VISUAL ACUITY AND EYE REFRACTION DISTURBANCES FOLLOWING BRAIN INJURY IN SCHOOL-AGED CHILDREN

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Summary

Visual disturbances may result in a long-term complication after mild traumatic brain injury(mTBI) in children. The purpose of the study was to assess the visual acuity (VA) disturbances and refractive status in children with persisting symptoms after mild traumatic brain injury. The research aimed 48 patients with persisting visual symptoms after mTBI. Visual symptoms and refractive status were assessed. 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, compared to the control group, were 62% of patients had the visual acuity ranged almost in 1.0, just 14% (7 patients) was ranged 0.09-0.5 and in 24% (12 patients) - la 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, compared to the control group, were 66% of patients had the visual acuity ranged almost in 1.0, just 24% (12 patients) was ranged 0.09-0,5 and in 14% (5 patients) – 0,6—0.8. VA is affected primarily after head trauma although it improves in a time perioranginged between 3 and 6 months after the trauma. Autorefraction data usually will reveal slight hyperopia with a possible astigmatic component ranging between 1D to 3D, and in 4.1%–8.2% cases slight myopia also ranged between 1D and 3D.

Keywords: *visual acuity, brain injury, children*

Rezumat

Dereglări ale acuității vizuale și refracției în cazul copiilor după traumatism cranio-cerebral

Afecțiunile vizuale la copii pot surveni în urma unui traumatism cranio-cerebral (TCC). Scopul studiului a fost de a determina incidența dereglărilor vizuale apărute în urma unui TCC în cadrul populației pediatrice. Studiul a inclus 48 de pacienți cu afecțiuni vizuale persistente post TCC. A fost examinată acuitatea vizuală (AV) și refracția. Astfel, în cadrul lotului de pacienți post TCC, AV pentru ochiul drept a fost de 0.09-0.5 la 83.7% (40 de pacienți), la 16.3% (8 pacienți) – ochiul drept 0,6-0,8, comparativ cu lotul de control, unde 62% dintre pacienți aveau AV de aproximativ 1.0, doar 14% (7 pacienți) au prezentat 0.09-0.5 și la 24% (12 pacienți) - la 0.6—0.8, conform tabelei Snellen.

AV pentru ochiul stâng în lotul cercetat a fost de 0.09—0,5 la 89.8% (43 de pacienți), la 10.2% (5 pacienți) – 0.6—0.8, în comparație cu lotul de control, 66% de pacienți au prezentat AV de aproape 1.0, doar 24% (12 pacienți) au prezentat 0.09-0,5 și 14% (5 pacienți) – 0,6—0.8.

AV este afectată în primul rând după un TCC, deși se ameliorează simțitor în decurs de 3-6 luni după traumatism.

Datele autorefractometriei arată o ușoară hipermetropie cu o posibilă componentă astigmatică cuprinsă între 1D și 3D, iar în 4.1%–8.2% din cazuri o ușoară miopie care, de asemenea, variază între 1D și 3D.

Cuvinte-cheie: *acuitate vizuală, traumatism cranio-cerebral la copii*

Резюме

Нарушение остроты зрения и рефракции при черепно-мозговой травме у детей

Зрительные осложнения при черепно-мозговой травме у детей могут появится как в остром, так и в более позднем периоде. Целью исследования было оценить нарушения остроты зрения (ОЗ) и рефракционного статуса у детей с сохраняющейся симптоматикой после черепно-мозговой травмы.

В исследование были включены 48 пациентов с сохраняющимися зрительными симптомами после перенесенной ЧМТ. Оценивались зрительные симптопы и рефракционный статус. Таким образом, в группе пациентов с ЧМТ острота зрения для правого глаза составила 0.09—0. 5 ед. у 40 больных (83.7%), у 8 больных (16.3%) – 0.6-0.8 ед., по сравнению с контрольной группой, где у большинства больных (62%) острота зрения составила почти 1.0 ед., только у 14% (7 больных) – ОЗ была в пределах 0,09-0,5 ед., и у 24% (12 больнных) – 0,6-0,8 ед. Острота зрения левого глаза в основной группе составила 0.09—0,5. ед у 89.8% (43 пациентов), у 10.2% (5 пациентов) – 0.6—0.8 ед., а в контрольной группе, у 66% острота зрения составила 1.0 ед., у 24% (12 пациентов) - 0.09-0,5 ед. и у 14% (5 пациентов) – 0,6—0.8 ед. Острота зрения снижается сразу после черепно-мозговой травмы, и улучшается через 3-6 месяцев после травмы.

