vision and visual attention [13, 14].

Most adults achieve a full neurological recovery in 1-2

weeks [15-17]. They may have prolonged symptoms and a long recovery period [18-22]. Approximately 50%-90% of adults who have had TBI have symptoms of vestibular impairment or ocular-motor dysfunction [23-26], but limited studies confirm the prevalence of vestibular-ocular disorders among children and teenagers [27, 28].

Notions of morphopathology

Based on the data presented by J. Johansson, the integrity of the vestibular, oculomotor and somatosensory systems is necessary to enable human beings to navigate and operate in a complex visuo-spatial environment [29]. This system is composed of specialized neural connections, which interact at different levels of the craniospinal axis to ensure the possibility of maintaining balance, of coordinating the movements of the eyeballs. The given system consists of sensory organs (retina, semicircular canals) and otolithic organs, as well as mechanoreceptors, having the ability to process primary information, which is then projected at the level of the spinal cord, CNS (basal, cerebellar, thalamus nuclei, cerebral cortex, basal ganglia) [30, 31]. The components of the given process include the vestibulo-spinal reflex (VSR), which determines the position of the head, neck and trunk during dynamic movements. Subsystem injury may result in injury to subsequent mechanisms. By this, it is sometimes possible to explain the exact topography of the lesion. The presence of vertigo, instability of balance, disturbance of vision, fog in front of the eyes are conclusive signs of VSR damage at different levels, and the morphopathological aspect cannot be fully elucidated. In the case of TBI, an important role in the installation of vertigo and dizziness is post-traumatic paroxysmal positional vertigo (PPPV), labyrinth contusion, perilimbal fistula, endolymphal hydrops, otolithic disorders and vestibular disorders in 46% of cases, according to data presented by J. Johansson [29]. Such accusations as, disturbed view, diplopia, reading difficulty are the result of dysfunction of accommodation, vision, insufficient convergence, visual field disorder and nerve paralysis, attests Brad P. Barnett and RE Ventura [10, 11]. Without a well-argued neuro-anatomical attitude regarding post-contusion disorder, visual rehabilitation could be compromised.

Wolf JA et al. have shown that axonal injury at the time of TBI occurs rarely upon impact [32]. More often, however, axonal stretching results in an irregular flow of ion transport, increased intra-axonal calcium ion concentration, and activation of the calcium proteolysis system (calpain proteolytic system). This promotes cytoskeletal proteolysis associated with irreversible axonal changes, in particular disassembly of the endoplasmic reticulum, notes Saatman KE et al. [32, 33].

Increased intra-cellular calcium concentration could lead to increased release of glutamate that activates N-methyl-D-aspartate receptors, and promotes depolarization of neurons, reports Barkhoudarian G et al. [34]. Damaged cells try to return to normal homeostasis by activating transport mechanisms. Increased cellular metabolism and glucose transport require numerous membrane pumps. Overloading of the given system leads to a depletion of energy sources, increased Ca influx into the mitochondria, impaired oxidative-basic metabolism, increased lactose glycolysis, and a final local acidosis as well as edema.

Evolutionary axonal inflammation becomes excessively severe and leads to a process called secondary axotomy. In patients with TBI, spectral magnetic, neurophysiological and electrophysiological data show that their recovery period can be 30-40 days, and in some patients it may take years, denotes Johnson EV [35]. Gardner RC emphasizes that, the age of patients plays an important role, as the developing brain is more vulnerable to traumatic action [36].

Visual dysfunctions after TBI can influence all the elements of the vision: visual acuity, accommodation, visual field, photosensitivity, color perception, contrast sensitivity, pupillary functions, sacadic movements, visual memory. Alteration symptoms may be present as a result of injury to the associated, efferent, or common region pathways, Singman EL and Brad P. Barnett mention [2, 10, 37].

Afferent injury after TBI can occur by a disturbance of visual acuity, contrast sensitivity and color perception. As a rule, these manifestations are bilateral. In the case of post-traumatic optic neuropathy or direct lesion of the orbit the symptoms may be unilateral. Direct trauma can be easily diagnosed with standard ophthalmological equipment. In patients with retro-bulb trauma, signs of proposis, ptosis, and decreased color perception will be suspected, according to Singman, E. L. and Brad P. Barnett [2, 10].

