Bilateralna optikonevropatija po zdravljenju z amiodaronom - prikaz primera

Amiodarone-associated bilateral optic neuropathy: a case report

Avtor / Author

Ustanova / Institute

Anela Bečić Turkanović[1], Dušica Pahor[1,2]

[1]Univerzitetni klinični center Maribor, Oddelek za očesne bolezni, Maribor, Slovenija;

[2]Univerza v Mariboru, Medicinska fakulteta, Katedra za oftalmologijo, Maribor, Slovenija

[1]University Medical Center Maribor, Department of Ophthalmology, Maribor, Slovenia;

[2]University of Maribor, Faculty of Medicine

Ključne besede:
amiodaron, optična nevropatija, vidna ostrina, vidno polje.

Key words:
Amiodarone, optic neuropathy, visual acuity, visual field.

Članek prispel / Received
28.10.2016
Članek sprejet / Accepted
08.11.2016

Naslov za dopisovanje / Correspondence
Anela Bečić Turkanović dr.med., Oddelek za očesne bolezni, Univerzitetni klinični center Maribor, Ljubljanska ulica 5, 2000 Maribor, Slovenija.
Telefon +386 23211630
Fax: +386 23312393
E–pošta: anela.becic@gmail.com

Izvleček

Namen: *Amiodaron je antiaritmik, ki se v klinični praksi pogosto predpisuje. Kljub pogosti uporabi pa so znani številni neželeni učinki; povzroča okvaro pljuč, ščitnice, kože, živčnega sistema, jeter in oči. Med očesnimi zapleti so najpogosteje opisovani roženični mikrodepoziti, v redkih primerih pa lahko pride tudi do optikonevropatije. Namen prispevka je predstaviti primer bilateralne toksične optikonevropatije tri mesece po uvedbi terapije z amiodaronom, ki je eden izmed redkih neželenih učinkov zdravljenja z amiodaronom.*

Poročilo o primeru: *Predstavljen je primer 51-letnega bolnika, ki je bil hospitaliziran na Očesnem oddelku Univerzitetnega kliničnega centra Maribor zaradi poslabšanja vida najprej na desnem nato še na levem očesu. Pri bolniku je bila tri mesece pred sprejemom zaradi motenj ritma po prebolelem perikarditisu uvedena terapija z amiodaronom. Opravljeni so bili kompletni oftalmološki pregled in ostale preiskave za izključitev znanih vzrokov optikonevropatije.*

Abstract

Purpose: *Amiodarone is a commonly prescribed anti-arrhythmic drug; however, its use is limited by many adverse effects. Amiodarone causes lung, thyroid gland, skin, central nervous system, liver, and eye disorders. Corneal microdeposits are the most common ocular adverse effects, while more serious tissue damage such as optic neuropathy can also occur. The aim of this report was to describe a case of a toxic optic neuropathy, which is one of the adverse effects of amiodarone treatment.*

Case report: *This is a case report of a 51-year-old patient hospitalized at the Department of Ophthalmology University Medical Center Maribor because of blurred vision, first in his right eye and later in his left eye. Three months before admission, the patient began treatment with amiodarone because of arrhythmia after acute pericarditis. The patient underwent a complete ophthalmic examination and other tests for all known optic neuropathy causes.*

Ob sprejemu je bolnik navajal meglen vid desnega, čez 7 dni pa še levega očesa. Razen obojestranske korneje verticilate in edema optičnega diska je bil oftalmološki pregled v mejah normale. Tako statična kot kinetična perimetrija sta pokazali obojestranske izpade v vidnem polju. Fluoresceinska angiografija in OCT preiskava papil sta pokazali eksudacijo na papilah vidnih živcev. Sedmi dan hospitalizacije je bila terapija z amiodaronom ukinjena. V naslednjih tednih je prišlo do izboljšanja subjektivnih težav. Edem leve papile se je resorbiral 3,5 tedna, desne pa 5 tednov po ukinitvi terapije z amiodaronom. Kinetična in statična perimetrija sta pokazali izboljšanje vidnega polja 10 tednov po ukinitvi terapije z amiodaronom.

