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Visual acuity disturbances following brain injury in school-aged children

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Abstract

Background: Visual disturbances may result in a long-term complication after mild traumatic brain injury (mTBI) in children. These problems may affect both near work and reading, and thus affect activities of daily life and the child's return to school activity. The purpose of the study was to assess the visual acuity disturbances and refractive status in children with persisting symptoms after mild traumatic brain injury.
Material and methods: Forty-eight patients with persisting symptoms after mild traumatic brain injury and anomalies of visual acuity were included. Visual symptoms and refractive status were assessed during the eye examination.
Results: Thus, in the mTBI group the visual acuity for the right eye was of 0.09-0.5 in 83.7% (40 patients), in 16.3% (8 patients) – right eye 0.6-0.8, comparing to the control group, where 62% patients had the visual acuity ranged almost in 1.0, just 14% (7 patients) ranged 0.09-0.5 and in 24% (12 patients) – 0.6-0.8. The visual acuity for the left eye in the research group was of 0.09-0.5 in 89.8% (43 patients), in 10.2% (5 patients) – for the left eye was 0.6-0.8, comparing to the control group, where 66% patients had the visual acuity ranged almost in 1.0, just 24% (12 patients) it ranged 0.09-0.5 and in 14% (5 patients) – 0.6-0.8.
Conclusions: Visual acuity (VA) is affected primary after head trauma although it has big chances to get better with a vision therapy in a time period ranged between 3 and 6 months after the trauma. In most of the cases, we speak of a blurred vison in the near work and relative unclear perception at far. Autorefraction data usually will reveal a slight hyperopia with a possible astigmatic component ranged between 1D to 3D, and in 4.1%-8.2% of cases a slight myopia referring to the spherical compound and 18.4%-32.7% astigmatic compound, also ranged between 1D and 3D.
Key words: visual acuity, brain injury, children.

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Introduction

Traumatic brain injury is one of the most common causes of neurological morbidity, and is more often encountered in childhood and adolescence than at any other time of life [1-3]. Concussions in young people are usually diagnosed in about 90% of all traumatic brain injuries [4]. One in five children will experience a concussion by the age of 10 years [5]. As more frequent are referred falls (51%) and sports-related activities (25%) [5, 6]. The highest rates of sports-related concussion are reported in males aged 10–19 years, although young females are also involved [7, 8]. As speaking of the reported rate of concussion, contact football has the highest incidence, although all sports-related activities entail some risk [9].

Concussion is defined as a form of mild-traumatic brain injury that occurs because of a direct impact to the head or impact to the body that causes transmission of forces to the head and brain [10].

The pediatric brain has different mechanical and compositional properties (e.g. increased water content, decreased myelin, increased transition of acceleration-deceleration forces due to decreased neck strength). This increases the possibility for brain tissue displacement and shear injury [11, 12]. These properties can amplify the complicated neurometabolic cascade that comes after a concussion injury, resulting in increased vulnerability of the immature brain to secondary insults (e.g. second-impact syndrome) and more prolonged recovery [13-15]. As for the future, the prefrontal cortex, the region responsible for executive function, is particularly vulnerable to injury in adolescence [15, 16].

The visual consequences affect the input of visual information: it may prevent the patient from having clear and single vision at all distances. Poor visual impulse will undoubtedly affect visual information processing. Given the diffuse axonal injury often occurring with mTBI, damage to neurological pathways involving vision will influence negatively on the speed, accuracy, and sustaining ability to process and integrate visual information within a multisensory context [17].