Данные авторефракции обычно выявляют легкую гиперметропию с астигматическим компонентом в пределах 1D-3D, а в 4.1%–8.2% случаев легкая миопия также находится в диапазоне от 1D до 3D.

Ключевые слова: *острота зрения у детей, черепно-мозговая травма*

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]. Over the last several decades' sport-related concussions have been recognized as a major health concern in young athletes, being a public health problem [7,8]. 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].

A patient with mTBI presents with a constellation of general dysfunctions [17]. This is not surprising as referred to the global nature of the 2-phase brain insult that is typical in mTBI [18]. This initially involves the cranial area and underlying brain tissue in the region of the direct external force (i.e., the coup). 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 [19]. 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 [17].

Eye tracking correlated with concussion symptoms and detected convergence and accommodative abnormalities associated with concussion in the pediatric population. It demonstrates utility as a rapid, objective, noninvasive aid in the diagnosis of concussion [21].

In the beginning, we mentioned that the middle brain is the most sensible area for mTBI in children. 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 [20]. 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 [17]. 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 [20]. And here comes the paradigm, since the spinal cord comes less more 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 [23]. As we go back to anatomy innervation peculiarities of the ciliary body, we can find out that the ciliary body is also known to receive sympathetic innervation via long ciliary nerves [20]. And this would explain why we can confront with myopia after head injury.

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. Accommodative spasm tends to occur in young individuals because they have such strong accommodative reserve [22].

The aim of the study is to determine the entity and incidence of visual acuity loss and eye refraction disturbance after mTBI in children.

Materials and methods

Forty-eight patients had been referred to the Department of Emergency Unit of the State Mother

and Child Health Care Institute from 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.

Results

As mTBI appears unpredictable most of the patients have been hospitalized in the first 6 h- 87.8% (43 patients), that is 98%. Of the patients, 81.7% (40 children) have been hospitalized for 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 et al. (2017) mentioned that two drops of 1% w/v sulfate 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 of blurred distance vision after a rear impact car crash, having previously been emmetropic [39].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 for three times, and auto refraction was repeated 30 minutes after the last drop [23]. Also, 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 [40]. 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, in which all children underwent a complete neuro-ophthalmologic examination including VA, cycloplegic refraction, and sensorimotor testing [41]. Thus, we decided to use the method of tropicamide 1% already used in our research.

Results As we refer to the age of the patients, we may outline that teenagers' boys have been the most affected, age ranged between 15-18 years-45%, also 11-14 years 25% and school children age ranged 7-10 years 29.2% (table 1-2). We may outline that most of the mTBI occur in teenagers followed by school children, while the children with the age ranged from 11-14 years has been less referred as being affected (x2= 3.412a, gl=2, p<0.01).

Referring to the type of trauma we may outline that mTBI occurred mostly, being divided as localized trauma lesion in 39.6%, localized lymphatic lesions in 18.7%, cranium deformities in 14.6%, clear concussion in 16.7% and associated epidural hemorrhages in 10.4%. The patients hospitalized with concussion have been later re-evaluated and determined to have mTBI as diagnose. The natures of trauma have been classified as following: falling from heights 54,2%, vehicle accidents in 31.2%, falling objects in 8.3% and sport related in 6.3%. For the patients involved in vehicle accidents, the ophthalmologic examination has been undergone later as the general status of the child was compromised.

Speaking deficiency has been determined in 37.5% (18 patients), while 62.5% (30 patients) – presented a clear, but delayed speech. A peripheral nervous system examination revealed an average disturbance of 41.7% (20 patients), while for the 58.3% (29 patients) no problems have been determined. Pathologic reflexes have been present in 39.6% (19 patients).

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 have been determined in the group of patients that have been involved in intracerebral hematoma evacuation Ocular motility has been 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 6.3% (in 3 patients), in 56.2% (27 patients) had a complete peripheral motion, while in 18 patients (37.5%) it was not possible to evaluate.

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

Thus, in the mTBI patients the VA for the right eye was of 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, were 60.4% (29 patients) had the VA ranged 0.9-1.0, just 25% (12 patients) VA was 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 we had 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, were 62.5% (30 patients) had the VA ranged almost in 1.0, just 27.1% (13 patients) VA was 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).

