OPIS PRZYPADKU CASE REPORT

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# Diagnostic challenges in optic nerve neuropathy in an 8-year-old patient–case report



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## HIGHLIGHTS

Case report of an 8-year-old with optic neuropathy highlighting diagnostic challenges and the importance of multimodal imaging and genetic testing in differentiating hereditary causes.

## ABSTRACT

**Background**: Optic neuropathies in children have diverse etiologies and may lead to permanent vision loss. The most common hereditary forms, such as autosomal dominant optic atrophy and Leber hereditary optic neuropathy, require careful differential diagnosis.

**Objective**: To present diagnostic challenges in a child with optic neuropathy and highlight the role of imaging and electrophysiological tests.

**Case report**: An 8-year-old girl presented with bilateral visual acuity deterioration. Examination revealed optic disc atrophy and thinning of retinal nerve fiber layer and ganglion cell complex. Electrophysiological tests were normal, and infections and vitamin deficiencies were excluded.

**Conclusions**: The findings suggested hereditary optic neuropathy, emphasizing the importance of multimodal diagnostics and genetic testing.

**Key words:** autosomal dominant optic atrophy, Leber hereditary optic neuropathy, fundus imaging


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## INTRODUCTION

Optic nerve neuropathies in children represent a considerable diagnostic challenge due to their heterogeneous etiology, which includes inflammatory, metabolic, genetic, ischemic, and traumatic causes. These difficulties are compounded by nonspecific clinical symptoms and the need for extensive differential diagnosis with other ophthalmic and neurological conditions [1]. Among genetically determined neuropathies, the most common is Kjer's optic atrophy, inherited in an autosomal dominant manner (ADOA, autosomal dominant optic atrophy). This disorder usually manifests in the first or second decade of life, is characterized by incomplete penetrance, and shows variable severity of symptoms even within families. Clinical features include progressive loss of visual acuity, impaired color vision, and central, paracentral, or centrocecal scotomas. Optic disc pallor may range from mild, confined to the temporal segment, to diffuse involvement. Most patients lack extraocular symptoms, although sensorineural hearing loss may occur. In the majority of cases, mutations in the *OPA1* gene are responsible, with an estimated prevalence of 1 in 50,000 [2, 3]. Differential diagnosis should also include Leber hereditary optic neuropathy (LHON), which although more common in young adult males, can present in childhood. LHON results from mitochondrial DNA mutations and manifests with sudden, painless unilateral central vision loss, typically followed by involvement of the second eye within a few months [4, 5]. Prematurity, perinatal events, and developmental disorders are additional risk factors, potentially causing irreversible impairment. Furthermore, compressive lesions must be considered in pediatric patients [6]. Rarely, optic neuropathies result from vitamin $B_{12}$ or $B_9$ deficiency. Such cases present bilaterally, painlessly, and progress slowly, with diagnosis depending largely on careful history taking. Overlapping etiologies can further obscure the clinical picture [7, 8].

