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# Clinical, demographic characteristics, and treatment protocols of optic neuropathies: Three-year follow-up experiences from a tertiary hospital in Turkey

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Burcu Taskiran Kandeger[1], Ozlem Tok[2], Levent Tok[2]

[1] Department of Ophthalmology, Konya City Hospital, Konya, Turkey
[2] Department of Ophthalmology, Suleyman Demirel University, Isparta, Turkey

**Keywords**
Optic Nerve Diseases; Nonarteritic Anterior Ischemic Optic Neuropathy; Optic Neuritis; Optic Nerve Injuries

**Abstract**

**Background:** This study was conducted to review the demographic and clinical characteristics, treatment protocols, and visual outcomes of patients with optic neuropathy.

**Methods:** This historical cohort study analyzed the clinical features of 91 patients with optic neuropathy followed up for three years at a university hospital in Turkey.

**Results:** Non-arteritic anterior ischemic optic neuropathy (NA-AION) was the most common group among the optic neuropathy subgroups (47.2%), and optic neuritis (ON) was the second most common group (38.5%). The mean age of symptom onset for NA-AION was 64.97 ± 12.15 years, significantly higher than the mean age of onset for ON (40.28 ± 15.52 years). Most of the patients with NA-AION had at least one systemic disease causing microangiopathy [51.1% had diabetes mellitus (DM), 33.3% had hypertension (HTN)]. Among the patients with ON, 51.4% were idiopathic, and 25.7% were multiple sclerosis (MS)-related ON cases. Patients with ischemic optic neuropathy (ION), ON, and traumatic optic neuropathy received pulse intravenous (IV) corticosteroids, and eleven patients with NA-AION received acetylsalicylic acid (ASA) therapy in addition to corticosteroids. There was a statistically significant increase in visual acuity in NA-AION and ON groups (P = 0.019). It was observed that the cases of ON peaked in the winter months in Turkey.

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Corresponding Author: Burcu Taskiran Kandeger
Email: drburcutaskiran@gmail.com

**Conclusion:** In the differential diagnosis between NA-AION and idiopathic ON, the presence of one or more vascular systemic diseases and mean age may be the main factors. IV steroid treatment given to patients with NA-AION in the acute phase may significantly improve visual acuity.

## Introduction

Optic neuropathy is a group of diseases with many different reasons for its etiology, and visual functions are impaired due to optic nerve damage. Ischemic, inflammatory, traumatic, compressive, infiltrative, and metabolic optic neuropathy are six main subgroups.[1] Visual field and color vision examination, retinal nerve fiber layer (RNFL) thickness analysis, and orbital and cranial magnetic resonance imaging (MRI) are the necessary auxiliary tests mainly used in diagnosis.[2] Common clinical features are decreased visual acuity or blurred vision, relative afferent pupillary defect (RAPD), impaired color vision, and visual field defect compatible with affected cell areas. Decreased visual acuity in the acute phase can range widely from mild impairment to severe visual loss (20/20 and no light perception). The optic disc may appear normal, edematous, pale, or atrophic. Optic neuropathy can result in degrees of retinal ganglion cell loss and axonal damage, resulting in severe and permanent visual function loss.[3,4]

There are limited studies of demographic and clinic data on common optic neuropathies.[3] There is no complete consensus regarding the treatment of some of its subgroups.[5-9] Our study evaluated the common optic neuropathies' demographic and clinical features, treatment protocols, and outcomes.

## Materials and Methods

Ninety-one patients with optic neuropathy who were followed up for three years at Suleyman Demirel University, Medical Faculty Hospital in Turkey, were included in the study. The data of patients whose records were complete and confirmed definite diagnosis of patients with optic neuropathy followed in our ophthalmology department between 2016 and 2019 were included in the study. Subjects with retinal and uveal inflammation signs were excluded from the study. A retrospective analysis of demographic and clinical characteristics and treatment protocols was performed. All patients were classified into subgroups after the same ophthalmologists completed ophthalmologic and systematic examinations.