Visual dysfunctions may be one of the causes that would lead to poor patient integration after TBI in daily activity. The Rivermead Post Concussion Symptoms questionnaire is composed of a series of specific questions, which relate to the appreciation of visual function. The patient is asked to determine the severity of the visual symptomatology: how blurry the vision is or how clear the diplopia is. Follow-up studies in patients with average TBI and outpatients showed predominantly visual symptoms after three months, blurred vision 6.0-16.2% and diplopia 2.0-6.2% according to studies by Laborey M et al. [30]. Studies that included patients in the stage of subacute middle TBI show accommodation spasm 24.2-62.0%, convergence deficit 23.3-56.3%, and oculomotor deficit 6.0-51.3%, as Alvarez et al. mention [38]. Case-control studies that included patients with subacute mid-stage TBI and practically healthy patients revealed a higher prevalence of binocular and motor dysfunctions, Capo-Aponte et al. [39]. One of the progressive studies of the patient after the mean TBI determined the presence of an accommodative spasm 23.0% and a convergence deficit in 25.0%, Magone M. T. et al. [40]. Likewise, prospective researches assessing the dysfunction of the accommodation, as well as those regarding the assessment of the insufficiency of convergence, have determined that these are more pronounced in the patients who have suffered an average TBI, according to the data of J. Johansson [29].

Similarly, Magone M. T. et al. found situations in which the patients addressed the problem of difficulty in reading,

both during the subacute period of the trauma and over one year after the trauma [40].

Kapoor N. et al. determine a specific form of stimulation techniques for patients after TBI, called oculomotor visual rehabilitation (OVR). The latter uses combined techniques of motor training as well as the attention training to improve visual defects. Optometrists trained in the instucting patients to optimize divergence/convergence, fixation, saccadic movements choose to perform a certain spectrum of techniques that involve both motor and perceptual training. This complex of exercises helps patients determine not only the visual deficiency that is characteristic to them, but also the motor movements that could diminish them. Both manual machines to train these functions, as well as specialized computer programs can be used. Patients with concomitant strabismus and complex diplopic changes are preferred to be treated under the guidance of both an ophthalmologist and a neurologist/neurosurgeon. Disorders such as convergence/divergence and the accommodative reserve are sensitive to orthopedic treatment. However, when using such techniques, in a patient who has had a TBI, we must take into account the neurological symptoms that may include depression, fatigue, and concentration deficiency [41].

Afferent optical pathways deficits

Decreased visual acuity

The dysfunction of the afferent optic pathways could cause a decrease in visual acuity, color perception, light sensitivity and contrast sensitivity. Decreased post-traumatic vision may be omitted by the patient in case the condition is monocular. As a rule, the decrease in monocular visual acuity may be due to trauma to the orbit and the ocular, notes Cockerham GC et al. [8].

Data on visual acuity in patients after TBI are not clear and fully studied. One study shows visual acuity in patients after TBI between 6/30 and light perception at 13% and 1.6% respectively [42].

In his study, Richard A. Armstrong mentions that patients, who suffer from TBI, may experience a decrease in visual acuity that may persist for a long time [42].

Kenneth J. Ciuffreda et al. mention that, after a TBI, we could determine a growing myopia or hyperopia in the patient [43]. This seems paradoxal at first glance. Increased myopia can be explained by altered functioning of sympathetic NS, which occurs following a TBI. The disturbance of the function of the humoral systems of control of the curvature of the crystalline leads to an insufficient relaxation, in the case of distance sight, so that myopia becomes more accentuated.

On the other hand, an increase in the hypermetropic indicators could be explained by the altered functioning of parasympathetic NS, which may also occur following TBI. Thus, the ability to increase the accommodation to compensate for a residual hyperopia is compromised. That is why, a hyperopia that is not manifested becomes evident. This can also be characterized by the patient's sensation of transient fog in front of the eyes, which demonstrates a function of precarious parasympathetic NS.

These patients will not be directed to a progressive optical correction, but will choose a separate correction both at a distance and at close range. This fact can be explained by the hypersensitivity shown by patients to changes in the optical correction, a hypersensitivity that comes, in fact, from an alteration of the sensitivity caused by TBI [43].

The accommodative spasm, which indicates a pseudomyopia, as a rule seems to be associated with myosis or excessive convergence. Likewise, this spasm can appear not to be associated with anything being due to the psychogenic response [44].

The visual field

Suchoff IB mentions that visual field deficits are observed quite often after TBI. Usually, their presence signals a severe TBI, but they can also be detected in the case of an average TBI with the involvement of an optic chiasm or as a result of post-traumatic neuropathy [9].