## CASE REPORT

An 8-year-old female patient presented to the Emergency Department of the Prof. K. Gibiński University Clinical Center, Medical University of Silesia in Katowice, due to progressive visual deterioration. The parent acting as the legal guardian provided informed consent for the patient's participation in the study. On examination, best-corrected visual acuity (BCVA) was 0.9 in the right eye (OD, *oculus dexter*) and 0.7 in the left eye (OS, *oculus sinister*). Cycloplegic refraction measured +1.0 Dsph, -1.0 Dcyl ax. 6 in the OD and +1.25 Dsph, -0.75 Dcyl ax. 2 in the OS. The examination was performed after administration of three doses of 1% tropicamide. Fundus imaging (Carl Zeiss Meditec, Inc. CLARUS 700 v1.1) revealed pale optic discs in both eyes (OU, *oculus uterque*), with well-defined margins, located at the level of the fundus, without signs of edema. Retinal arteries and veins were of normal caliber and course (fig. 1). Color vision testing with Ishihara plates was normal. The patient was admitted to the pediatric ophthalmology department for urgent diagnostic evaluation due to features of bilateral optic nerve atrophy in OU. Ultrasonography revealed a few vitreous opacities in OU, with otherwise normal posterior segment echograms and regular contour of the posterior poles. Optical coherence tomography (OCT, Carl Zeiss Meditec, Inc.) of the macula of the retina (fig. 2a, b) revealed preserved foveal morphology with slight flattening of the foveal contour. Central retinal thickness measured 230 µm in the OD and 221 µm in the OS, indicating mild thinning. A marked reduction in retinal nerve fiber layer (RNFL) thickness was noted, measuring 68 µm in the OD and 70 µm in the OS. Ganglion cell complex (GCC) analysis showed thinning to approximately 70 µm, findings consistent with optic nerve atrophy (fig. 3a, b). Static perimetry (Humphrey Field Analyzer, Carl Zeiss Meditec, Inc.) demonstrated relative paracentral visual fields defects near the fixation point in OU, typical for early neuronal damage. Despite these changes, overall visual field function was nearly preserved (VFI, Visual Field Index: 98%) (fig. 4a, b). Photopic negative response (PhNR) testing (RETeval, TM, LKC Technologies, Inc.) showed a critically reduced PhNR amplitude, confirming ganglion cell dysfunction and, consequently, optic nerve atrophy (fig. 5). Results of pattern visual evoked potentials (pVEP) and flash visual evoked potentials (fVEP, LKC Technologies Vision Defined, TM, UTAS, TM Visual Diagnostic System) were within normal limits, which is atypical in the context of features characteristic for Kjer's optic atrophy (fig. 6a, b). Magnetic resonance imaging (MRI) of the brain revealed normal central nervous system structures, with no abnormalities of the globes or orbital tissues. During hospitalization, infectious causes (cytomegalovirus, Lyme disease, toxoplasmosis, toxocariasis) were excluded. Serum vitamin D and $B_{12}$ levels were within normal limits. Testing for antineutrophil cytoplasmic antibodies (pANCA, cANCA) was negative. Antinuclear antibodies (ANA) IgG were present at a titer of 1:100. The diagnostic work-up pro-

FIGURE 1

Color fundus photographs of the right and left eye.




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FIGURE 2a

OCT of the macula of the right eye.

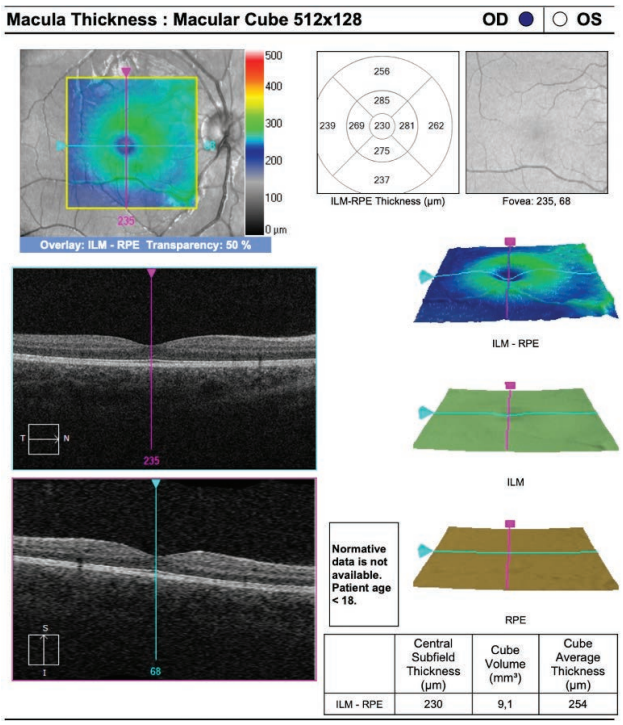



FIGURE 2b

OCT of the macula of the left eye.