The Ethics Committee of Suleyman Demirel University Medical Faculty approved our study (desicion number: 2019-133).. The study was conducted in line with the principles of the Declaration of Helsinki. Diagnostic criteria of optic neuropathies include:

**1. Ischemic optic neuropathy (ION)**

***A. Non-arteritic anterior ION (NA-AION):*** Acute, unilateral vision loss or blurry vision, optic disc edema, visual field loss compatible with disc edema (mainly altitudinal scotoma - scotoma in the horizontal half of the visual field), RAPD, presence of disc at risk in the other eye, normal levels of C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), absence of other ocular, systemic, or neurological diseases that might explain the patient's visual impairment.[6]

***B. Arteritic anterior ION (A-AION):*** Acute, unilateral, or bilateral vision loss (usually profound), pale optic disc edema (chalky white optic disc), relatively older age, systemic features such as anorexia, weight loss, myalgia, headache, jaw pain, and increased levels of CRP and ESR[10]

**2. Optic neuritis (ON)**

Subacute, unilateral vision loss or blurred vision, pain with eye movements, visual field defect (especially centrocecal scotoma), color vision loss, RAPD, relatively young age, absence of other ocular, systemic, or neurological diseases that might explain the patient's visual impairment are common ON clinical findings.[11]

***A. Multiple sclerosis (MS)-associated ON:*** Common ON clinical findings mentioned above and presence of one of the following criteria:

- MS history
- Barkhof criteria, supporting MS clinic in MRI and positivity of oligoclonal band in cerebrospinal fluid[12,13]

***B. Neuromyelitis optica (NMO)-associated ON:*** Common ON clinical findings mentioned above and presence of at least two of the three criteria:

- Transverse myelitis that extends continuously along with three or more vertebral segments in MRI
- Brain MRI does not meet the diagnostic criteria for MS
- NMO-immunoglobulin G (NMO-IgG) seropositivity[4,14]

***C. Systemic diseases-associated ON:*** Common ON clinical findings mentioned above and presence of active infectious disease (syphilis, cat scratch disease, etc.) or non-infectious inflammatory disease (Behcet's disease,

rheumatoid arthritis, sarcoidosis, etc.)[14]

***D. Idiopathic ON:*** Common ON clinical findings mentioned above, and normal neurological and systemic examination on follow-ups[14]

**3. Traumatic optic neuropathy**

Presence of vision loss, RAPD, color vision loss, visual field impairment within three weeks after orbital or head trauma that could not be explained by other causes[15]

**4. Compressive optic neuropathy**

Loss of vision, impaired color vision, visual field defect compatible with compression area, cupping, pallor in the neuroretinal rim, thinning of the RNFL, and compression of the optic nerve (due to tumor, thyroid orbitopathy, aneurysm, etc.) confirmed by cranial MRI[16]

**5. Infiltrative optic neuropathy:**

Subacute vision loss, impaired color vision, visual field defect, RAPD, optic nerve infiltration findings confirmed by orbital MRI[17]

**6. Metabolic optic neuropathy**

***A. Hereditary optic neuropathies:*** Leber's hereditary optic neuropathy (LHON): Subacute severe vision loss, deterioration in color vision and visual field defect, peripapillary telangiectasia, which symptoms develop first in one eye and approximately 6-8 weeks later in the other eye, and optic disc pallor, usually in men in the second or third decade, and in cases where vision loss cannot be explained by any other reason.[18]

Autosomal dominant atrophy (Kjer disease): Subacute mild vision loss, sectoral optic disc edema or pallor, color vision loss, visual field defect, usually in the first decade in men that could not be explained by any other reason.[18]

***B. Toxic optic neuropathy:*** Bilateral, painless, subacute (except methanol intoxication) vision loss, typically central and centrocecal visual field defects, history of using toxic agents on the optic nerve, and vision loss in cases where no other reason can be explained.[18]

All statistical analyzes were made using the SPSS software (version 22, IBM Corporation, Armonk, NY, USA). Descriptive statistics such as percentage, mean, and standard deviation (SD) were made initially. Chi-square test was used for comparison of categorical data. Paired sample t-test and Wilcoxon signed-rank test were used to compare the variables of the two dependent groups. A P-value $< 0.05$ was considered statistically significant.