Kenneth J. Ciuffreda et al. attest that defects in the field of vision usually refer to certain sectors, which are missing or seem to be sensory suppressed following the action of the trauma on the primary visual pathways. These areas can take shape from hemianopsies to small regions with reduced sensitivity. The symptoms in this case may be different, starting with accentuated visual difficulties up to minor visual effects. Visual field deficits were determined in 35% of the population with visual changes after TBI [43]. Some patients may benefit from using recessed prisms, such as Fresnel or Peli prisms, according to Ross NC et al. [45]. The training itself should include stimulation by luminous targets of both the deficient sector and the visual field. There are certain programs that improve the visual field by stimulating cortical function, by training the patient to better understand the visual field deficits or even to align their eyeballs to these deficits, according to data presented by Plow EB, Obretenova SN et al. [46].

In his study William V. Padula et al. determine an appearance of the visual deficiencies somewhat correlated with the anatomical area in which the tissue injury occurred afterwards in the TBI [47]. Thus, lesions that have included the visual tract damage above the optic chiasm can most often cause hemianopsy-like visual defects, but they are not always congruent. Depending on the affected area, the person may or may not be aware of the loss of visual abilities. The temporal lobe injury most often causes the appearance of an upper quadrant with oblique margins. Vision-spatial negation can be manifested either as a form of a total subjective loss of vision perception on the affected side or an objective lack of vision, notes Padula W. in his study on neurosensory rehabilitation questions of patients [47].

Richard Armstrong mentions that a number of studies reveal problems in the visual field in patients after TBI [42]. Patients with bitemporal hemianopsies reported by Padula JH et al., attributed to optic chiasm injury, were also attested [47].

Color perception, contrast sensitivity

R. Armstrong suggests that there are few studies that would talk about color perception in patients after TBI. However, data are found in the literature that would say that a case-control study of 11 patients after TBI and 11 control patients would suggest a poor perception of a primary color [42].

Lemke et al. determined that 21% of patients with TBI showed a low contrast sensitivity, which resulted in a low quality of life [48].

Efferent optical pathways deficiency

Accommodation

The accommodative dysfunctions (the accommodative step, the insufficiency of the accommodation and the inability to accommodate) can lead to an intermittent or constant blurred vision, depending on the severity of the injury. These come with the neurological changes of the trauma, but can affect other ocular manifestations.

Kenneth J. Ciuffreda et al. mentions that the accommodation disorder in patients who have had a TBI is manifested largely by a moderate impairment [43]. This seems controversial. A study in presbyopia and pre-presbyopia patients, who underwent TBI, elucidated by Thiagarajan P. et al. shows that the accommodation deficit will be determined in 24.4% and 41.1% respectively [26]. On the other hand, a case-control study of 50 patients after TBI and 50 control patients performed by Olver JH et al. determine equal numbers of the accommodation deficit [49].

Nystagmus

Nystagmus represents an oscillation of the eyeballs. This can be moderate or determined in case of major and wide oscillations. Usually, however, patients present with a slow, small amplitude nystagmus, which is accentuated if we cover an eye. Similarly, nystagmus may appear as a consequence of ophthalmoplegia. It can occur following the injury of the optical chiasm which stimulates certain pathological regions of the pituitary gland [30].

According to data presented by Geiger G. et al., which included 65 patients with central nystagmus following a TBI, favored by an extension of the spinal cord [23]. On the other hand, the patients who acted with a force that led to flexion-extension of the same sector presented associated vestibular and sensory disorders [42].

Scherer MR et al., in their research determined a pathological nystagmus with the feeling of dizziness in 50% of the patients exposed to TBI, and in the non-symptomatic patients the incidence was 33% [17].

Extra-ocular motility

The eye's motility system is highly sensitive to TBI, so the appearance of heterophoria is a common occurrence in patients after TBI. Patients who develop heterophoria most often will demonstrate diplopia. Skull muscles paralysis seems to lead to a deficiency of ocular motility commonly encountered [50]. The cranial nerves are very sensitive to TBI because their pathway is along the base of the skull. Unfortunately, the assessment of the motility of the eyeballs is difficult to establish in the first hours after the trauma because the patient is mostly often in a coma. Many signs, such as, for example, the third pair of cranial nerves are felt within a few months. Lagoftam may occur in patients after TBI due to a paresis of the facial nerve. In the case of fractures of the skull base the most frequent paresis of the nerve is that of the ipsilateral facial nerve of the motor neuron [51].