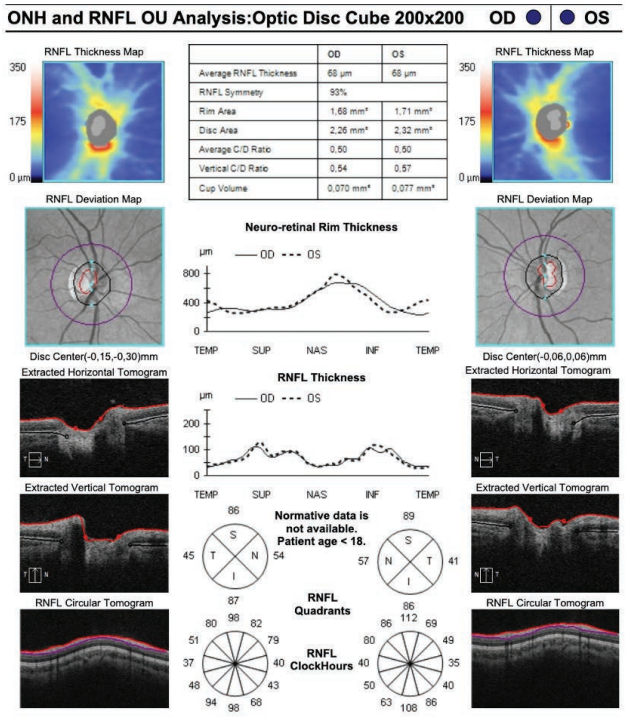



FIGURE 3

A. OCT of the optic nerve head in both eyes.
B. Retinal ganglion cell complex (GCC) thickness assessment.

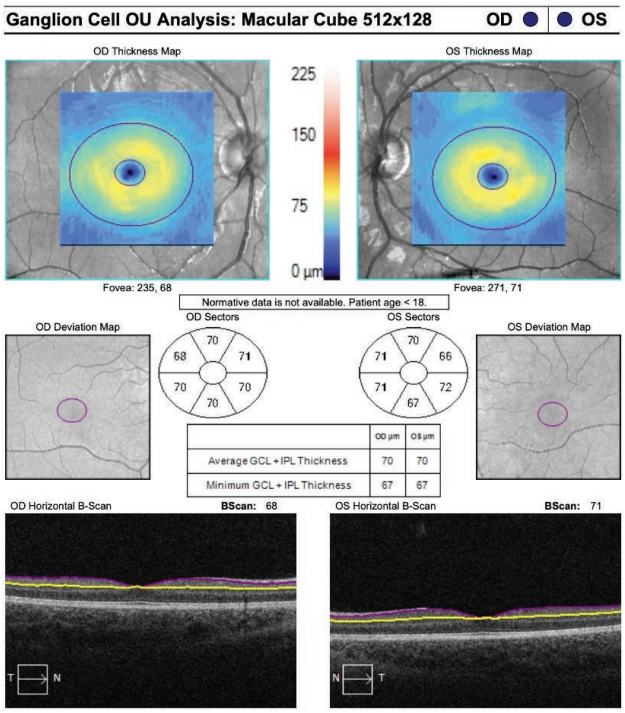
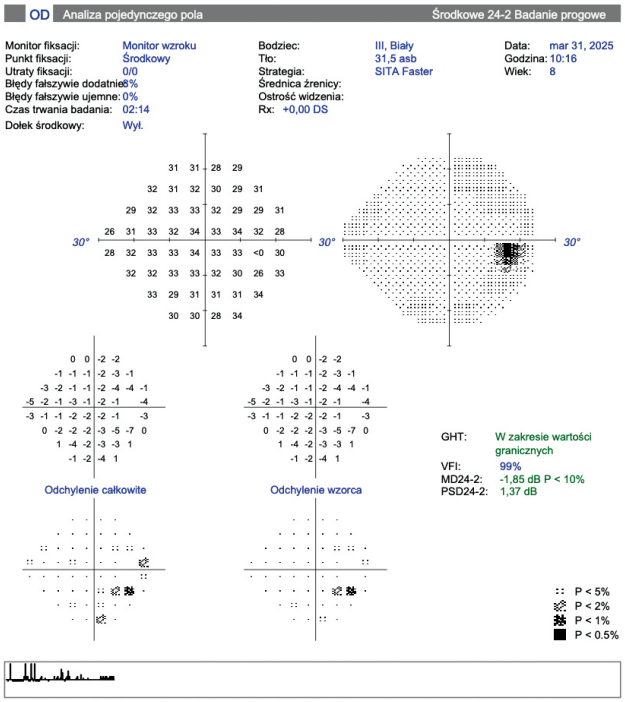



FIGURE 4a

Static perimetry of the right eye.