## Results

***Demographic features*:** The total number of patients diagnosed with optic neuropathy was 91, consisting of 43 with NA-AION (47.2%), 2 with A-AION (2.1%), 18 with idiopathic ON (19.7%), 9 with MS-related ON (9.8%), 4 with NMO-related ON (4.4%), 4 with systemic diseases-associated ON (4.4%), 4 with traumatic optic neuropathy (4.4%), 4 with compressive optic neuropathy (4.4%), two with toxic optic neuropathy (2.2%), and one with autosomal dominant atrophy (1.0%). 43% were women (n = 40) and 57% were men (n = 51). While patients with NA-AION had male predominance (64.4%), there was female predominance in patients with ON (60%). The mean age of disease onset was 64.97 ± 12.15 (36-91 years) in NA-AION; 16.2% of the patients were under 50 years old. Both patients with A-AION were women, and the average age of onset of the disease was 76.5 years. The mean age of disease onset was 40.28 ± 15.52 (18-79 years) in ON. The demographic data of patients with optic neuropathy are summarized in table 1.

**Table 1.** Demographics of patients with optic neuropathy

| | No. of patients | Gender (women/men) | Age at onset (year) (mean ± SD) |
|---|---|---|---|
| Ischemic optic neuropathy | 45 | 17/28 | 64.97 ± 12.15 |
| Non-arteritic anterior | 43 | 15/28 | 64.44 ± 12.16 |
| Arteritic anterior | 2 | 2/0 | 76.50 ± 2.12 |
| Optic neuritis | 35 | 21/14 | 40.28 ± 15.52 |
| Multiple sclerosis-associated | 9 | 7/2 | 29.78 ± 10.97 |
| Neuromyelitis optica-associated | 4 | 4/0 | 61.00 ± 16.06 |
| Systemic disease-associated | 4 | 3/1 | 43.00 ± 12.83 |
| Idiopathic optic neuritis | 18 | 7/11 | 40.33 ± 13.75 |
| Traumatic optic neuropathy | 4 | 0/4 | 37.00 ± 15.30 |
| Compressive optic neuropathy | 4 | 2/2 | 56.75 ± 13.50 |
| Metabolic optic neuropathy | 3 | 0/3 | 32.00 ± 21.63 |
| Toxic | 2 | 0/2 | 44.00 ± 8.49 |
| Hereditary | 1 | 0/1 | 8.00 ± 0.00 |
| Total | 91 | 40/51 | 52.80 ± 18.67 |

SD: Standard deviation

***Clinical features:*** There were 43 patients in the NA-AION group. Patients who presented within the first two weeks after their symptoms started were included in the study. One patient had NA-AION one week after cataract surgery and was evaluated due to perioperative ION. Five of the patients had previous other eye involvement, and one developed other eye involvement during the follow-up period. The mean best-corrected visual acuity (BCVA) at presentation was 0.31 ± 0.40, and the final BCVA evaluated six months later was 0.35 ± 0.39 (P = 0.019) (Table 3). Almost all visual field defect patterns were observed; the most common visual field defect was in the inferior altitudinal scotoma. When comorbidities were evaluated, 70.4% had at least one systemic vascular disease in the NA-AION group. The most common was diabetes mellitus (DM) (51.1%), hypertension (HTN) rate was 33.3%, and 25.0% of patients had both DM and HTN. 16.2% of patients had ischemic heart disease, 11.6% had cerebrovascular disease (CVD), 9.3% had hyperlipidemia, and 9.3% had chronic obstructive pulmonary disease (COPD). There were two patients in the A-AION group; both patients had complaints of night sweating and fatigue and had ESR values of 72 and above and CRP values of 100 and above. Biopsy was not performed because they did not accept temporal artery biopsy. Visual acuities at presentation were hand motion and finger counting at two meters, and there was no change in visual acuity after treatment.