A patient with mTBI presents with a constellation of general dysfunctions [18]. This is not surprising as referred to the global nature of the 2-phase brain insult that is

typical in mTBI [19]. In the first or primary phase, the immediate, biomechanically based response is installed and typical coup-contrecoup injury. This initially involves the cranial area and underlying brain tissue in the region of the direct external force (i.e., the coup). Then, due to the differential deceleration/acceleration inertial forces between the rigid cranium and the 2.5-pound jellylike brain mass, there is injury to the opposite brain pole region (i. e., the contrecoup). In addition, there are concurrent rotational, translational, and screw movements of the brain within the cranium, thus causing more brain contusion and damage (e.g., stretching), especially to the white matter fiber tracts, a key problem in mTBI. In addition, there is concomitant flexing and twisting of the highly susceptible midbrain region, especially in children, with this being a primary oculomotor control area. This primary phase is then followed by the secondary phase of the brain injury occurring from days to months afterward, with it being of a biochemically-based nature. It results in a network of events at the cellular level, thus producing cell damage and death, and related toxic events, to the brain and its environment. The degree of cellular insult during this secondary phase is crucial for the patient's recovery; the more the damage, the poorer the recovery [20]. Together, the comprehensive and global effects of the primary and secondary injury phases will produce abnormalities in the sensory, motor, perceptual, cognitive, attentional, behavioral, pharmacologic, somatic, and linguistic domains in many patients with TBI [18].

Speaking of primary patient care, more precisely visual function assessment, it is necessary to refer to the conceptual model of vision care in mTBI [18]. It has been developed as a pyramid scheme, being structured for an organized approach to the patient concerning vision assessment. Further, it will be necessary to clearly focus on the basic tier of this pyramid that states for basic vision examination, including basic refractive status, the general binocular/oculomotor status, and the ocular and general health status.

After mTBI, there can be found either increased myopia or new/increased hyperopia in a patient, which on first blush seems to be contradictory. This comes quite difficult to be explained as both of them may be presented. That is why it is important to try on building up models that would be able to explain the presence of both.

At the beginning, it was mentioned that the middle brain is the most sensible area for mTBI in children. It is known that the third cranial nerve (oculomotor nerve) contains parasympathetic nerve fibers that regulate the iris and lens of the eye. Its origin is in the Edinger-Westphal nucleus of the midbrain, afterwards preganglionic axons travel to the orbit and synapse on the ciliary ganglion. The ciliary ganglion contains two types of postganglionic neurons: one innervates smooth muscle of the iris and is responsible for pupillary constriction, and the other innervates ciliary muscle and controls the curvature of the lens [21]. The affirmation would be whether the stretching and twisting of this area would induce a prevalence of hyperopia in children after mTBI. The latter can be explained by an abnormally functioning parasympathetic system. Thus, the ability to increase accommodation to compensate for any residual, uncorrected hyperopia is compromised, and hence the latent hyperopia becomes manifest, perhaps with intermittent blur reflecting the ability to compensate only partially [18].

On the other hand, increased myopia can be explained by an abnormally functioning sympathetic system, common in mTBI, so that the pharmacologic control system of the crystalline lens cannot reduce "relax" accommodation fully and sufficiently with distant gaze, and thus increased myopia and blur become manifest. Sympathetic preganglionic neurons originate in the lateral horns of the 12 thoracic and the first 2 or 3 lumbar segments of the spinal cord [21]. Moreover, here comes the paradigm, since the spinal cord comes less many involved during mTBI why than should we confront with myopia in this case.

Traumatic myopia is a clinical entity that may be seen following ocular blunt trauma and is characterized with a usual range of -1.00 to -6.00 diopters (D) in the injured eye, or sometimes in both eyes. It is sudden onset and mostly transient, recovering within a few weeks after the trauma, although some cases may stand for a longer period. Possible causes that may lead to this condition are as follows: spasm of the ciliary body, increased crystalline lens effective power secondary to its forward shift, ciliochoroidal effusion causing forward displacement of the crystalline lens-iris diaphragm, axial thickening of the natural lens, and other sources of choroidal [22].

As to previous anatomy innervation peculiarities of the ciliary body, they found out that the ciliary body is also known to receive sympathetic innervation via long ciliary nerves [21]. And this would explain the possibility to confront with myopia after head injury.

