OPIS SERII PRZYPADKÓW CASE SERIES REPORT

DOI: 10.24292/01.OT.410126

Suspected optic neuritis at the ophthalmological emergency department

Antoni Sieńko[1], Małgorzata Łabuś[1], Tomasz Maciejczyk[1], Sebastian Sirek[2, 3], Dorota Wyględowska-Promieńska[2, 3]

[1] Student Scientific Club at the Department of Ophthalmology, Faculty of Ophthalmology, Department of Ophthalmology, Faculty of Medical Sciences in Katowice, Medical University of Silesia in Katowice
Head: prof. Dorota Wyględowska-Promieńska, MD, PhD

[2] Ophthalmology Clinic, Department of Ophthalmology, Faculty of Medical Sciences in Katowice, Silesian Medical University in Katowice
Head: prof. Dorota Wyględowska-Promieńska, MD, PhD

[3] Department of Adult Ophthalmology, Prof. K. Gibinski University Clinical Center of the Silesian Medical University in Katowice
Head: prof. Dorota Wyględowska-Promieńska, MD, PhD

HIGHLIGHTS

The study analyzes 71 cases of suspected optic neuritis, highlighting the importance of early diagnosis and comprehensive clinical evaluation to preserve visual function in patients.

ABSTRACT

Introduction: Optic neuritis is damage and swelling of the optic nerve myelin, presenting with pain during eye movements, decreased visual acuity, color vision deficiency, and visual field loss. It mainly affects people aged 20–45 years, particularly Caucasian women.
The aim: Assessment of the effectiveness of optic neuritis diagnostics at the ophthalmological emergency department
Materials and methods: Analysis of 71 cases from the Emergency Department of the University Clinical Center in Katowice (January–June 2022).
Results: Eye pain was reported in 5 patients, color vision deficiency in 18, and 11 were referred for hospitalization.
Conclusions: Early diagnosis is crucial for vision preservation.

Key words: optic neuritis, visual evoked potentials, VEP, ophthalmological emergency

INTRODUCTION

Optic neuritis is a nosological entity with a demyelinating nature. It is a disease with a diverse etiology and course, and in light of developing new diagnostic methods, it still poses a challenge in terms of proper diagnosis and treatment [1]. The symptoms are nonspecific and primarily include unilateral optic neuritis, pain with eye movements, visual acuity decrease, color vision deficiency, visual field impairment, rapid symptom progression (within 2 weeks), and, importantly, in most cases, a normal optic disc image in the examination of the fundus of the eye (optic disc swelling occurs in about 35% of patients on average) [2]. The etiology of optic neuritis includes a wide range of factors with autoimmune, infectious, and systemic origins.

The incidence of optic neuritis is about 4–5 people per 100,000 inhabitants per year, according to studies conducted in Sweden and Denmark [1, 3]. Worldwide, the incidence of unilateral optic neuritis ranges from 0.94 to 2.18 per 100,000 people annually [2]. Optic neuritis most commonly affects people aged 20 to 45 years. It occurs twice as often in women as in men and is more common in Caucasians living in temperate climate [1].

MATERIALS AND METHODS

As part of the emergency ophthalmology service in the Emergency Room of the Adult Ophthalmology Department at the K. Gibinski University Clinical Center of the Medical University of Silesia in Katowice, a total of 71 patients were examined for suspected optic neuritis between January and June 2022. 48 were first-time patients, and 22 were returning patients. Women presented more frequently (43) compared to men (28). The median age was 34 years, with women having a median age of 37 years and men 34 years.

The best corrected visual acuity (BCVA) of the right eye (OD) was 0.76, and the BCVA of the left eye (OS) was 0.68. The median visual acuity for both OD and OS was 0.91. The average visual acuity was 0.77 (±0.32) for OD and 0.69 (±0.37) for OS. The mean intraocular pressure (IOP) was 15.22 (±2) mmHg for OD and 15.38 (±2.47) mmHg for OS. Using the air-puff tonometry method, in none of the patients values of IOP were elevated.

One of the typical symptoms of optic neuritis, pain with eye movements, was present in 5 patients (3 men and 2 women). Another common symptom in this condition, color vision deficiency, was observed in 18 patients (12 women and 6 men). The coexistence of both symptoms (pain with eye movements and color vision deficiency) occurred in 6 patients, all of whom were women (fig. 1).