There were 35 patients with ON in the study. Of the patients, 25.7% (9 patients) were associated with MS, 11.4% (4 patients) were associated with NMO, 11.4% (4 patients) were associated with systemic diseases, and 51.4% (18 patients) were in idiopathic ON. Seven of the patients with MS were newly diagnosed after the ON attack. In the group associated with systemic diseases, two of the patients had Behcet's disease, one had rheumatoid arthritis, and one had the active infectious disease (syphilis). The rheumatologist added anti-metabolite drugs to patients with Behcet's disease and rheumatoid arthritis. The infectious diseases specialist consultation was requested for the patient with ON with a history of suspicious sexual intercourse. He was diagnosed with active syphilis infection. 54.3% of the patients with ON had optic disc edema at admission; optic disc appearance was normal in the rest. The complaint of pain was present in 45.7% and was most frequently present in patients with MS-associated ON with 66.7%. When the attack months were examined, it was found that they peaked in the winter months in Turkey, December, January, and February. The mean BCVA at presentation was 0.31 ± 0.32, and the final BCVA evaluated six months later was 0.61 ± 0.37 (P < 0.001) (Table 3). The most common visual field defect was centrocecal scotoma for the ON group.

At admission, the visual acuity of patients with traumatic optic neuropathy varied between no light perception and 0.4. Two patients were admitted after a traffic accident and two patients after isolated blunt head trauma. No penetrating trauma to the optic nerve was in the MRI of any of the patients, and they were evaluated in the indirect group. At the time of admission, the optic disc was pale in 2 patients and had normal appearance in two patients.

All patients with compressive optic neuropathy had a pituitary tumor in the etiology. Visual acuities varied between no light perception and 0.7, and there was no significant change in visual acuity during follow-up. One of the patients with metabolic optic neuropathy was followed up with toxic optic neuropathy after an unknown origin of tobacco consumption and one after radiotherapy due to nasopharyngeal carcinoma. One patient with progressive bilateral visual loss and temporal paleness of optic disc at the age of 8 was diagnosed with autosomal dominant atrophy, and the diagnosis was made clinically. The clinical characteristics of patients with optic neuropathy are presented in table 2.

***Treatment:*** In 49 patients with ischemic and traumatic optic neuropathy, 1 mg/kg of oral methylprednisolone treatment was started after three days of intravenous (IV) methylprednisolone and discontinued gradually. In 35 patients with ON, 1 mg/kg of oral methylprednisolone treatment was started after seven days of IV methylprednisolone and discontinued gradually. In addition to steroid treatment, 11 patients with ION received acetylsalicylic acid (ASA) treatment based on the neurologist's recommendation. In addition to steroids, anti-metabolite therapy was given to a patient with Behcet's disease and a patient with rheumatoid arthritis in the ON group. One patient with active syphilis had penicillin treatment (18-24 million units/day via IV divided q4-6hr × 10 days).

Two patients with compressive optic neuropathy (which were followed up with neurosurgery) had transsphenoidal tumor resection. It was recommended that the other two patients be followed up with the visual field examinations.