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FIGURE 4b

Static perimetry of the left eye.

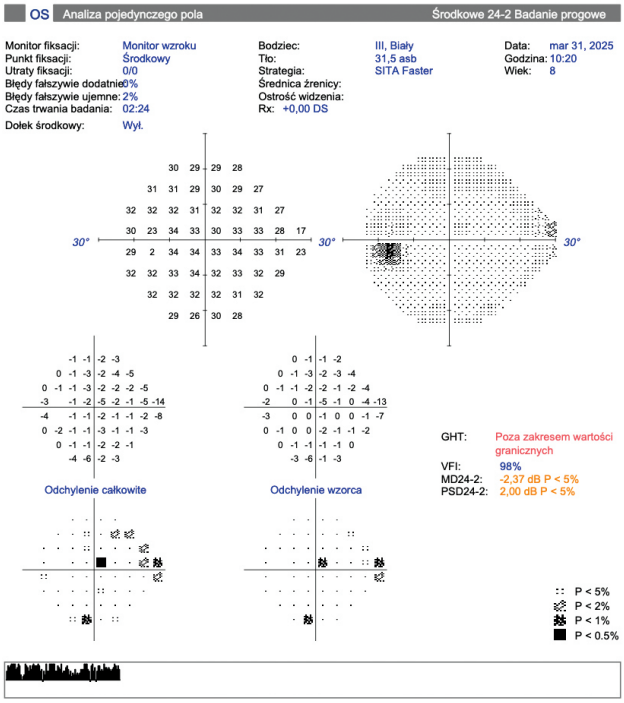
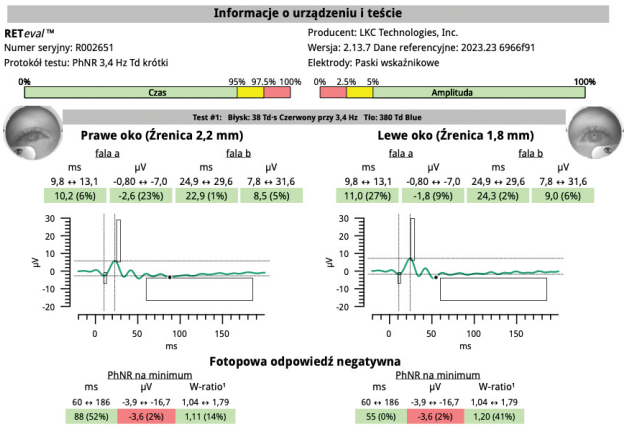



FIGURE 5

Photopic negative response (PhNR) testing.




¹W-ratio = (b - $p_{min}$) / (b - a) co jest odwrotnością "PTR" jak opisano u Mortlock (2010)
gdzie a, b, i $p_{min}$ są napięcia linii podstawowej określonej jako
a: pik fali a, b: pik fali b, i $p_{min}$: minimum fali PhNR.

FIGURE 6a

Pattern visual evoked potentials (pattern VEP).