Deficiencies of oculomotor muscles can lead to binocular and accommodative dysfunctions that occur as a result of injury to the cranial nerves: CN III (oculomotor nerve), CN IV (trochlear nerve) and CN VI (abducens nerve), says Suchoff IB in his studies. But most of the time, the damage of one of the nerves is not obvious and can only be assumed. It is stipulated in the literature that computerized tomography, nuclear magnetic resonance do not cause changes [47].

As a consequence, the strabismus appears after a paresis of a cranial nerve or the injury of extraocular muscles, especially in case of damage of the integrity of the orbit. Esophoria or exotropia are also consequences of TBI. Binocular vision disorder often occurs after TBI, with the installation of latent force or fusion disorder [52]. One of the main accusations the patient has is diplopia. And the latter can occur even when the patient is ready to be discharged.

The World Health Organization estimates that strabismus accounts for 2-3% of the child population [36]; occurs more frequently at the age of 2-3 years. In 36% of cases, strabismus is complicated by amblyopia [53, 35]. In the Republic of Moldova, according to the statistical data and the annual activity reports, in the ocular nosological structure in the children taken under supervision, the strabismus holds the third place and constitutes 15-20% [54].

In general, it is proposed to evaluate the patient who has suffered TBI using the following aspects of ocular motility: fixation, amplitude of the saccades and the ability to track an object.

Stereognosia

The brain association systems together with the motor fusion are based on a mesencephalic network, where the oculomotor areas are located, as well as the areas corresponding to the view from the frontal lobes of the brain [55, 56].

A study shows that 10 patients, who have had a TBI, show an altered stereognosis at close and distant sight. Patients were investigated using stero-tests at distances of 3 m and 40 cm respectively. Thus, the data compared with those of the control patients did not show obvious differences in both the monocular and the binocular examination. The authors of the study Ciuffreda KJ et al. conclude that the disorder of stereognosis in post-TBI patients is not a problem of binocular perception, but a consequence of the disorder at a higher level associated with diffuse cortical injury [43, 57].

Convergence represents the movement of the medial eyeballs. This mechanism starts when patients look at an object closer than 5-6 m. A convergence deficit is one of the most common symptoms associated with TBI [50]. Thiagarajan et al. denotes the fact that 56.3% of patients who

have had a TBI attest some vision disorders [26]. Similarly Ciuffreda et al. points out that 42.5% of patients suffer from signs of insufficient convergence [43]. It is proposed to examine the patient using the cover test, to determine the proximity point of convergence, to evaluate the heterophoria according to the tactic Von Graefe, the amplitude of positive and negative fusion.

Pupillary reaction

Patients with TBI present a slow reaction of the pupillary response, as well as signs of anisocoria. The causes that could lead to the formation of a fixed and dilated pupil could be: transtentorial herniation (the sign of the Hutchin pupil), resembling the third pair of facial nerve, traumatic mydriasis or orbit fracture. In patients with TBI, they may have a narrow pupil, which may indicate the presence of Horner syndrome, traumatic myosis, pontine hemorrhage, and the Hutchinson pupil [52].

Kenneth J. Ciuffreda et al. notes that the assessment of pupillary reflex is important in the acute stage of TBI and could indicate the appropriate treatment options. According to a study in Portland, Oregon from 2012-2013 on a number of patients who had a TBI, 5 patients were diagnosed with pupillary reflex problems.

Similarly, a case-control study of 17 patients who had a TBI and 15 control patients revealed a difference in certain parameters: constriction velocity, maximum and average speed, maximum diameter and amplitude of constriction [43].

Papillary edema is a common neuro-ophthalmological complication. This occurs within the first 48 hours as a sign of intracerebral or extracerebral hemorrhage and is an absolute indication for surgery. If it appears after one week, it indicates a cerebral edema [52].

According to the literature data we find the notion of post-traumatic optic neuropathy (PTON) which has an incidence of about 0.7% -2.5% cases after a closed or open TBI. Its clinical picture is presented by signs such as decreased visual acuity, lack of color perception and color sensitivity, pupillary defect and a lack of changes at the back of the eye [58].

On the other hand, we find the same neuropathy, classified according to the anatomical sector involved: anterior PTON characterized by inflammation of the optic nerve papilla and posterior PTON with an unchanged image of the optic nerve papilla in the acute stage [58].