Although increased accommodation appears to be uncommon in brain lesions, accommodative paresis is not. It has been reported in Wilson disease, encephalitis, and left parietal infarct or hematoma. Among patients with lesions of the dorsal midbrain, accommodative paresis may change with accommodative spasm. This suggests a linkage of the mechanisms involved in excess and deficient accommodation while brain stem damage is present. For example, some lesions may interfere with inhibition, while others interfere with activation of the accommodative portion of the parasympathetic (Edinger-Westphal) sub nucleus of the third cranial nerve. Accommodative spasm tends to occur in young individuals, because they have such strong accommodative reserve [23].

The mechanism of the accommodative spasm is uncertain. In cat models, accommodation is directed by a pathway from the lateral supra Sylvian cortex bilaterally to the ocular motor nuclei. Stimulation of this area also produces convergence and miosis, but accommodation may be selectively activated. Experimental accommodative spasm has not been demonstrated [23].

Material and methods

Forty-eight patients were referred to the Department of Emergency Unit of the State Mother and Child Health Care Institute, Chisinau, Moldova due to persisting visual symptoms after mild traumatic brain injury. The patients were examined for visual dysfunction primary in the first 72 hours after the trauma occurred.

As mTBI appears unpredictable, most of the patients were hospitalized in the first 6 h – 87.8% (43 patients), 40 children – 81.7% have been hospitalized more than 7 days, making possible a more complex examination. Visual acuity was measured in 48 traumatic brain injury patients. All studies used a Snellen chart/card or comparable metric to assess visual acuity. The measures noted a clear decreased visual acuity in the initial acute phase for both eyes after trauma (fig. 3-4).

The cycloplegic refraction is being evaluated individually after head trauma as mentioned by different authors. Hughes F.E. et al. mentioned that two drops of 1% w/v atropine sulphate administered into the patient's right eye provided immediate relief of the patient's visual symptoms in a 34-year-old female who developed sudden onset blurred distance vision after a rear impact car crash, having previously been emmetropic [24]. On the other hand another group of authors used in their clinical trial cycloplegic refraction evaluated with one drop of tropicamide 1% which was instilled every five minutes three times, and auto refraction was repeated 30 minutes after the last drop [22]. In addition, cycloplegic refraction performed by using cyclopentolate of 1% in a trial of 117 children with bilateral nasolacrimal duct obstruction has been reported in the review literature [25]. Due to the fact that, specific cycloplegic refraction used in neurological compromised patients has not been found in the review literature, or authors didn't mention a clear propensity for it, as for example in a trial of children with cortical impairment [26], it was decided to use the method of tropicamide 1% already used in this research.

As to the eligibility criteria the patients were divided into two groups. The first, research group, included patients that had undergone a mTBI in the last 72h and were hospitalized at the Mother and Child Health Care Institute. The patients were selected as reviewed the medical cards that demonstrated no visual disturbances before and no other chronic systemic pathology. The second group of patients was selected at the out-patient department of the Mother and Child Health Care Institute that presented with visual pathology including only refractive status disturbances with no other organic visual pathology. In addition, as reviewed, the medical cards demonstrated no visual disturbances before and no other chronic systemic pathology that may induce errors of objective examination.

Results

As to the age of the patients, teenagers boys were the most affected, age ranged between 15-18 years (45%), 11-14 years (25%) and school children age ranged 7-10 years (29%), (table 1-2). It can be outlined that most of the mTBI occur in teenagers followed by school children, while the children at the age from 11-14 years were less referred as being affected (x2= 3.412a, gl=2, p<0.01).