Ultimately, in 11 patients, further diagnostics for optic neuritis were conducted in a hospital setting. After thorough diagnostics were implemented, the final diagnoses for the hospitalized patients were ischemic optic neuropathy (3 patients), uveitis (2 patients), and optic neuritis, which was diagnosed in 4 of the admitted patients.

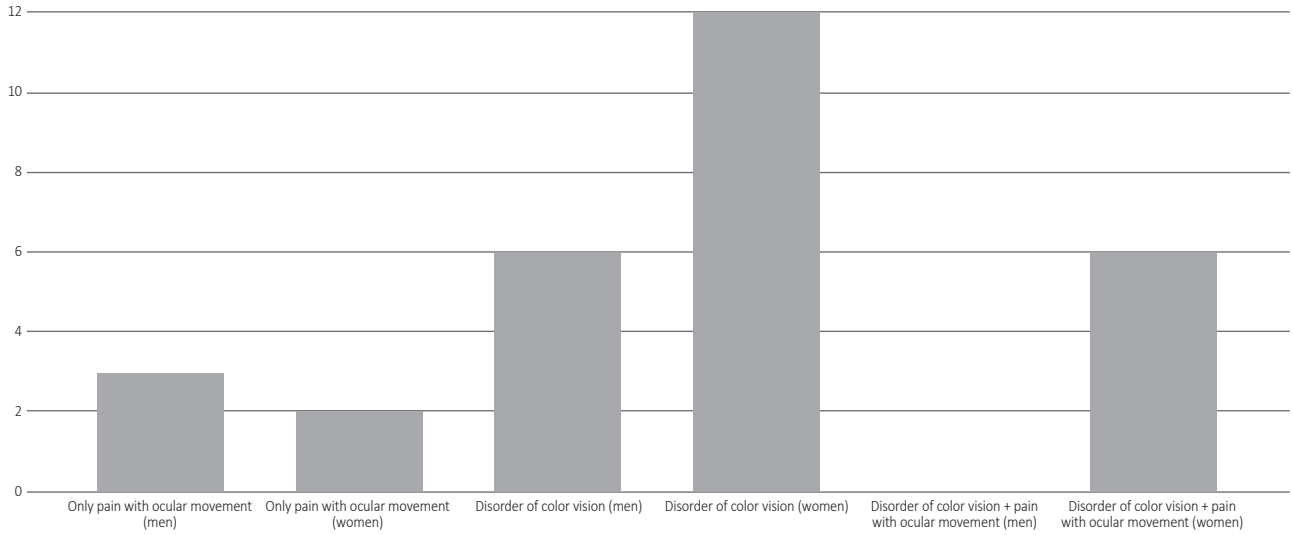
CASE REPORTS

Case 1

The first case involves a 17-year-old female patient who presented to the emergency room only one day after the onset of symptoms such as visual acuity decrease in the

FIGURE 1

Incidence of color vision deficiency and pain with the eye movements appearance of patients reporting to K. Gibinski UCC Emergency Room from January to June 2022 with an optic neuritis suspicion.



OS, pain with the eye movements in OS, and color vision deficiency. She had a past medical history of unspecified demyelinating changes. The initial ophthalmological examination revealed visual acuity (visus) of 4/50 in LE and 5/5 in OD. IOP was measured at 14 mmHg in OD and 15 mmHg in OS.

The patient underwent visual field testing; optical coherence tomography (OCT) of the retina (macular OCT), visual evoked potentials (VEP), and magnetic resonance imaging (MRI) were performed. The MRI revealed demyelinating changes located supra- and infratentorially. Additionally, the screening test showed a positive IgM antibody titer for Lyme disease, which was recommended to be confirmed by the Western Blot test.

Systemic treatment with methylprednisolone and topical treatment with dexamethasone and diclofenac were initiated. The therapy resulted in improvement of the local condition, and the patient was discharged from the hospital with further recommendations. Upon discharge, the ophthalmologic examination showed visual acuity of 5/5 in both eyes (visus OS and OD). Intraocular pressure was 18 mmHg in OD and 19 mmHg in OS.

Case 2

Case 2 involves an 11-year-old male patient who also presented to the emergency room just one day after symptom onset. The boy was presenting symptoms typical of optic neuritis, such as a decrease in visual acuity in OS and color vision deficiency in OD.

