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Clinical and Functional Characteristics of Visual Functions in Children with Partial Optic Nerve Atrophy

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Abstract Partial optic nerve atrophy (PONA) represents a progressive neurodegenerative disorder characterized by irreversible damage to optic nerve fibers and subsequent visual impairment [1]. In pediatric populations, PONA is most commonly associated with intracranial tumors, non-neoplastic cerebrovascular diseases, traumatic brain injury, hydrocephalus, and infectious-inflammatory diseases of the central nervous system. Despite advances in neuro-ophthalmology, therapeutic options remain limited and are largely aimed at stabilizing visual function rather than restoring it [2-4]. This study evaluates the clinical and functional characteristics of visual impairment in children with PONA of various etiologies and assesses the effectiveness of combined conservative therapy including transcranial magnetic stimulation (TMS). Forty pediatric patients (80 eyes) were followed for two years. Results demonstrated statistically significant improvement in visual acuity and electrophysiological parameters in patients receiving combined therapy compared to drug therapy alone. The findings support the inclusion of TMS in complex rehabilitation programs for pediatric PONA.

Keywords Partial optic nerve atrophy, Children, Visual functions, Visual acuity, Visual field, Visual evoked potentials (VEP), Optic coherence tomography (OCT)

1. Introduction

Partial optic nerve atrophy (PONA) in children is a chronic, progressive optic neuropathy characterized by degeneration of retinal ganglion cell axons and subsequent thinning of the optic nerve head. It represents the end stage of various pathological processes affecting the anterior visual pathway [4,5]. Unlike congenital optic nerve hypoplasia, partial atrophy develops after a period of previously normal or relatively preserved optic nerve structure and function. The condition is clinically manifested by decreased visual acuity, visual field defects, impaired contrast sensitivity, and characteristic ophthalmoscopic findings such as optic disc pallor, attenuation of retinal vessels, and reduced neuroretinal rim volume. Optic nerve atrophy accounts for approximately 7–12% of pediatric visual impairment cases worldwide and remains one of the leading causes of irreversible childhood blindness [5,7,9]. The incidence varies by region due to differences in access to perinatal care, neuroimaging, and early diagnosis of intracranial pathology. In tertiary pediatric ophthalmology centers, up to 20–25% of children referred for unexplained vision loss are ultimately diagnosed with some form of optic neuropathy, including partial atrophy. The risk of developing optic atrophy increases significantly in children with delayed treatment of intracranial hypertension, prolonged papilledema, or repeated inflammatory episodes affecting the optic nerve [6,8]. The pathogenesis of PONA involves axonal degeneration of retinal ganglion cells secondary to mechanical compression, ischemia, inflammation, or metabolic dysfunction. Chronic elevation of intracranial pressure leads to impaired axoplasmic flow, resulting in papilledema and subsequent optic nerve fiber loss if untreated. In ischemic mechanisms, reduced perfusion of the posterior ciliary arteries compromises the microcirculation of the optic nerve head [11,13]. Experimental data suggest that neuronal apoptosis plays a central role in disease progression. Once axonal damage exceeds a critical threshold (approximately 40–50% fiber loss), spontaneous recovery becomes unlikely due to limited regenerative capacity of central nervous system neurons. This explains why therapeutic strategies are primarily aimed at neuroprotection and functional stabilization rather than structural restoration [12]. Children with PONA present with variable degrees of visual dysfunction depending on etiology and duration of the underlying condition. Visual acuity at diagnosis ranges widely, from mild reduction (0.6–0.8 decimal) to profound impairment (≤0.05). Bilateral involvement occurs in

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approximately 60–70% of cases, particularly in systemic or intracranial causes. Clinical studies in pediatric populations demonstrate that nearly half to more than half of treated children experience improvement in visual acuity. On average, visual acuity shows a modest but clinically meaningful increase following a course of approximately ten to fifteen treatment sessions. Electrophysiological assessment reveals a noticeable rise in visual evoked potential amplitude, accompanied by a measurable shortening of P100 latency, indicating enhanced conduction along the visual pathways. In addition, long term follow up shows stabilization of retinal nerve fiber layer thinning, with progression significantly slowed or halted over a period of one to two years.

Aim of research

To study the features of visual outcomes of partial optic nerve atrophy of various etiologies in children during follow-up.