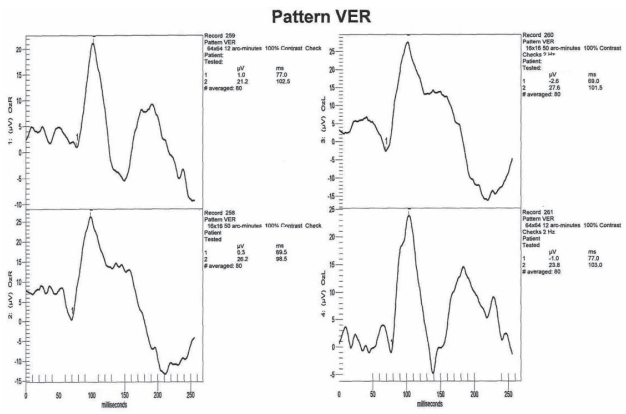
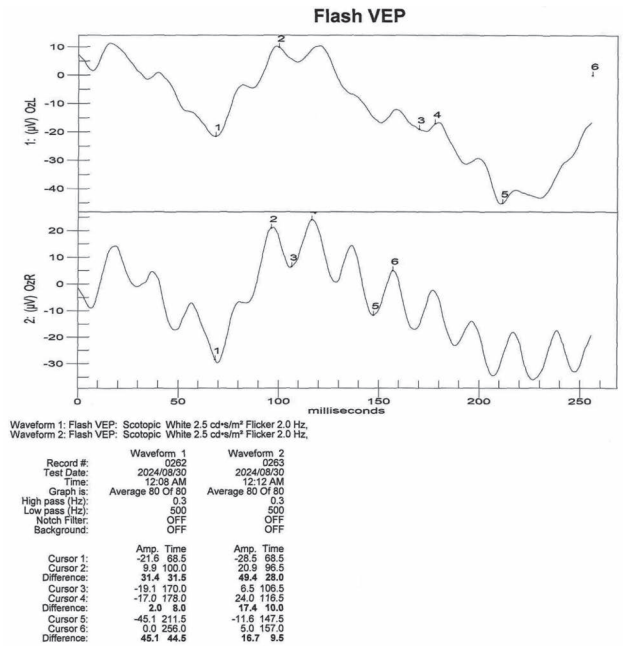



FIGURE 6b

Flash visual evoked potentials (flash VEP).




vided a comprehensive assessment of the ocular condition, allowing further outpatient investigations. The patient was discharged in good general and ocular condition with recommendations for follow-up.

## DISCUSSION

Diagnosis of optic nerve neuropathy is particularly demanding given the wide variety of potential causes. Symptoms often develop gradually and appear similar regardless of etiology, which complicates early recognition and appropriate targeting of tests. The coexistence of multiple mechanisms may blur classic clinical features, increasing the risk of misdiagnosis. In the presented patient, a broad differential must be considered, including ADOA, LHON, autoimmune conditions, nutritional neuropathy, prematurity, low birth weight, and compressive lesions [2, 4, 6, 8]. Characteristic features of these conditions are summarized in table 1, with those present in our patient highlighted. Patients with pathogenic *OPA1* variants display highly variable phenotypes. Diagnosis is usually made in childhood following slowly pro-