The initial ophthalmological examination showed visual acuity of 5/5 in OD (without correction) and 1/50 in OS. Intraocular pressure was 17 mmHg in OD and 16 mmHg in OS. The patient underwent OCT of the posterior optic disc and macula, brain MRI with contrast, and VEP testing. The VEP test indicated the function of ganglion cells was about 50% of normal in both eyes, with a slightly prolonged latency in OS.

The treatment included dexamethasone as well as magnesium and potassium supplementation, resulting in clinical improvement and visual acuity of 5/5 in OD and 1.5/50 in OS. Intraocular pressure was 16 mmHg in OD and 18 mmHg in OS. The patient was referred to the Ophthalmology Clinic, Genetics Clinic, and Pediatric Clinic for further observation and treatment. Further diagnostics revealed the cause of the symptoms to be Leber's congenital amaurosis (LCA).

Case 3

The next case involves a 35-year-old male patient who presented to the emergency room two days after symptom onset, reporting decreased visual acuity in OD and pain with the eye movements. The patient's medical history included ongoing diagnostics for multiple sclerosis (MS).

Upon admission, the visual acuity was 0.5/50 in OD and 5/5 in OS (without correction). IOP was 16 mmHg in both eyes. Visual field testing, VEP, and MRI were performed. These tests revealed an absolute scotoma in the central visual field of OD and MRI changes located supra- and infratentorially, typical of a primary demyelinating process.

Systemic treatment with methylprednisolone and local treatment with diclofenac and dexamethasone were administered. Upon discharge, ophthalmological examination showed visual acuity of 5/5 in both eyes, with intraocular pressure of 17 mmHg in OD and 16 mmHg in OS. The systemic and local treatment resulted in an improvement considering the presence of clinical symptoms. The patient was referred to a neurology clinic for further diagnostic workup.

Case 4

The last case involves a 31-year-old female patient who presented to the Ophthalmology Department on the same day her ophthalmic symptoms had appeared, and those were: pain with the eye movements in OS and color vision deficiency in OS.

The initial ophthalmological examination showed visual acuity of 5/5 in OD and 5/7 in OS. Intraocular pressure was 16 mmHg in OD and 17 mmHg in OS. The patient underwent visual field testing and MRI, which revealed demyelinating changes in the white matter located supratentorially. The left optic nerve in the retrobulbar segment showed increased signal intensity and stronger contrast enhancement.

Treatment with cefuroxime, probiotics, and methylprednisolone was initiated, leading to gradual improvement in the ophthalmic condition and visual acuity of 5/5 in both eyes. Intraocular pressure was 16 mmHg in both eyes. The patient was urgently referred to the Neurology Clinic and Ophthalmology Clinic for follow-up.

DISCUSSION

Etiology

In most cases, optic neuritis is one of the first manifestations of MS. The cause of inflammation in this case is likely an autoimmune reaction leading to demyelination, characterized by perivascular cuffs, T-lymphocyte infiltration, and plasma cells [1]. Another disease that presents with optic neuritis is acute disseminated encephalomyelitis (ADEM). The symptoms of inflammation occur mainly in children, more often following an infection or vaccination [1, 4]. Optic neuritis can also occur in autoimmune diseases such as *neuromyelitis optica* spectrum disorders (NMOSD). In these cases, transverse myelitis is also observed, and in most cases, anti-AQP4 antibodies are detected [1]. There are also SION, RION, and CRION, which refer to isolated

optic neuritis with a single, recurrent, and chronically recurrent course, respectively. These types of inflammation also have an autoimmune basis [1, 4].

In autoimmune processes that cause optic neuritis, several antibodies against specific proteins are also involved. These include the aforementioned anti-AQP4 antibodies associated with NMO, optic neuritis associated with antibodies against CRMP5 (CRMP5-ON), and optic neuritis associated with the disease syndrome with antibodies against myelin oligodendrocyte glycoprotein (MOG-ON) [1, 4]. Infectious and systemic factors are much less common causes of optic neuritis, but there are a number of microorganisms that can cause this inflammation. The inflammatory process may be initiated by the spread of infection involving the orbits or paranasal sinuses, or it may occur in the course of a systemic infection or systemic disease [1].