Zaključek: *Toksična optikonevropatija, povzročena z amiodaronom, ima klinično sliko podobno nearteritični sprednji ishemični optikonevropatiji (NA-AION). Pomembno je, da toksično optikonevropatijo prepoznamo, saj lahko s prekinitvijo izpostavljenosti toksinu preprečimo ireverzibilne okvare optičnega živca in posledično vida. Zaradi pogoste klinične rabe amiodarona bi bile, kljub relativno redki incidenci toksične optikonevropatije, priporočljive redne letne kontrole pri oftalmologu.*

The patient initially presented with blurred vision in his right eye, and also in his left eye after 7 days. Except for corneal micro-deposits and optic disc edema, the results of an ophthalmic exam were normal. Static and kinetic visual field examinations showed bilateral visual field defects. Fluorescein angiography showed bilateral fluorescein dye leakage from the optic disc. Amiodarone treatment was terminated on the 7th day of hospitalization, and the patient reported improvement of vision in subsequent weeks. The edema in his left optic disc resorbed at 3.5 weeks and in his right optic disc at 5 weeks after termination of amiodarone treatment. Kinetic and static perimetry showed improvement in the visual field 10 weeks after the termination of amiodarone treatment.

Conclusions: *Toxic optic neuropathy had a similar clinical course as non-arteritic anterior ischemic optic neuropathy. Prompt diagnosis is crucial because treatment with amiodarone can prevent irreversible visual impairment. Because of the frequent clinical use of amiodarone, and despite the low incidence of toxic optic neuropathy, regular annual follow-up examinations should be performed.*

INTRODUCTION

Amiodarone is a commonly prescribed antiarrhythmic drug. However, its use is limited because of numerous serious adverse effects that can cause damage to many organs and tissues, such as the lung, thyroid, skin, nervous system, liver, and eye. According to Vassallo et al., amiodarone caused pulmonary toxicity in 1%–17% of the patients, blue-grey skin discoloration in 4%–9%, hypothyroidism in 6%, hyperthyroidism in 0.9%–2%, hepatitis and cirrhosis in < 3%, and rare cases (0.3% annually) of peripheral neuropathy. Ocular toxicity presents as corneal micro-deposits in > 90% of the patients and rare optic neuropathy in ≤ 1%–2% of the patients (1).

The duration, but not dose, of amiodarone treatment is a risk factor for neurotoxicity. However, there have been case reports describing patients with bilateral optic neuropathy after only 6.5 weeks of amiodarone treatment (2,3), and males are more commonly affected than females (1).

Although the pathophysiology of amiodarone-induced toxic optic neuropathy is still unclear, Mansour et al. reported lamellar inclusions within large axons without axon loss in a patient, and suggested that drug-induced lipidosis was responsible for this complication (4).

Patients with toxic optic neuropathy present with either acute or insidious monocular vision loss, or acute bilateral or insidious visual loss, while one third of these patients are asymptomatic (5). When compared with non-arteritic anterior ischemic optic neuropathy (NA-AION), toxic optic neuropathy presents with better visual acuity, bilateral involvement, mild optic nerve dysfunction, and resolution of disc edema

within months after the cessation of amiodarone treatment (1).

CASE PRESENTATION

A 51-year-old male with no previous ocular history who complained of blurred vision in his right eye (RE) was admitted to our department. After 1 week, the patient reported worsening of blurred vision in his RE and blurred vision in his left eye (LE). His past medical history included medically controlled arterial hypertension and acute pericarditis 3 months prior to his initial visit. Amiodarone therapy was initiated because of arrhythmia following pericarditis.

The patient underwent a complete ophthalmic examination including a color vision test. The best-corrected visual acuity was 1.0 in both eyes, although the patient reported blurred vision in his RE. The patient saw eight out of 15 color test plates in the Ishihara pseudoisochromatic color vision plate test with each eye. A clinical examination showed a slightly positive relative afferent pupillary defect in the RE, which was negative in the LE. A slit-lamp examination showed corneal micro-deposits in both eyes. A fundus examination showed a hyperemic and swollen optic disc in the RE and a normal disc in the LE (Figure 1 and 2).

Initial visual field testing with a Humpfrey Field Analyzer using a sita standard program showed visual field defects in the lower two quadrants between 15–30°, which progressed on the 6th day of hospitalization to the upper temporal quadrants between 20–30° in the RE. An initial static perimetry test of the LE showed was relatively normal, while visual field defects were noted on the 6th day of hospitalization in the upper temporal and partial upper nasal quadrants between 20–30°. Kinetic perimetry showed OCTOPUS perimeter concentric narrowing of the visual fields in both eyes.

On the 7th day after admission, the patient reported worsening of symptoms in his RE and also blurring vision in his LE. A fundus examination showed a slightly swollen optic disc in his LE and an additional visual field test showed exacerbation in both eyes (Figure 3a, b, c, d).