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gressive bilateral disturbances. Syndromic forms (DOAplus) may also occur [9]. Skidd et al. report sensorineural hearing loss in around 60% of ADOAplus patients and progressive external ophthalmoplegia in 46%, with associations also described with multiple sclerosis, stroke, cardiomyopathy, and spastic paraplegia [10–12]. Van Laar et al. highlight that *OPA1*, expressed in neuronal mitochondria, is essential for their function, and mutations may predispose to neurodegeneration, potentially linking ADOA with Parkinson's disease, Alzheimer's disease, or Huntington's chorea [13]. Visual impairment in *OPA1*-related disorders is irreversible and varies from subclinical deficits to legal blindness. Al Othman et al. describe biallelic *OPA1* variants leading to the Behr phenotype, characterized by early onset severe symptoms and systemic features [14]. Diagnosis is based on optic disc examination, typically showing bilateral temporal pallor, and confirmed by OCT with RNFL thinning and GCC reduction. Visual fields demonstrate central or centrocecal scotomas, often with tritanopia. OCT angiography can reveal microcirculatory damage correlating with visual function [10, 15, 16]. VEP abnormalities are frequent (prolonged latency, reduced amplitude) but not constant [17]. Animal models with *OPA1* mutations confirm functional deficits and reduced PhNR amplitude, similar to those observed in patients (tab. 1) [18]. In the presented patient, visual disturbances began in early childhood, progressed slowly, and were bilateral. Pale optic discs, GCC thinning, and reduced PhNR amplitude indicating optic nerve atrophy, though normal VEP results are atypical for ADOA. LHON is caused by mitochondrial point mutations, most commonly m.3460G>A (ND1), m.11778G>A (ND4), and m.14484T>C (ND6). It predominantly affects young adult males with bilateral, painless, subacute central vision loss due to retinal ganglion cell (RGC) degeneration. Symptoms usually affect one eye first, with the fellow eye involved within a year. Incomplete penetrance means not all carriers lose vision. Early symptoms include blurred or foggy vision, loss of color perception, and development of central scotomas, leading to severe visual impairment. Fundus features include peripapillary telangiectasias and RNFL edema, followed by optic atrophy. OCT confirms RNFL thinning, especially temporally (tab. 1) [5, 19]. In our patient, the onset in childhood, simultaneous bilateral involvement, and slow progression differ from typical LHON. Autoimmune retinal vasculitis (RV) must also be considered. Abu El-Asrar et al. describe its occurrence in Behçet's disease, sarcoidosis, Wegener's granulomatosis, and Churg–Strauss syndrome [20]. Positive ANA supports suspicion of autoimmune processes, particularly systemic lupus erythematosus (SLE). Madurapperuma et al. presented a case of bilateral RV as the first manifestation of SLE, with progressive bilateral vision loss, fundus hemorrhages, macular edema, and vascular narrowing. Fluorescein angiography (FFA) confirmed occlusive retinal vasculitis, high titers of ANA and anti-double-stranded DNA antibodies (anti-dsDNA) were detected. Corticosteroid therapy improved vision [21]. Donnithorne et al. described two pediatric SLE patients, who tested positive for ANA: one with branch retinal artery occlusion, vitreous hemorrhage, tractional retinal detachment and ischemic optic neuropathy requiring aggressive immunotherapy but with poor outcome, and another with hemorrhages and cotton-wool spots who responded well to rituximab and cyclophosphamide. Differences in clinical presentation and treatment response between these two pediatric SLE cases highlight the variable severity of retinal vasculitis in this disease and the heterogeneous response to therapy (tab. 1) [22]. In the present case, such fundus changes and systemic autoimmune features were absent. Prematurity and perinatal events also predispose to optic nerve atrophy. Scott et al. reviewed international studies confirming significant associations with RNFL thinning, enlarged cup-to-disc ratio, and optic nerve changes [6]. Our patient was born at term without complications. MRI excluded compressive lesions. Nutritional and toxic neuropathies are further considerations. Deficiency of vitamin $B_{12}$, folate, or copper may cause bilateral, painless, slowly progressive vision loss with central scotomas and temporal disc pallor, often aggravated by toxins such as ethambutol or amiodarone [7, 8]. In our patient, laboratory results excluded deficiencies, and history did not reveal relevant exposures. Family history was significant for glaucoma and maternal myopia. Frequent epistaxis was unrelated. The progressive bilateral pattern and family history strongly support a genetic neuropathy. The *OPA1* gene encodes an inner mitochondrial membrane protein essential for mitochondrial fusion, membrane stability, and cell survival [23, 24]. RGCs are particularly vulnerable due to their high energy demands. Han et al., using next-generation sequencing (NGS) in 2022, identified 9 novel mutation variants in 11 unrelated Chinese families. During this study, more than 400 pathogenic *OPA1* mutation variants were reported in the Leiden Open Variation Database [23]. Next-generation sequencing (NGS) is the most effective diagnostic tool and helps broaden understanding of the genetic basis of disease. Experimental approaches, such as U1 splicing factor modification, have been shown to correct splicing defects and restore transcript expression in vitro [24]. In LHON, inheritance is maternal, and environmental triggers such as smoking and alcohol increase disease penetrance. Several pathogenic mechanisms are known, one involves dysfunction of complex I (CI) of the electron transport chain, the NADH:ubiquinone oxidoreductase. The aforementioned missense mutations (ND1, ND4, ND6) are associated with CI. Damage to the respiratory chain leads to ATP synthesis failure, oxidative stress, and RGC degeneration. Consequently, visual signals are improperly processed, leading to severe visual impairment. Idebenone, a synthetic

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benzoquinone, is currently the only approved therapy. Diagnosis relies on mtDNA analysis using targeted panels or whole mtDNA sequencing [5, 19]. In our case, absence of genetic results complicates diagnosis, since identification of a variant is crucial to confirm hereditary neuropathies. Importantly, a negative result does not entirely exclude ADOA, highlighting the complexity of diagnosis.