2. Materials and Methods

A total of 40 pediatric patients, representing 80 affected eyes, diagnosed with partial optic nerve atrophy were included in the study. All patients received examination and treatment at the Ophthalmology Department of the Clinic of the Tashkent Pediatric Medical Institute, as well as at the Republican Scientific and Practical Medical Center of Neurosurgery. All patients underwent a comprehensive diagnostic evaluation that included both ophthalmological and neurological assessment to ensure accurate diagnosis and identification of underlying etiological factors. The ophthalmological examination began with the measurement of best corrected visual acuity using standard visometric methods. Visual field testing was performed through both static and kinetic perimetry to assess the extent and pattern of visual field defects. Anterior segment structures were evaluated by biomicroscopy, while detailed examination of the optic nerve head and retina was carried out using direct and indirect ophthalmoscopy with photographic documentation for objective monitoring over time. Instrumental and laboratory investigations were conducted to determine possible intracranial or systemic causes of partial optic nerve atrophy. Neuroimaging studies included magnetic resonance imaging or multislice computed tomography of the brain to identify structural abnormalities, compressive lesions, or post traumatic changes. Functional assessment of the visual pathway was performed using visual evoked potentials, allowing evaluation of conduction along the optic nerve and central visual pathways. In addition, all patients underwent standard laboratory testing in accordance with pediatric clinical guidelines. A multidisciplinary approach was applied in the management of all children. Each patient was evaluated by a neurologist and a pediatrician to assess general and neurological status. When sedation or specific procedures were required, consultation with an anesthesiologist was also obtained to ensure safety and appropriate peri procedural care.

3. Results and Discussion

The study population consisted of an equal distribution of boys and girls, with 20 (50%) male and 20 (50%) female patients, ensuring balanced gender representation. The age of the participants ranged from early childhood to late adolescence, specifically from 3 to 17 years. The avarage age of the patients was 11 years, with a standard deviation of less than one year, reflecting a relatively homogeneous age distribution. All patients were followed prospectively for a period of 2 years, allowing for comprehensive evaluation of clinical dynamics, functional outcomes, and structural changes over time.

4 principal etiological groups were identified among the examined pediatric patients.

The largest proportion of cases was represented by first group, which included children with non neoplastic cerebrovascular diseases, accounting for 23 patients or 57,5% of the total patients. This group comprised arteriovenous malformations, aneurysms, cavernous angiomas, venous malformations, and fistulas. Inclusion criteria for this category consisted of a history of surgically treated non neoplastic cerebrovascular pathology, symmetrical decrease in visual acuity, pediatric age, and confirmed presence of partial optic nerve atrophy. The second group consisted of patients with post traumatic partial optic nerve atrophy and included 9 children, representing 22,5% of the study population. Among them, 6 cases were associated with orbital fractures accompanied by direct injury to the visual pathways, while 3 cases followed severe traumatic brain injury without direct structural damage to the optic pathways. The third group comprised 5 patients, corresponding to 12,5% in whom partial optic nerve atrophy developed in the context of hydrocephalic hypertensive syndrome. The fourth group included other less common causes and accounted for 3 patients in total. 2 children, or 5%, were diagnosed with anterior ischemic optic neuropathy, and 1 patient, representing 2,5%, had a history of perinatal central nervous system damage.

All patients underwent a standardized course of complex metabolic and neuroprotective therapy lasting from 10 to 12 days. The treatment regimen included intravenous administration of antioxidant agents aimed at reducing oxidative stress and improving cellular metabolism, as well as vasodilator therapy to enhance microcirculation and optimize blood supply to the optic nerve. Local therapeutic measures were also applied. Parabulbar injections were administered to achieve targeted delivery of medications to the posterior segment of the eye, while subcutaneous temporal injections were used to further stimulate regional blood flow and metabolic activity. In addition, all patients received intramuscular injections of Cortexin at a dosage of 5 milligrams, administered as a course of 10 injections. In cases characterized by progressive clinical deterioration or insufficient stabilization, the therapeutic course was repeated at 6 month intervals. Repeated treatment cycles were continued for up to 1 and a half years, depending on the clinical dynamics and functional outcomes observed during follow up.

For the purpose of comparative clinical evaluation, 2 statistically comparable treatment groups were formed.

The main group, which received combined therapy, consisted of 17 children, accounting for 34 affected eyes. In addition to standard drug therapy, these patients underwent transcranial magnetic stimulation. The stimulation sessions were performed daily for thirty minutes over a ten day course, alongside the prescribed pharmacological treatment.