**Table 2.** Clinical findings of patients with optic neuropathy

| | Follow-up period (month) (mean ± SD) | RAPD presence [n (%)] | Color vision impairment [n (%)] | Optic disc appearance [n (%)] | | | Recurrence rate [n (%)] | Recurrence interval (month) (mean ± SD) |
|---|---|---|---|---|---|---|---|---|
| | | | | Normal | Edematous | Pale | | |
| Ischemic optic neuropathy | 9.42 ± 10.46 | 17 (37.8) | 38 (84.4) | 0 (0) | 45 (100) | 0 (0) | 6 (13.3) | 10.73 ± 40.54 |
| Non-arteritic anterior | 9.81 ± 10.54 | 17 (39.5) | 36 (83.7) | 0 (0) | 43 (100) | 0 (0) | 6 (14.0) | 11.23 ± 41.42 |
| Arteritic anterior | 1.00 ± 0.00 | 0 (0) | 2 (100) | 0 (0) | 2 (100) | 0 (0) | 0 (0) | - |
| Optic neuritis | 12.86 ± 9.06 | 22 (62.9) | 29 (82.9) | 16 (45.7) | 19 (54.3) | 0 (0) | 6 (17.1) | 11.18 ± 30.41 |
| Multiple sclerosis-associated | 14.67 ± 4.36 | 6 (66.7) | 7 (77.8) | 8 (88.9) | 1 (11.1) | 0 (0) | 2 (22.2) | 22.67 ± 47.41 |
| Neuromyelitis optica-associated | 14.25 ± 14.93 | 2 (50.0) | 4 (100) | 3 (75.0) | 1 (25.0) | 0 (0) | 3 (75.0) | 38.00 ± 44.00 |
| Systemic disease-associated | 15.00 ± 7.39 | 2 (50.0) | 3 (75.0) | 1 (25.0) | 3 (75.0) | 0 (0) | 0 (0) | - |
| Idiopathic optic neuritis | 11.17 ± 10.00 | 12 (66.7) | 15 (83.3) | 4 (22.2) | 14 (77.8) | 0 (0) | 1 (5.6) | 1.33 ± 5.66 |
| Traumatic optic neuropathy | 1.25 ± 0.50 | 2 (50.0) | 3 (75.0) | 2 (50.0) | 1 (25.0) | 1 (25.0) | 0 (0) | - |
| Compressive optic neuropathy | 3.50 ± 1.73 | 0 (0) | 1 (25.0) | 2 (50.0) | 0 (0) | 2 (50.0) | 0 (0) | - |
| Metabolic optic neuropathy | 16.67 ± 17.47 | 0 (0) | 1 (33.3) | 0 (0) | 1 (33.3) | 2 (66.7) | 0 (0) | - |
| Toxic | 7.00 ± 7.07 | 0 (0) | 0 (0) | 0 (0) | 1 (50.0) | 1 (50.0) | 0 (0) | - |
| Hereditary | 36.00 ± 0.00 | 0 (0) | 1 (100) | 0 (0) | 0 (0) | 1 (100) | 0 (0) | - |
| Total | 10.36 ± 9.85 | 41 (45.1) | 74 (81.3) | 20 (22.0) | 66 (72.5) | 5 (5.5) | 12 (13.2) | 9.59 ± 34.18 |