Infectious factors include pathogens such as: *Bartonella*, *Brucella*, Chikungunya fever, cytomegalovirus, coronavirus, *Coxiella burnetti*, dengue, Epstein–Barr virus, echovirus, ehrlichiosis, Henoch–Schonlein purpura, hepatitis B and C, *Herpes simplex*, histoplasmosis, HIV, human herpesvirus 6, hypertrophic pachymeningitis, IgG subclass deficiency, Inoue-Melnick virus, leprosy, Lyme disease, measles, mumps, *Mycoplasma pneumoniae*, neurotoxocarosis, ocular cat-scratch disease, post-vaccination optic neuritis, rubella, streptococcus, syphilis, tick-borne encephalitis, toxoplasmosis, tuberculosis, typhus, varicella zoster virus, West Nile virus, Zika virus, and Whipple disease [1, 4].

Diagnosis

The diagnosis of optic neuritis is based on the presence of clinical symptoms as well as changes observed in imaging and laboratory studies [1]. The diagnostic criteria for optic neuritis introduced in 2022 by Petzold et al. include three clinical criteria (A, B, C) and three paraclinical criteria [1].

The clinical criteria are as follows:

A: Subacute vision loss in one eye accompanied by pain in the orbit that worsens with eye movements, reduced contrast sensitivity, or color vision impairment, and the presence of a relative afferent pupillary defect (RAPD).

B: Painless vision loss with all other features of criterion A.

C: Vision loss in both eyes with all other symptoms mentioned in criteria A or B.

The paraclinical criteria include:

- In OCT examination: optic disc swelling corresponding to visual disturbances or a difference in mGCIPL (macular ganglion cell-inner plexiform layer) thickness between the eyes of >4% or 4 μm, or a difference in pRNFL (peripapillary retinal nerve fiber layer) thickness of >5% or 5 μm within 3 months of symptom onset.
- In MRI examination: contrast enhancement of the optic nerve and its sheaths in the acute phase of the disease, or further enhancement of the intrinsic signal (appearing brighter) within 3 months of symptom onset.
- Biomarkers: the presence of antibodies against AQP4, MOG, or CRMP5 in the blood serum or intrathecal synthesis of IgG in cerebrospinal fluid (oligoclonal bands).

For a definitive diagnosis of optic neuritis, the study authors suggest the following combinations of criteria:

- Clinical criterion A and a positive result in one of the paraclinical tests.
- Clinical criterion B and two positive paraclinical results of different types.
- Clinical criterion C and two positive results from different paraclinical tests, with at least one being an MRI result.

A possible optic neuritis may be suggested by:

- The presence of one of the clinical criteria (A, B, or C) in the acute phase of the disease, without positive paraclinical results, if the appearance of the fundus corresponds to the typical picture of optic neuritis and is consistent with the natural course of the disease over time in follow-up examinations.
- A positive result from one or more tests with a medical history suggesting optic neuritis.

If methods to confirm or exclude the presence of the symptoms mentioned in the paraclinical criteria are unavailable, the diagnosis can be based solely on clinical criteria [1].

Differential diagnosis

The difficulties in differentiating the diseases diagnosed in patients admitted to the K. Gibinski UCC, especially in the early stages of diagnosis, arise from many similarities in both objective and subjective symptoms and challenges in interpreting the results of initial diagnostic tests.

The hospitalized patients reported symptoms (sudden decrease in visual acuity, visual field defects, color vision deficiency, rapid progression of symptoms) common to all of the entities they have been diagnosed with; these were: optic neuritis, ischemic optic neuropathy, uveitis and iridicysticis, which caused difficulties in interpreting the results of tests such as BCVA, perimetry, ophthalmoscopy, and color vision testing using the Ishihara plates [3, 5, 6]. Therefore, the decision to extend the diagnostics towards optic neuritis should be made carefully and based on several key factors that consider differentiation from other diseases.

The importance of taking a thorough medical history is emphasized, focusing on characteristic differentiating symptoms. Among the patients we analyzed, certain patterns regarding the occurrence of symptoms could be observed (tab. 1).

TABLE 1

Summary of symptoms of optic neuritis, ischemic optic neuropathy, uveitis, and iridocyclitis observed in patients presenting to the Emergency Department of the University Clinical Center of the Medical University of Silesia (UCK SUM) from January to June 2022 with suspected optic neuritis.