While examining patients in 3-6 months after the mTBI occurred we had the following numbers: VA for the right eye was of 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 25% (12 patients) VA was ranged 0.09-0.5 and in 25% (12 patients) - la 0.6-0.8 (x2= 46,929a, gl=2, p<0,001) (fig. 4).

For the left eye we had the following results. VA was of 0.09-0.5 in 4.2% (2 patient), 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, were 58.3% (28 patients) had the VA ranged 0.9-1.0, just 33.3% (16 patient) VA was ranged 0.09-0.5 and in 8.3% (4 patients) - la 0.6-0.8 (x2= 51,281a, gl=2, p<0,001).

So, we may notice that mTBI patients present a clear visual deficiency occurrence as compared to the control group of patients.

Thus, after the measurements we may conclude that most mTBI patients present hyperopic values for both eyes (Table 1).

Table 1

Refraction status

Eye	*Refraction (sph diopters)*	*Research*		*Control*		x^2	*gl*	*p*
		Pa-tients	%	Pa-tients	%			
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 astigmatic compound we may outline that we received hyperopic values mostly (Table 2).

Table 2

Refraction status

Eye	*Refraction (cyl diopters)*	*Research group*		*Control*		x^2	*gl*	p_1
		Nr. abs	%	*Nr. 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			

Patients have been re-evaluated after a period of time scheduled between 3-6 months after the trauma. The examination concerned refraction status for both groups.

The repeated measurements for refraction status revealed a more average spread for values (Table 3).

Table 3

Refraction status in 3-6 months after mTBI

Eye	*Refraction (sph diop-ters)*	*Research*		*Control*		x^2	*gl*	p_1
		Pa-tients	*%*	*Pa-tients*	*%*			
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			

For the astigmatic compound we may outline that we received hyperopic values mostly (Table 4).

Table 4

Refraction status in 3-6 months after mTBI

Eye	*Refraction (cyl diopters)*	*Research*		*Control*		x^2	*gl*	$P1$
		Pa-tients	*%*	*Pa-tients*	*%*			
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			

Discussions

The examination in children 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 our 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, we tried on focusing 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, we tried on explaining 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 the visual cortex back to the occipital lobe. On the other hand, we have 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 we may face. For instance, whiplash-type trauma has been reported on causing decreased accommodation, muscle paresis, and even maculopathy, along with jaw-neck function affection [25.26]. 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 dysfunction 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 [27]. With a mild spasm, the patient may be making themselves pseudo-myopic with blurred vision [28]. 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 [29.30]. As we refer 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 [30].

Treatment may include tinted lenses and overlays, corrective and prismatic lenses, vision therapy and rehabilitation [32]. Special attention should be given to latent or uncorrected hyperopic patients, who may become symptomatic following a TBI, some of researchers declare [31]. Ophthalmologists should always remain suspicious of nonorganic visual loss in case there is inconsistency between symptoms and findings [36].

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 [33]; although, there has not been any guideline for atropine concentration for accommodative spasm. Some of authors [34] prescribed 1% atropine once a day and spectacle of +1.0 in both eyes to control the accommodation of patient with near reflex spasm. While administered 1% atropine [35] 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.

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 [17]. There are small data that refer to visual acuity alteration in children and we would definitely agree with some of the researches that claim that the clinical findings in some of the patients can be marginal and would not necessarily prompt spectacle treatment in healthy subjects [37]. Despite this, the spectacles may appear to provide a subjective relief. This appears to confirm with previous clinical observations that patients with acquired brain injury may be hypersensitive to even low degrees of refractive error and binocular functional anomalies [38].

Conclusions

Visual acuity disturbance can be commonly experienced in children after mTBI being ranged below 0.5 as referred to the Snellen chard in up to 83.3%-89.6% cases in the first 24-72 hours. But 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.

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 accommodative disfunction since most of the patients complained of difficulties reading and near work blurred perception. As going through time in 4-6 months after mTBI we may outline 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.

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 we may outline 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 a hypothesis a ciliochoroidal effusion, chanke of the iris-lens diaphragm or accommodative spasm.

Refraction state after mTBI in children should be re-evaluated since it has a passing character. Glass prescription should 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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