RAPD: Relative afferent pupillary defect; SD: Standard deviation

**Table 3.** Visual acuity of patients with optic neuropathy

| | N | Visual acuity at presentation (mean ± SD) | Visual acuity at last follow-up (mean ± SD) | Z | P |
|---|---|---|---|---|---|
| Ischemic optic neuropathy | 45 | 0.310 ± 0.400 | 0.350 ± 0.390 | -2.35 | 0.019 |
| Non-arteritic anterior | 43 | 0.320 ± 0.410 | 0.370 ± 0.390 | -2.26 | 0.024 |
| Arteritic anterior | 2 | 0.001 ± 0.000 | 0.020 ± 0.280 | -1.00 | 0.317 |
| Optic neuritis | 35 | 0.310 ± 0.320 | 0.610 ± 0.370 | -4.16 | < 0.001 |
| Multiple sclerosis-associated | 9 | 0.280 ± 0.280 | 0.710 ± 0.320 | -2.37 | 0.018 |
| Neuromyelitis optica-associated | 4 | 0.080 ± 0.150 | 0.230 ± 0.450 | -1.34 | 0.180 |
| Systemic disease-associated | 4 | 0.530 ± 0.310 | 0.830 ± 0.170 | -1.84 | 0.066 |
| Idiopathic optic neuritis | 18 | 0.330 ± 0.350 | 0.600 ± 0.380 | -2.73 | 0.006 |
| Traumatic optic neuropathy | 4 | 0.180 ± 0.170 | 0.230 ± 0.210 | -1.00 | 0.317 |
| Compressive optic neuropathy | 4 | 0.330 ± 0.380 | 0.400 ± 0.470 | -1.00 | 0.317 |
| Metabolic optic neuropathy | 3 | 0.400 ± 0.170 | 0.410 ± 0.450 | -0.45 | 0.655 |
| Toxic | 2 | 0.450 ± 0.210 | 0.470 ± 0.620 | -0.45 | 0.655 |
| Hereditary | 1 | 0.300 | 0.300 | - | - |
| Total | 91 | 0.310 ± 0.350 | 0.450 ± 0.400 | -4.78 | < 0.001 |

SD: Standard deviation

The patient who developed toxic optic neuropathy due to tobacco consumption of unknown origin stopped consumption, and the neurologist recommended ASA treatment. The patient who developed toxic optic neuropathy after radiotherapy and had autosomal dominant atrophy was only followed up, but no treatment was administered.

## Discussion

Optic neuropathies can be seen at almost any age and result in severe visual acuity and visual field loss. It is essential to identify disease subgroups and consult with other departments for a multidisciplinary approach and appropriate treatment protocols to preserve visual function.[14] The study aims to evaluate the demographic and clinic characteristics and treatment protocols of different subgroups of optic neuropathy.

***Demographic features:*** When all optic neuropathy subgroups were evaluated, we found that ION was the most common (49.5%), and ON was the second (38.5%) most common subgroup. Karti et al. reported that these two groups accounted for approximately 90.0% of their study.[3]

In the present study, there was male predominance (64.4%) in the NA-AION group and female predominance (60.0%) in the ON group. Similar rates have been reported in different studies. In our study, there were two patients with A-AION, both of them were women, and their mean age was 76 years; this is consistent with the literature reporting that A-AION is more common in older women.[19] It was observed that the cases of ON peaked in the winter months in Turkey. Patients with traumatic optic neuropathy and compressive optic neuropathy were diagnosed in the third and fourth decades, consistent with published reports.[15,16] All patients with traumatic optic neuropathy were men, as men were more exposed to trauma.

***Clinical features and treatment protocols:*** Fundoscopic examination of the optic nerve head yields important clues in optic neuropathies' diagnosis and subgroup classification. In NA-AION, there is always total or sectoral edema in the optic disc when visual loss begins. In addition, the disc may be hyperemic, and often splinter hemorrhages can be observed at the disc margin.[10] In the present study, disc edema was observed in all the patients in the NA-AION group since they were in the anterior group and visual loss had begun. In A-AION cases, the optic disc edema is often pale; its appearance is often described as "chalky white".[19] Both of our patients with A-AION had pale optic disc edema. Studies have shown that optic disc edema is observed in 35%-60% of cases of ON, and the remaining cases can have a normal appearance.[4] In the present study, disc edema was observed in 54% of the patients with ON.

The presence of additional systemic vascular disease was very high in the NA-AION group in the present study. Hayreh and Zimmerman reported a DM rate of 34% and an HTN rate of 45% in patients with NA-AION,[8] and Preechawat et al. reported a DM rate of 21% and an HTN rate of 35% in patients with NA-AION.[20] In the present study, the DM rate was 51.1%, and the HTN rate was 33.3%, higher than previous studies. We

observed that systemic vascular comorbidities played more important role in the etiopathogenesis of NA-AION.