	Sudden decrease in visual acuity	Pain with eye movements	Color vision deficiency	Swelling of the optic nerve disc	Systemic symptoms	Visual field defects
Optic neuritis	+	+	+	-	-	+
Ischemic optic neuropathy	+	-	-	+	+/-	+
Uveitis	+	+	+	+	+	+
Iridocyclitis	+	-	-	-	+/-	-

A significant role in leading to the suspicion of optic neuritis is often played by pain with eye movements, which frequently occurs in this condition [1, 4]. Another differentiating symptom is optic disc edema, which can occur in ischemic optic neuropathy, uveitis, and in cases of papilledema, which is a manifestation of many diseases associated with increased intracranial pressure [5–7]. There are instances where optic disc edema occurs in the course of optic neuritis, but as stated by Petzold et al., this happens on average in only about 35% of patients [1]. It has been observed that the presence of optic disc edema in cases of optic neuritis is more common in Asian patients and when the inflammation affects both eyes [8].

Greater attention should also be paid to the patient's systemic and internal medicine symptoms, which are less likely to correlate with optic neuritis [1, 5, 6].

The most common symptoms reported by patients diagnosed with optic neuritis were a sudden decrease in visual acuity, color vision deficiency, and pain with eye movements. The first of these symptoms is undoubtedly one of the most frequent in the course of optic neuritis. The Optic Neuritis Study Group found blurred vision in 40% of the 448 patients studied [9]. Regarding color vision deficiency and their type, in a study conducted by Schneck et al., among a group of 438 patients, mixed red-green and blue-yellow color defects were present in the majority [10]. Petzold et al. mention pain with eye movements as one of the strongest indicators of optic neuritis [11]. In the Optic Neuritis Study Group study mentioned above, 92.2% of patients with vision impairment experienced pain that worsened with eye movements [9].

Patients largely experienced central visual field loss, which also raises suspicion and points towards the mentioned disease entity. In studies by Keltner et al., abnormalities were found in all visual fields of affected eyes at the onset of the disease, and in 74.7% of cases, in the eyes on the opposite side to where the inflammatory process was developing [12].

Among the patients presented in this work, the inflammation was retrobulbar, and demyelinating changes typical of MS were observed in MRI. As stated by Petzold et al., studies in which patients mainly had optic neuritis associated with MS showed a higher sensitivity of MRI compared to older studies. However, he also mentions that the sensitivity of MRI in detecting optic neuritis is only 20–44% considering all types of optic neuritis [1, 13].

It should be noted that the timing of patients' presentation to the emergency department was significant for the effectiveness of the applied therapy and further prognosis. In the cases mentioned above, the longest period after which a patient presented to the emergency department was 2 days – allowing for the rapid initiation of therapy, which resulted in significant improvement in visual acuity in most hospitalized patients. This hypothesis seems to be confirmed by a series of studies in which 8 patients presenting with a recurrence of the disease were given glucocorticosteroids; this resulted in no loss of vision function and relief of pain. The diagnoses of patients participating in this study included optic neuritis due to MS, RION, and NMO [2, 14]. Therefore, comparing the efficacy of glucocorticosteroids in treating optic neuritis due to various disease entities seems justified. A retrospective analysis of patients diagnosed with NMO showed that earlier (within 3 days of symptom onset) intravenous administration of methylprednisolone for the treatment of optic neuritis recurrence was associated with greater preservation of retinal nerve fiber layer thickness [2, 15]. The results of studies on optic neuritis due to MS also indicate early administration of glucocorticosteroids as a favorable prognostic factor in the context of the frequency of optic neuritis occurrence and preservation of more retinal ganglion cells after an episode of inflammation. However, these cases concerned the rat model of MS, so further studies are warranted to confirm this hypothesis [2, 16].

The therapy used with methylprednisolone improved the clinical condition of the patients, but it should be remembered that in a significant number of cases, it caused an increase in IOP. In studies on the efficacy of methylprednisolone in the treatment of acute optic neuritis, no improvement in visual acuity was found 6 months after the inflammation with the use of 1 g of this drug intravenously for 3 days, followed by its oral administration in small doses

for the next 11 days. However, an improvement in the visual field and color vision was noted [2, 17, 18]. Interestingly, intravenous administration of methylprednisolone contributed to a delay in the diagnosis of MS in patients with typical optic neuritis by 2 years [19], but this difference became less significant over time [2, 20]. An increase in IOP after systemic glucocorticosteroid administration usually occurs after chronic use; however, cases of a sudden increase in intraocular pressure after short and intensive glucocorticosteroid treatment have been reported [21, 22].