Table 1. Trial groups devided by sex

Sex	Research group		Control group	
	Patients	%	Patients	%
Boys	34	70.8	22	45
Girls	14	29.1	26	54

Table 2. Trial groups devided by age

Age	Research group		Control group	
	Patients	%	Patients	%
7-10 years	14	29.1	22	45.8
11-14 years	12	25	11	22.9
15-18 years	22	45.8	15	31.2

Referring to the type of trauma it may be observed that mTBI occurred mostly, being divided as localized trauma lesion in 40.8%, localized lymphatic lesions in 16.3%, cranium deformities in 1.3%, clear concussion in 16.3% and associated epidural hemorrhages in 10.2% of patients. The patients hospitalized with concussion were later re-evaluated and determined to have mTBI as diagnose. The natures of trauma were classified as following: falling from heights in 55.1%, vehicle accidents in 30.6%, falling objects in 8.2% and sport related in 6.1% of patients. For the patients involved in vehicle accidents the ophthalmologic examination was conducted later as the general status of the child was compromised. Speaking deficiency was determined in 36.7% (18 patients), while 63.3% (31 patients) – presented a clear, but delayed speech. A peripheral nervous system examination revealed an average disturbance in 40.8% (20 patients), while for 59.2% (29 patients) no problems have been determined. Pathologic reflexes were present in 38.8% (19 patients), (fig. 1).

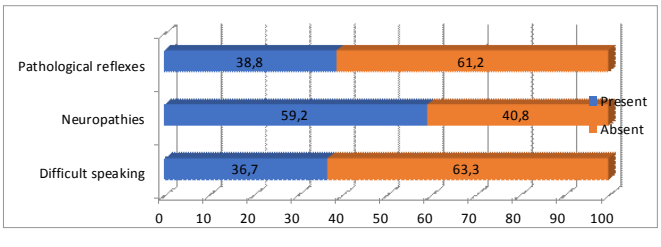

Fig. 1. Neurological findings

Cranial nerve examination, oculomotor (III), trochlear (IV) and abducens (VI), that are involved in eye motion and stability in 1/3 (15 patients) revealed late photoreaction and anisocoria. Mostly the changes were determined in the group of patients that underwent intracerebral hematoma evacuation. Ocular motility was decreased in most of the axes, with a lack of motion in case of patients presenting hematoma of the periorbital tissue.

Examination of general motility revealed a peripheral

paresis in 6.1% (3 patients), 57.1% (28 patients) had a complete peripheral motion, while in 18 patients (36.7%) it was not possible to evaluate.

Examination of the vestibular function underwent 25 patients since in the rest of the patients it was not possible to perform due to the unclear general state. Thus, positive results were determined in 79.2% (19 patients), in 8.3% (2 patients) – unstable results, in 12.5% (3 patients) – unstable results from left to right (fig. 2).

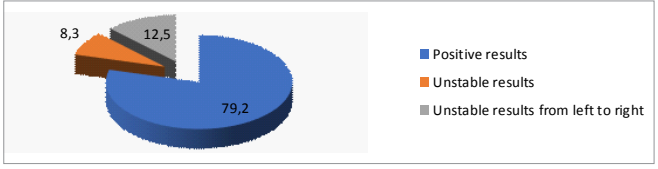

Fig. 2. Vestibular function examination

Thus, in the mTBI patients the VA for the right eye was 0.09-0.5 in 83.3% (40 patients), in 16.7% (8 patients) – AV OD 0.6-0.8, comparing to the control group, 60.4% (29 patients) had the VA ranged 0.9-1.0, just in 25% (12 patients) VA ranged 0.09-0.5 and in 14.6% (7 patients) VA was established between 0.6-0.8 (x2= 46.929a, gl=2, p<0.001) (fig. 3).

For the left eye were received the following results. Thus, in the mTBI patients the VA for the left eye was of 0.09-0.5 in 89.6% (43 patients), in 10.4% (5 patients) – VA for the left eye was 0.6-0.8, comparing to the control group, 62.5% (30 patients) had the VA ranged almost 1.0, just in 27.1% (13 patients) VA ranged 0.09-0.5 and in 10.4% (5 patients) – VA was established between 0.6-0.8 (x2= 51.281a, gl=2, p<0.001), (fig. 3).