A traumatic injury of the optic nerve may occur as a result of a direct action of a bone fragment on it or it may be caused by an indirect mechanism of edema or ischemia. If we refer to the first variant, then we can say that a CT scan would help us determine the causative bone fragment. In case of ischemia or edema, an MRI examination would be more appropriate. In many cases the administration of high doses of corticosteroids would have a beneficial effect in case of worsening of tissue perfusion due to the indirect mechanism [30].

According to statistical data, the clinical picture of delayed PTON is more frequently encountered than that present in the acute stage of trauma. According to the International Optic Nerve Trauma Study (IONTS), the latter was determined in 13 cases of total patients investigated (approx. 10%), according to Levin La et al., delayed PTON appears due to a full spectrum of mechanisms [20]. Crowe et al. have described a clinical case of a patient who was accused of losing visual acuity on the 9th day after frontal TBI, as a result of secondary bleeding and inflammation of the optic nerve and optic chiasm [25]. Eidlitz-Markus et al. also described a clinical case of a 16-year-old patient who had symptoms of PTON delayed after 2 months of TBI with a blunt object [59]. The authors were not completely sure of the mechanism that triggered PTON, but the pale appearance of the optic nerve demonstrated an underlying ischemic mechanism. Likewise, Kay B. Kang et al. described a case of PTON delayed by the manifestation of a pale and edema optic papilla, with a triggering ischemic mechanism due to compression of the posterior ciliary artery, following inflammation of the medial right muscle [58].

From the point of view of an anatomical division we could say that the anterior PTON is more often encountered than the posterior one. Following a retrospective study by Goldenberg-Cohen, papillary edema was determined in 6 patients (15%) investigated [60]. Similarly, Brodsky et al. illustrated in the study three patients with previous PTON who had visual acuity of 1.0 0.1 respectively and light perception [61]. All three of these patients were previously hit by a blunt object in one eye. The patients were young people, more than that, two of them presented the so-called optical cup-less disk. Then the authors came up with the hypothesis that, in the case of an optic cup-less disk, the peripapillary inflammation of the sclera after a trauma, could induce the formation of a sclera channel, which in turn causes an interruption of the axonal transmission. This induces the formation of axonal conglomerates and, respectively, the inflammation of the optic disk [58].

Examination of the visually evoked potentials

The visually evoked potentials (VEP) refer to the electrophysiological signal arising from the neural activity correlated to the region corresponding to the visual cortex in response to a certain visual stimulus fixed in time [62, 63]. The primary activity recorded refers to the activation of the photoreceptor cones in the central area at 150 degree of the visual field [62, 64]. This corresponds to ~ 50% of the primary visual cortex [64, 65]. The registration of VEP activity was established as a method determined to assess objective and quantitative data in order to describe the integrity of the primary visual paths [66-69]. When applying a high luminescence stimulus and low temporal frequency, parvocellular pathways dominate in the processing of visual information [70], and on the other hand when using a low luminescence stimulus and high temporal frequency, the magnocellular pathways dominate. As a reference, in recent studies it has been shown that patients with TBI in the anamnesis have an increased coherent movement threshold, suggesting deterioration of the magnocellular pathways [71]. Thus, we could conclude that subjects with TBI in the anamnesis exhibit a

disturbance of the magnocellular pathways [72-74], which in turn would be responsible for processing the visual information with low luminescence, they hypothesize that the VEP data will be delayed in time and have decreased amplitude compared to control subjects [75].

Conclusions

1. A detailed clinical examination as well as a spectrum of neuro-ophthalmic investigations play a vital role in identifying the location and type of TBI lesion.

2. The mechanism for establishing visual disorders following a TBI is multifactorial. These can occur both as a result of a primary axonal injury induced by the force applied, as well as a secondary axonotomy induced by a primary ischemic process.

3. Taking into account the plasticity of the nervous tissue of the children the visual disorders can manifest themselves after the acute stage of TBI in the form of a post-contusion visual symptom.

4. The alternation of the refractometric data cannot be considered as the basis of an immediate optical correction, considering that these can be manifestations of transient pseudo-myopia or excessive accommodation.

5. The visual field defects could be an indication in the assessment of the anatomical area that was subjected to the lesion, highlighting over time the ability of the nervous tissue to restore its basic functions.

6. The long-term presence of disorders of fusion, convergence, tracking ability, stereognosis may be more pronounced in the pediatric population, as they may delay the restoration of the child's educational process.

7. Ophthalmoscopic changes in children are largely dependent on the degree of TBI, but these have a more pronounced tendency to manifest after the acute period of the disease in the form of delayed post-traumatic optic neuropathy.

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