An observation that cannot be overlooked is the presence of demyelinating changes in the fMRI in 3 out of 4 patients diagnosed with optic neuritis. In these cases, the inflammation was retrobulbar, a type of inflammation strongly correlated with the future occurrence of MS in patients. As is known, about 20–50% of patients with a history of optic neuritis will develop MS [23]. According to studies conducted by Atkins et al. on the treatment of optic neuritis, the 10-year risk of developing MS in the group of patients with at least one T2 lesion on MRI was 56%, while the 10-year risk with a normal baseline MRI was 22% [24]. The criteria for diagnosing MS are the McDonald criteria, while the criteria for diagnosing optic neuritis share only a small component with them. This is one of the paraclinical criteria, which speaks of further contrast enhancement of the intrinsic signal within 3 months of symptom onset [1].

CONCLUSIONS

Color vision deficiency, pain with eye movements, and a sudden decrease in visual acuity were the most common symptoms among patients presenting to the emergency department with suspected optic neuritis; a significant problem was also the loss of the central visual field. However, these symptoms are not pathognomonic for this condition, so for its diagnosis, it is necessary to correlate them with the patient's overall condition and perform a series of diagnostic tests. Among the available diagnostic methods, MRI and VEP proved to be the most sensitive. A promptly made diagnosis allowed for the rapid administration of methylprednisolone, which contributed to an improvement in the clinical condition of the patients; however, in some cases, this treatment resulted in an increase in intraocular pressure.

CORRESPONDENCE
med. stud. Antoni Sieńko
Student Scientific Club at the Department of Ophthalmology, Faculty of Ophthalmology, Department of Ophthalmology, Faculty of Medical Sciences in Katowice, Medical University of Silesia in Katowice
40-514 Katowice, ul. Ceglana 35
e-mail: antsienko@gmail.com

ORCID
Antoni Sieńko – ID – https://orcid.org/0009-0001-6753-7895
Małgorzata Łabuś – ID – https://orcid.org/0009-0003-2799-4375
Tomasz Maciejczyk – ID – https://orcid.org/0009-0005-2517-2508
Sebastian Sirek – ID – https://orcid.org/0000-0002-3138-3011
Dorota Wyględowska-Promieńska – ID – http://orcid.org/0000-0001-5336-3460

References

1. Petzold A, Fraser CL, Abegg M et al. Diagnosis and classification of optic neuritis. Lancet Neurol. 2022; 21(12): 1120-34. http://doi.org/10.1016/S1474-4422(22)00200-9.
2. Toosy A, Mason DG, Miller DH. Optic neuritis. Lancet Neurol. 2014; 13(1): 83-99. http://doi.org/10.1016/s1474-4422(13)70259-x.
3. Koch-Henriksen N, Hyllested K. Epidemiology of multiple sclerosis: Incidence and prevalence rates in Denmark 1948–64 based on the Danish Multiple Sclerosis Registry. Acta Neurol Scand. 1988; 78(5): 369-80. http://doi.org/ 10.1111/j.1600-0404.1988.tb03672.x.
4. Rejdak R. Zapalenie nerwu wzrokowego – różnicowanie, diagnoza, leczenie. II Międzynarodowa Konferencja Szkoleniowa "Nowe spojrzenie na okulistykę"; 3 czerwca 2023. Kraków.
5. Morrow MJ. Ischemic optic neuropathy. Continuum (Minneap Minn). 2019; 25(5): 1215-35. http://doi.org/10.1212/CON.0000000000000767.
6. Bonnet C, Brézin AP. Uvéites, éléments d'orientation diagnostique. J Fr Ophtalmol. 2020; 43(2): 145-51. http://doi.org/10.1016/j.jfo.2019.03.038.
7. Osaguona VB, Kayoma DH. Etiology of Optic Disc Swelling in a Tertiary Care Center in Nigeria. Niger J Clin Pract. 2020; 23(12): 1690-4. http://doi.org/10.4103/njcp.njcp_333_20.