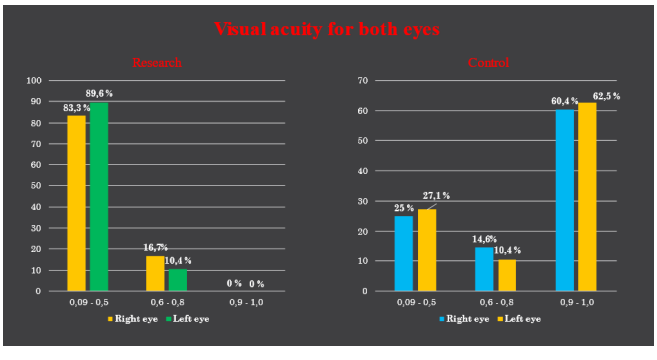

Fig. 3. Visual acuity following mTBI in children (research versus control) (%)

While examining patients in 4-6 months after the mTBI occurred were received the following numbers: VA for the right eye was 0.09-0.5 in 4.2% (2 patients), in 6.2% (3 patients) – AV OD 0.6-0.8, and 0.9-1.0 in 89.6% (43 patients) comparing to the control group, where 50% (24 patients) had the VA ranged 0.9-1.0, just in 25% (12 patients) VA ranged 0.09-0.5 and in 25% (12 patients) – 0.6-0.8 (x2= 46.929a, gl=2, p<0.001), (fig. 4).

For the left eye, were received the following results. VA was 0.09-0.5 in 4.2% (2 patients), in 8.3% (4 patients) – AV OD 0.6-0.8, and 0.9-1.0 in 87.5% (42 patients) comparing to the control group, 58.3% (28 patients) had the VA which ranged 0.9-1.0, just in 33.3% (16 patients) VA ranged 0.09-0.5 and in 8.3% (4 patients) – 0.6-0.8 (x2= 51.281a, gl=2, p<0.001), (fig. 4).

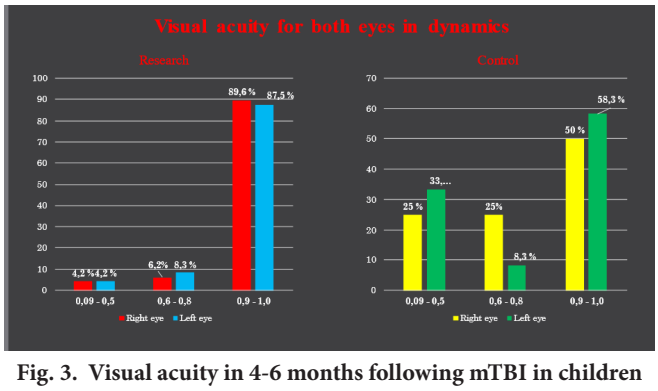

Fig. 3. Visual acuity in 4-6 months following mTBI in children (research versus control) (%)

Therefore, it ia easy to notice that mTBI patients present a clear visual deficiency occurrence as compared to the control group of patients.

Thus, after the measurements it may be concluded that 93.75% (45 patients), of the mTBI patients present hyperopic values for the right eye, comparing to the control group of hyperopic patients 70.8% (34 patients). In 6.25% (3 patients) of mTBI group was determined myopic values for the right eye comparing to 29.2% (14 patients) of the control group (x^2=9.523^a, gl=2, P <0.001). For the left eye there were the following hyperopic data in 95.8% (46 patients) for the mTBI group, compared to 66.7% (32 patients) – control group. For the left eye were received the following numbers concerning myopia – 33.3% (16 patients) in the control group, and 4.2% (2 patients) in the mTBI group (x^2= 15.682^a, gl=2, p <0.001).

For the astigmatic compound were received hyperopic values mostly. For the right eye there were the following values: hyperopic data in 68.75% (33 patients) in the mTBI group, and 31.25% – myopic data. As for the control group there were 56.25% of hyperopia and in 43.75% of patients – myopia (x^2= 0.924^a, gl=1, p <0.001), (tab. 3).