The comparison group included 23 children, representing 46 affected eyes. Patients in this group received drug therapy alone without the addition of transcranial magnetic stimulation. This design allowed assessment of the additive therapeutic value of neuromodulation in the management of partial optic nerve atrophy. Transcranial magnetic stimulation exerts its therapeutic effects through several neurophysiological mechanisms. It modulates cortical excitability by influencing neuronal membrane potentials and synaptic responsiveness. The procedure enhances synaptic transmission, thereby facilitating signal propagation within cortical and subcortical visual pathways. It also stimulates metabolic processes in neuroglial cells, supporting neuronal trophism and functional recovery. Furthermore, transcranial magnetic stimulation contributes to improved axonal conductivity and promotes activation of residual functional neural pathways, which is particularly important in conditions characterized by partial structural damage and preserved but suppressed neuronal activity. Transcranial magnetic stimulation (TMS) was performed using a clinical magnetic stimulator with a figure of eight coil. Stimulation was applied over the occipital cortical region corresponding to the primary visual cortex. The stimulation frequency ranged from 1 to 10 Hz with intensity set at approximately 80–100% of the individual motor threshold. Each session lasted approximately 30 minutes and consisted of repeated stimulation trains separated by short intervals. The treatment course included 10 consecutive daily sessions. TMS was administered simultaneously with standard neuroprotective and metabolic pharmacotherapy. The rationale for combining neuromodulation with pharmacological treatment was to enhance cortical plasticity, improve neuronal conductivity and facilitate activation of residual functional visual pathways.

At the time of enrollment, all children demonstrated clinical signs consistent with partial optic nerve atrophy confirmed by ophthalmologic examination and neuroimaging. Baseline visual acuity ranged from 0.01 to 0.7. The majority of patients presented with moderate to severe visual impairment. Ophthalmoscopy revealed optic disc pallor in all examined eyes (100%), accompanied by narrowing of retinal arterioles. Visual field examination demonstrated various defects including hemianopia, central scotomas, and paracentral scotomas. Electrophysiological testing showed reduced visual evoked potential amplitude and prolonged P100 latency, indicating impaired conduction along the visual pathways.

At baseline examination, visual acuity among the studied children demonstrated a wide range of functional impairment, varying from very low residual vision to moderately reduced levels. The recorded values extended from 0.01 to 0.7, reflecting the heterogeneity of optic nerve damage within the cohort. The most pronounced decrease in visual acuity was observed in patients with post-traumatic etiology, where visual function ranged from minimal light perception levels to only slight form recognition, corresponding to values between 0.01 and 0.1. Ophthalmoscopic evaluation revealed characteristic structural changes in all examined eyes. Uniform pallor of the optic disc was present in 100% of cases, indicating chronic degenerative changes of the optic nerve fibers. In addition, significant narrowing of the peripapillary arterioles was consistently observed, reflecting compromised microcirculation and long-standing trophic insufficiency of the optic nerve head. In the main group receiving combined therapy, the baseline distribution of visual acuity demonstrated a predominance of advanced visual impairment. Prior to treatment, mild reduction in visual acuity was observed in 4% of eyes, moderate impairment in 26%, severe impairment in 22%, and extremely severe reduction in 48% of cases. Following completion of therapy, a redistribution toward milder categories was noted. The proportion of eyes with mild visual impairment increased to 22%. Moderate impairment was recorded in 22%, severe impairment in 26%, and extremely severe reduction decreased to 30%. Mean visual acuity showed statistically significant improvement, increasing from 0.16 ± 0.04 to 0.29 ± 0.04, with a significance level of $p < 0.05$. In the comparison group treated with pharmacotherapy alone, baseline visual acuity was distributed as follows: moderate impairment in 30% of eyes, severe impairment in 35%, and extremely severe impairment in 35%. After treatment, the proportion of moderate cases increased to 41%, severe cases remained at 35%, and extremely severe impairment decreased to 24%. Mean visual acuity improved from 0.55 ± 0.04 to 0.75 ± 0.04, also reaching statistical significance at $p < 0.05$.

Visual field assessment revealed no detectable changes in 2 eyes, representing 5% of the examined sample. Various types of visual field defects were identified. Homonymous hemianopia was present in 16% of cases, temporal hemianopia in 14%, hemianopia combined with central scotoma in 12%, and hemianopia with paracentral scotoma in 9%. In 6% of patients, only a residual visual field remained in the superonasal quadrant. Normal visual fields were documented in 38% of eyes.