## RESULTS

The diagnosis of optic neuropathy in children remains difficult because of heterogeneous etiology, overlapping pathogenic factors, and subtle clinical features. In the presented case, the bilateral onset, slow progression, and absence of autoimmune, nutritional, perinatal, or compressive factors point to a genetic cause, most consistent with ADOA. Multimodal ophthalmic imaging revealed typical findings such as pale discs, RNFL and GCC thinning, and impaired RGC function on electrophysiology. Certain features deviated from the classical phenotype, reflecting variability and diagnostic complexity. Family history supports genetic involvement and underscores the importance of molecular testing. Genetic confirmation would help establish a definitive diagnosis, though negative results cannot exclude ADOA. This case illustrates the challenges in diagnosing pediatric optic neuropathies and emphasizes the need for comprehensive multimodal assessment and genetic evaluation.

TABLE 1

Comparison of selected clinical, diagnostic, and therapeutic features of autosomal dominant optic atrophy, Leber hereditary optic neuropathy, and autoimmune retinal vasculitis (*italicized features indicate those observed in the presented patient*).

| Feature/Disease entity | Autosomal dominant optic atrophy | Leber hereditary optic neuropathy | Autoimmune retinal vasculitis |
|---|---|---|---|
| Etiology | Mutation most commonly in the *OPA1* gene, autosomal dominant inheritance | Point mutations in mitochondrial DNA, most often missense in *ND1, ND4, ND6* genes, maternally inherited | Autoimmune origin, production of autoantibodies, association with systemic diseases (e.g., SLE, Behçet's disease, sarcoidosis) |
| Onset/Symptoms dynamics | *First or second decade of life, slowly progressive, symmetric visual disturbances,* occurrence of syndromic forms | Young adults, more often males, subacute onset, unilateral central vision loss, with involvement of the second eye within a few months | Fluctuating, sudden or subacute onset, unilateral or bilateral |
| Clinical symptoms | Gradual vision loss potentially leading to complete blindness, color vision impairment (tritanopia), central, paracentral, centrocecal scotomas**,** *pale optic discs (temporal* or diffuse), possible extraocular symptoms (sensorineural hearing loss, external ophthalmoplegia, etc.) | Subacute, painless central vision loss, unilateral central vision defect with progression to the contralateral eye over several months, possible abnormalities in cardiac conduction system | Sudden vision loss or progressive deterioration, may present extraocular symptoms (e.g., headache, musculoskeletal complaints, joint pain, abdominal pain, nausea, fever, rash) |
| Diagnostic findings | *OCT: RNFL and GCC thinning,* VEP: possible abnormalities. Genetic testing: OPA1 mutation confirmation. Fundoscopy: *pale optic discs (especially temporal)* | *OCT: RNFL thinning*.<br>VEP: often abnormal, prolonged latency, reduced amplitude. Fundoscopy: edema of superior and inferior fiber arcades, axonal loss in papillomacular bundle, *pale optic discs (especially temporal)* | FFA: leakage due to vessel wall damage, occlusive arteritis and phlebitis. OCT: macular edema. Fundoscopy: cotton wool spots, retinal hemorrhages, vascular changes – arteriolar narrowing, venous segmentation, macular edema, optic disc edema |
| Treatment | No effective causal therapy | Idebenone, outcomes depend on mutation type | Steroid therapy, immunosupressive drugs, panretinal photocoagulation |

FFA – fluorescein angiography; GCC – ganglion cell complex; OCT – optical coherence tomography; RNFL – retinal nerve fiber layer; SLE – systemic lupus erythematosus; VEP – visual evoked potentials.

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**Authors' contributions:**
All authors have read, edited and approved the final manuscript. The authors alone are responsible for the content and writing of the paper.
Natalia Lekston conducted the examination and drafted the manuscript.
Sebastian Sirek helped to conduct the examination and drafting the article, revising it critically for important intellectual content.
Adrian Smędowski helped with revising it critically for important intellectual content and with the ophthalmological clinical information.
Dorota Wyględowska-Promieńska helped with the ophthalmological clinical information and final approval of the version to be submitted.

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The content presented in the article complies with the principles of the Helsinki Declaration, EU directives and harmonized requirements for biomedical journals


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