Table 3. Refraction status

Eye	Refraction (sph diopters)	Research		Control		x^2	gl	p
		Patients	%	Patients	%			
Right	0.00 till +3.00	45	93.75	34	70.8	9.523^a	2	<0.001
	0.00 till -3.00	3	6.25	14	29.2			
Left	0.00 till +3.00	46	95.8	32	66.7	15.682^a	2	<0.001
	0.00 till -3.00	2	4.2	16	33.3			

For the left eye were received the following values of hyperopic data: in 83.33% (40 patients) in the mTBI group, and 16.67% – myopic data, as for the control group – 56.25% hyperopia and in 43.75% of patients – myopia (x^2= 11.578[a], gl=2, p <0.001), (tab. 4).

Patients were re-evaluated after a period of time scheduled between 4-6 months after the trauma. The examination concerned refraction status for both groups.

The repeated measurements for refraction status revealed the following numbers: 75% (36 patients) of the mTBI patients present hyperopic values for the right eye, comparing to the control group of hyperopic patients – 45.8% (22 patients). In 25% (12 patients) of mTBI group were been determined myopic values for the right eye comparing to 54.2% (26 patients) of the control group (x^2=9.523[a], gl=2, p <0.01). For the left eye were obtained the following hyperopic values – 70.8% (34 patients) for the mTBI group, compared to 66.7% (32 patients) – control group. For the left eye were received the following myopic values – 33.3% (16 patients) in the control group, and 29.2% (14 patients) in the mTBI group (x^2= 15.682[a], gl=2, p <0.001), (tab. 5).

For the astigmatic compound were received hyperopic values mostly, for the right eye – hyperopic data 64.6% (31 patients) in the mTBI group, and 35.4%– myopic values. As for the control group there were 70.8% of hyperopia and in 29.2% of patients – myopia (x^2= 0.924[a], gl=1, p<0.05), (tab. 6).

For the left eye were received the following values of hyperopic data in 75% (36 patients) in the mTBI group, and 25%– myopic data. As for the control group there were 73% of hyperopia and in 27% of patients – myopia (x^2= 11.578[a], gl=2, p <0.05), (tab. 6).

Discussion

Visual dysfunction is a common occurrence after head trauma. Various aspects of vision are common to be affected as a consequence of a head trauma with many patients exhibiting multiple visual defects. These include aspects of primary vision, such as visual acuity which although not affected in all patients, can be a persistent deficit in some. The pediatric population is a quite difficult class of patients. The examination in this group may be affected along with the general status of the patient also the age inducing a non-cooperation patient, unable to clearly name the pictures or letters on the chart. The inability of children to self-assess and report symptoms after a brain injury can lead to the misdiagnosis of visual disturbance and a poor prognosis, and early diagnosis and proper treatments are keys to a better prognosis. Thus, early ophthalmologic examinations should be compulsory for children with head and face injuries.

As far as investigating the visual acuity loss in children the primary goal was to establish whether changes that appear may be considered permanent and important to be treated by vision therapy or glasses prescription. For that, it was essential to focus on the patient's primary vision concerns (inability/difficulty to read, draw, combine puzzle figures) and objective refractive data in order to reveal induced myopias /hyperopia by TBI. As it all comes from anatomical trails, the task was to explain whether a cause of the resulted myopia may be referred to the possible damaged pathways after a trauma. The afferent pathways that are coming from each optic nerve will eventually emerge into

Table 4. Refraction status

Eye	Refraction (cyl diopters)	Research group		Control		x^2	gl	p_1
		No abs	%	No abs	%			
Right	0.00 till +3.00	33	68.75	27	56.25	0.924[a]	1	<0.001
	0.00 till -3.00	15	31.25	21	43.75			
Left	0.00 till +3.00	40	83.33	27	56.25	11.578[a]	2	<0.001
	0.00 till -3.00	8	16.67	21	43.75			