8. Du Y, Li K, Yang J et al. Disc swelling and mild initial visual acuity loss predict a better short-term visual acuity outcome in bilateral acute optic neuritis. J Clin Neurosci. 2012; 19(10): 1380-2. http://doi.org/10.1016/j.jocn.2011.10.020.
9. The clinical profile of optic neuritis. Experience of the Optic Neuritis Treatment Trial. Optic Neuritis Study Group. Arch Ophthalmol. 1991; 109(12): 1673-8. http://doi.org/10.1001/archopht.1991.01080120057025.
10. Schneck ME, Haegerstrom-Portnoy G. Color vision defect type and spatial vision in the optic neuritis treatment trial. Invest Ophthalmol Vis Sci. 1997; 38(11): 2278-89.
11. Petzold A, Wattjes MP, Costello F et al. The investigation of acute optic neuritis: a review and proposed protocol. Nat Rev Neurol. 2014; 10(8): 447-58. http://doi.org/10.1038/nrneurol.2014.108.
12. Keltner JL. Visual Field profile of optic neuritis. Arch Ophthalmol. 2010; 128(3): 330. http://doi.org/10.1001/archophthalmol.2010.16.
13. Miller DH, Newton MR, van der Poel JC et al. Magnetic resonance imaging of the optic nerve in optic neuritis. Neurology. 1988; 38(2): 175-9. http://doi.org/10.1212/wnl.38.2.175.
14. Plant GT, Sibtain NA, Thomas D. Hyperacute corticosteroid treatment of optic neuritis at the onset of pain may prevent visual loss: a case series. Mult Scler Int. 2011; 2011: 815068. http://doi.org/10.1155/2011/815068.
15. Nakamura M, Nakazawa T, Doi H et al. Early high-dose intravenous methylprednisolone is effective in preserving retinal nerve fiber layer thickness in patients with neuromyelitis optica. Graefes Arch Clin Exp Ophthalmol. 2010; 248(12): 1777-85. http://doi.org/10.1007/s00417-010-1344-7.
16. Dutt M, Tabuena P, Ventura E et al. Timing of corticosteroid therapy is critical to prevent retinal ganglion cell loss in experimental optic neuritis. Invest Ophthalmol Vis Sci. 2010; 51(3): 1439-45. http://doi.org/10.1167/iovs.09-4009.
17. Beck RW, Cleary PA, Anderson MM Jr et al. A randomized, controlled trial of corticosteroids in the treatment of acute optic neuritis. The Optic Neuritis Study Group. N Engl J Med. 1992; 326(9): 581-8. http://doi.org/10.1056/NEJM199202273260901.
18. Beck RW, Gal RL. Treatment of acute optic neuritis: a summary of findings from the optic neuritis treatment trial. Arch Ophthalmol. 2008; 126(7): 994-5. http://doi.org/10.1001/archopht.126.7.994.
19. Beck RW, Cleary PA, Trobe JD et al. The effect of corticosteroids for acute optic neuritis on the subsequent development of multiple sclerosis. The Optic Neuritis Study Group. N Engl J Med. 1993; 329(24): 1764-9. http://doi.org/10.1056/NEJM199312093292403.
20. Optic Neuritis Study Group. Multiple sclerosis risk after optic neuritis: final optic neuritis treatment trial follow-up. Arch Neurol. 2008; 65(6): 727-32. http://doi.org/10.1001/archneur.65.6.727.
21. Gupta SR, Suhler EB, Rosenbaum JT. Intravenous methylprednisolone can cause an acute, vision-threatening rise in intraocular pressure. J Clin Rheumatol. 2010; 16(8): 397-8. http://doi.org/10.1097/RHU.0b013e3181ffdd4f.
22. Francois J. Corticosteroid glaucoma. Ann Ophthalmol. 1977; 9: 1075Y1080.
23. Galetta SL. The diagnosis and management of optic neuritis. (2017).
24. Atkins EJ, Biousse V, Newman NJ. The natural history of optic neuritis. Rev Neurol Dis. 2006 Spring; 3(2): 45-56.

Authors' contributions:
Antoni Sieńko: data collection and analysis, interpretation of results, preparation of part of the article, critical review of intellectual content.
Małgorzata Łabuś: data collection and analysis, interpretation of results, preparation of part of the article, critical review of intellectual content.
Tomasz Maciejczyk: data collection and analysis, interpretation of results, preparation of part of the article, critical review of intellectual content.
Sebastian Sirek: research concept and design, project supervision, critical review of intellectual content, final approval of the article for publication.
Dorota Wyględowska-Promieńska: critical review of intellectual content, final acceptance of the article for publication.

Conflict of interest:
None.

Financial support:
None.

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