Dyschromatopsy - color vision impairment - is a significant finding in the diagnosis of optic neuropathy. The rate of color vision impairment in patients with ON was 88.2% in Optic Neuritis Treatment Trial (ONTT);[21] Karti et al. reported it 77.5% in their study.[3] Similarly, in our research, we found the rate of color vision impairment to be 82.9%.

Visual field testing plays an essential role in diagnosing and classifying optic neuropathy. Although the type of scotoma cannot definitively determine the diagnosis, it provides important information for the diagnosis. The most common defect in NA-AION is altitudinal scotoma and in ON is central scotoma reported in previous studies.[8,12] All visual field defect patterns were observed in our study. The most common visual field defect in NA-AION was inferior altitudinal scotoma, and in ON was central scotoma, consistent with published reports.

ONTT and other studies showed that the high-dose IV steroids were effective in improving visual recovery, especially for contrast sensitivity.[21] In the present study, we observed statistically significant visual improvement after IV steroid treatment at 6 months in patients with ON ($P < 0.001$).

Visual acuity may range from no light perception to 1.00 (decimal) in ION.[10] Hayreh and Zimmerman, who published a large series of patients with ION, reported that 33% of patients had 1.00 (decimal) vision at the time of admission, and 21% had worse than 0.1 (decimal) vision.[8] In the present study, we found that only 16.2% of patients with NA-AION had 1.00 (decimal) vision at admission, and 37.2% had worse than 0.1 (decimal) vision. We think that this difference may be affected by the higher mean age of the patients in our study. In addition, it may be due to the higher rate of concomitant DM, which is one of the significant causes of microangiopathy in patients with NA-AION in our study.

In another study, Hayreh and Zimmerman divided the patients with NA-AION into two groups: those receiving steroid treatment (364 eyes) and those not receiving steroid treatment (332 eyes). After six months, they reported more significant visual improvement in the group that received steroid treatment in the acute period than in the group that did not receive treatment ($P = 0.001$).[8] Similarly, Prokosch and Thanos reported significant visual improvement after steroid (fluocortolone) treatment in a study with 60 patients with NA-AION.[22] Pakravan et al. reported that high-dose steroid treatment (43 patients) was not superior to a placebo (30 patients).[23] Similarly, Rebolleda et al. did not show beneficial effect in visual and anatomic outcomes of 10 patients with NA-AION.[24] There are conflicting results in the literature regarding the efficacy of steroid therapy in NA-AION.

In the present study, all patients with NA-AION received 1 mg/kg of oral steroid treatment after three days of pulse steroid treatment. A statistically significant visual improvement was observed when the controls were evaluated six months later. Visual improvement of 3 lines or more was considered a significant visual improvement. It was observed in 60% of patients at the end of 6 months. It has been shown that the anti-edema effect of steroid therapy accelerates the resolution of edema in patients with optic disc edema. Optic disc edema causes nerve ischemia and axon damage by creating a situation similar to compartment syndrome, especially where nerve cells, such as the lamina cribrosa, cannot expand. The earlier recovery of this compression of the optic nerve hypoplasia (ONH) may result in earlier normalization of axon functions and a better visual prognosis.[8]

The most important limitation of the present study is its retrospective design. In addition, the number of patients in some of our optic neuropathy subgroups was low. Our study was conducted in a single hospital and was not multicenter. Therefore, it is difficult to generalize the findings. Long-term prospective studies are needed to clarify optic neuropathies' subtypes and treatment protocols.

## Conclusion

This study provides valuable information about subgroups, clinical features, treatment protocols, and the visual outcomes of patients followed for common optic neuropathies for three years at a tertiary referral center in Turkey. We observed a high rate of systemic vascular comorbidity, especially DM, in patients with NA-AION. Age and the presence of additional systemic vascular diseases may be important in differentiating idiopathic ON and NA-AION. We found statistically significant visual improvements in patients with acute NA-AION after steroid therapy. Longitudinal studies in larger populations are needed to support and strengthen

our findings.

## Conflict of Interests

The authors declare no conflict of interest in this study.

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