Table 5. Refraction status in 4-6 months after mTBI

Eye	Refraction (sph diopters)	Research		Control		x^2	gl	p_1
		Patients	%	Patients	%			
Right	0.00 till +3.00	36	75	22	45.8	9.523[a]	2	<0.01
	0.00 till -3.00	12	25	26	54.2			
Left	0.00 till +3.00	34	70.8	32	66.7	15.682[a]	2	<0.001
	0.00 till -3.00	14	29.2	16	33.3			

Table 6. Refraction status in 4-6 months after mTBI

Eye	Refraction (cyl diopters)	Research		Control		x^2	gl	P_1
		Patients	%	Patients	%			
Right	0.00 till +3.00	31	64.6	34	70.8	9.523[a]	2	<0.05
	0.00 till -3.00	17	35.4	14	29.2			
Left	0.00 till +3.00	36	75	35	73	15.682[a]	2	<0.05
	0.00 till -3.00	12	25	13	27			

the visual cortex back to the occipital lobe. On the other hand, there are the efferent fibers that come from the frontal eye fields and synapse near the Edinger-Westphal nuclei. Anatomically the last ones are located in the immediate neighborhood for the oculomotor nuclei, and that is why even a mild trauma in this region could cause a lesion of the Edinger-Westphal nuclei [24]. The type of trauma can be also important in determining the kind of consequences one may face. For instance, whiplash type trauma has been reported on causing decreased accommodation, muscle paresis and even maculopathy [27, 28]. But some others declare unique cases of accommodation spam also present in this kind of trauma [24], thus making possible a different ophthalmologic outcome after mTBI. Data that reveal accommodative disfunction have been also reported by several other authors and this may involve accommodative insufficiency, accommodative infacility, or accommodative spasms that can cause a pseudo-myopia [29]. Most of the authors outline that in order to have an objective assessment of this issue an assessment of accommodative amplitudes, accommodative accuracy and accommodative facility should be done [30]. The role of the ophthalmologist in this case is very important because it should be the first one that starts a visual rehabilitation procedure. And this may involve prescribing glasses for reading or practicing rehabilitation visual exercises Management of accommodative disorders may include reading glasses with increased plus at near, or vision rehabilitation exercises [31, 32].

As to children, authors outline that in case of non-presbyopia patients, vision exercises are usually recommended as the first line treatment and may include accommodative lenses apply as well as accommodative push-up techniques. There is some evidence that 87-100% of patients with accommodative dysfunctions may show good results after with vision therapy [32].

Special mechanism that would define change in the refractive error has not been determined, although this group of patients may become more sensitive to small prescription changes or uncorrected refractive errors [33]. Special attention should be given to latent or uncorrected hyperopic patients, who may become symptomatic following a TBI, some of researchers declare [34, 35].

A Low-Concentration Atropine for Myopia Progression (LAMP) Study has revealed that 0.05, 0.025, and 0.01% atropine could prevent the progression of myopia [36]; although, there has not been any guideline for atropine concentration for accommodative spasm. Some of authors prescribed 1% atropine once a day and spectacle of +1.0 in both eyes to control the accommodation of patient with near reflex spasm [37]. While administered 1% atropine twice a day for 1 week with punctual occlusion has been reported to relax the accommodation of a patient with the spasm of near reflex [35, 38].