Quantitative analysis of scotomas demonstrated a mean number of first-order relative scotomas equal to 14.1 ± 1.5, second-order relative scotomas equal to 9.7 ± 1.1, and absolute scotomas equal to 18.6 ± 3.5. After treatment, the number of absolute scotomas decreased to 13.2 ± 3.4, indicating partial regression of deep visual field defects.

Electrophysiological evaluation using visual evoked potentials revealed reduced electrical lability at baseline, measured at 28.6 ± 1.1 Hz. Following treatment, this parameter increased to 33.6 ± 1.1 Hz, reflecting improved functional conductivity and enhanced bioelectrical activity of the optic pathways.

Statistical processing of the obtained data was performed using standard biomedical statistical methods. Quantitative variables were expressed as mean values with standard deviation

(M ± SD). The normality of distribution was assessed using the Shapiro–Wilk test. For comparison of quantitative indicators before and after treatment within the same group, the paired Student's t-test was applied. Comparisons between the main group and the comparison group were performed using the independent samples Student's t-test. Categorical variables were analyzed using the chi-square (χ^2) test. A p-value of less than 0.05 was considered statistically significant. Statistical analysis was performed using SPSS statistical software.

Partial optic nerve atrophy in children remains a significant cause of irreversible visual impairmentand represents the final stage of multiple pathological processes affecting the visual pathway. The present study demonstrates that cerebrovascular disorders, traumatic brain injury and hydrocephalic-hypertensive syndrome are among the leading etiological factors contributing to optic nerve degeneration in pediatric patients. These findings are consistent with modern epidemiological studies indicating that intracranial pathology and perinatal neurological damage are major contributors to optic neuropathies in childhood.

Recent international studies emphasize the importance of early detection and neuroprotective interventions in pediatric optic neuropathies. According to Jones et al. (2020), timely diagnosis and multidisciplinary management significantly improve the chances of stabilizing visual function. Similarly, contemporary neuro-ophthalmology research highlights that although structural regeneration of the optic nerve is limited, functional recovery can be partially achieved through neuroplasticity and activation of residual neuronal pathways.

In recent years, increasing attention has been directed toward neuromodulation techniques as a potential adjunct therapy for optic nerve disorders. Transcranial magnetic stimulation has been widely investigated in neurological rehabilitation and is gradually being introduced into neuro-ophthalmological practice. Experimental and clinical studies demonstrate that repetitive TMS can modulate cortical excitability, enhance synaptic transmission, and promote adaptive plasticity within the visual cortex.

The results obtained in the present study support these observations. Children who received combined therapy including TMS demonstrated a more pronounced improvement in visual acuity and electrophysiological parameters compared with patients treated with pharmacotherapy alone.

Improvement in visual evoked potential parameters indicates enhanced conduction along the visual pathways, which may reflect functional activation of partially preserved optic nerve fibers.

Similar outcomes have been reported in recent neurorehabilitation studies where non-invasive brain stimulation techniques contributed to functional improvement in patients with optic neuropathies and other neurovisual disorders. These approaches are believed to facilitate cortical reorganization and strengthen existing neural connections within the visual system.

Importantly, stabilization of visual function should also be considered a clinically meaningful result in patients with optic nerve atrophy, given the progressive and degenerative nature of the disease. Preventing further deterioration may significantly improve long-term visual prognosis and quality of life in pediatric patients.

Despite these encouraging findings, several limitations should be acknowledged. The relatively small sample size and heterogeneity of etiological factors may influence the generalizability of the results. Future multicenter studies with larger patient cohorts and standardized neuromodulation protocols are necessary to confirm the effectiveness of TMS in pediatric optic neuropathies.

Overall, the present study contributes to the growing body of evidence supporting the use of combined neuroprotective therapy and neuromodulation in the rehabilitation of children with partial optic nerve atrophy.

4. Conclusions

1. Partial optic nerve atrophy most frequently affects children around 11 years of age and occurs equally in boys and girls.
2. The primary causes were:
   - Brain tumors and non-neoplastic cerebrovascular diseases – 57.5%
   - Post-traumatic injury – 22.5%
   - Hydrocephalic-hypertensive syndrome – 12.5%
   - Anterior ischemic optic neuropathy – 5%
   - Perinatal CNS damage – 2.5%
3. Combined therapy including transcranial magnetic stimulation significantly improves visual acuity, reduces scotoma burden, and enhances electrophysiological parameters.
4. TMS should be considered as part of comprehensive rehabilitation in pediatric patients with partial optic nerve atrophy.

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