By analyzing the data, it may be outlined that in children there is a quite evident increase of hyperopia after head trauma, fact that may explain why most of the children complain of dizziness while reading, writing or even playing small toys. This may be for certain connected to convergence insufficiency that comes quite often after mTBI in children. The reason a hyperopia would be diagnosed in a child after TBI would be definitely connected with the altered function of the parasympathetic system and the impossibility to increase accommodation thus the latent hyperopia would become manifest. Moreover, this is one of the explanations found in resent studies although not referring strictly to children: the ability to increase accommodation to compensate for any residual, uncorrected hyperopia is compromised (e.g., slowed, delayed, ill-sustained), and hence the latent hyperopia becomes manifest, perhaps with intermittent blur reflecting the ability to compensate only partially [18]. There are small data that refer to visual acuity alteration in children and some of the researches claim that the clinical findings in some of the patients can be marginal and would not necessarily prompt spectacle treatment in healthy subjects [39]. Despite this, the spectacles may appear to provide a subjective relief. This appears to confirm with previous clinical observations that patients acquired brain injury may be hypersensitive to even low degrees of refractive error and binocular functional anomalies [40].

Received findings suggest that visual-vestibular processing deficits are present sub acutely following mild traumatic brain injury. Brain injury occurs frequently in children mostly affecting teenagers and early school children. It is true that little is known about the vision effects that may occur and the time prognosed for them to resolve. Although it may be assumed that brain plasticity in younger population is keener, there is still less information regarding time period and gravity of visual disturbances that may occur. As undergoing the basic ophthalmologic examination, it may be concluded that this study revealed that ocular manifestation almost all the time occurs in head trauma in children. The severity evaluation of these changes is compromised quite often since children become unable to co-operate due to the general state or the psychological status after the trauma (anxiety, marked phobias). Visual acuity is affected primary after head trauma although it has big chances to get resolved with a vision therapy in time period ranged between 4 and 6 months after the trauma. In most of the cases, we speak of a blurred vision in the near work and relative unclear perception at far. Autorefraction data usually will reveal a slight hyperopia with a possible astigmatic component ranged between 1D to 3D, and in a few cases a slight myopia also ranged between 1d and 3D. The reason this occurs can be explained by a latened activation of both sympathetic and parasympathetic systemic inducing ciliary process, local changes and misalignment of lens due to its increased/decreased curvature after trauma. Fewer patients require glasses correction since their return to school is delayed due to neurological status. Although it may be considered prescription for the near work optic correction. As for the future, new research data may have an important educational impact for these children and their parents, as well as for school personnel; for example, the development of return-to-learn school criteria.

Conclusions

1. Visual acuity disturbance can be commonly experienced in children after mTBI being ranged below 0.5 as referred to the Snellen chart in up to 83.3%-89.6% cases in the first 24-72 hours. However, it can be considered as being a transient situation since it becomes improved with no particular therapy in about 4-6 months after head trauma in 89.6%-87.5% in up to 0.8-1.0 as referred to the Snellen chart.

2. Exacerbated hyperopia is mostly encountered in children after head injury in the acute phase ranging from 93.75%-95.8% for the spherical compound as low as +3.00D and 68.75%-83.3% for the astigmatic compound. This issue can be explained by an accomodative disfunction since most of the patients complained of difficulties while reading and near work blurred perception. As going through time in 4-6 months after mTBI it may be outlined that hyperopia persists in almost 70.8%-75% for the spherical compound as low as +3.00D and 64.6%-75% for the astigmatic compound.

3. Induced myopia can be less determined in children after head injury in the acute phase ranging from 4.2%-6.25% for the spherical compound as low as -3.00D and 16.67%-31.25% for the astigmatic compound. As going through re-evaluation in 4-6 months after mTBI there are data that myopia persists in almost 25%-29.2% for the spherical compound as low as -3.00D and 25%-35.4% for the astigmatic compound. The entity of post mTBI myopia is still discussed between hypothesis of ciliochoroidal effusion, change of the iris-lens diaphragm or accomodation spasm.

4. Refraction state after mTBI in children should be re-evaluated since it has a passing character. Glasses prescription ahould be done carefully being related to the objective disturbance a child may have at near work or visual perception in the far.

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VV conceptualized the project and drafted the first manuscript. EB interpreted the data. JB critically revised the manuscript. All authors revised and approved the final version of the manuscript.

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Conflict of Interests
No competing interests were disclosed.