Fluorescein angiography (FA) at the onset of symptoms showed a bilateral dye leak, but FA after 1.5 month showed improvement in the RE but exacerbation in the LE (Figure 4a, b, c, d).

C-reactive protein, sedimentation rate, a complete blood count, electrolyte levels, creatinine, blood urea nitrogen, liver enzymes, antinuclear antibody, antineutrophil cytoplasmic antibody, urine analysis, angiotensin-converting enzyme, serological screening for Toxocara, Bartonella, Toxoplasmosis, Lyme borreliosis, and Treponema pallidum were all normal, except for slightly elevated levels

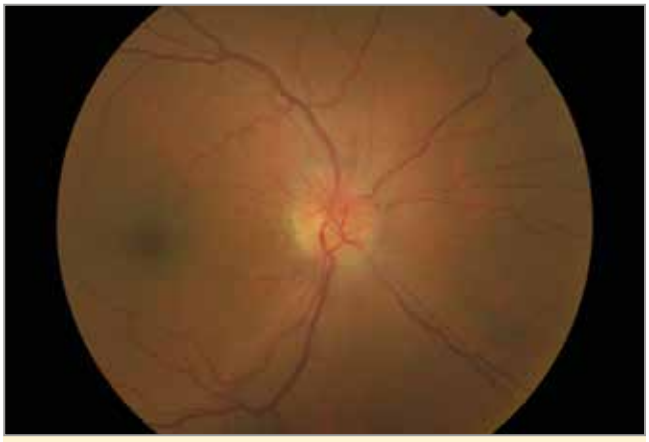

Figure 1: *Swollen and hyperemic optic disc in the right eye at admission.*

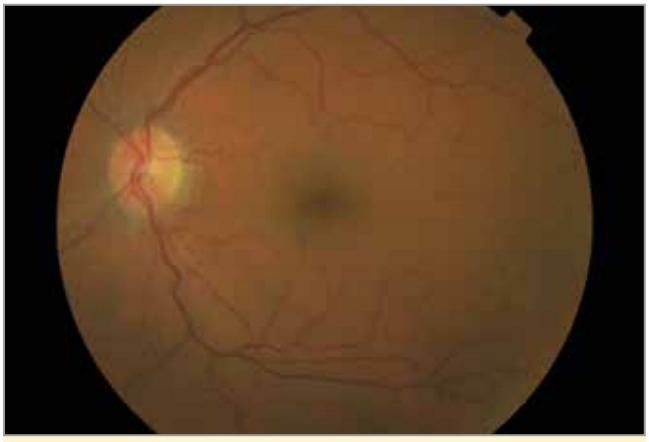

Figure 2: *Normal optic disc in the left eye at admission.*

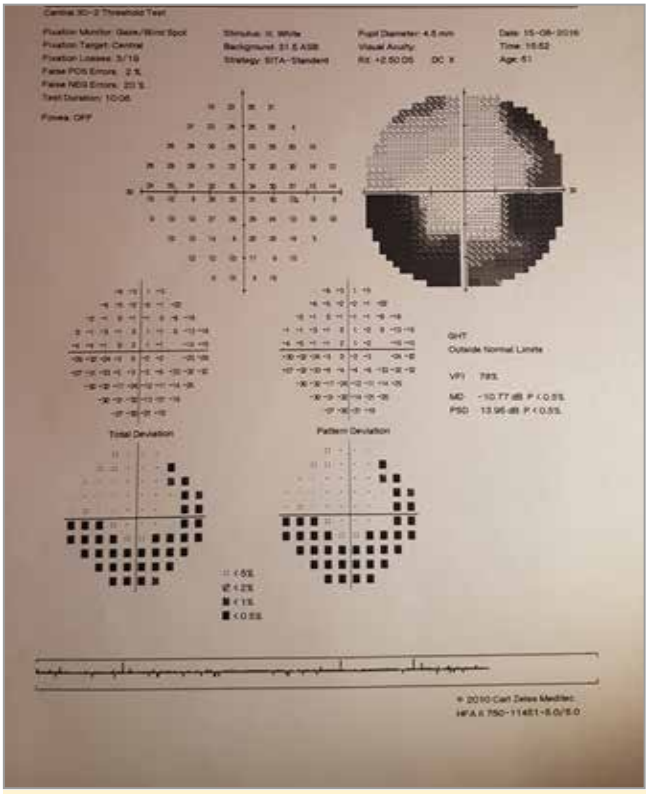

Figure 3a: *Visual field defects in the right eye 3 days after admission.*

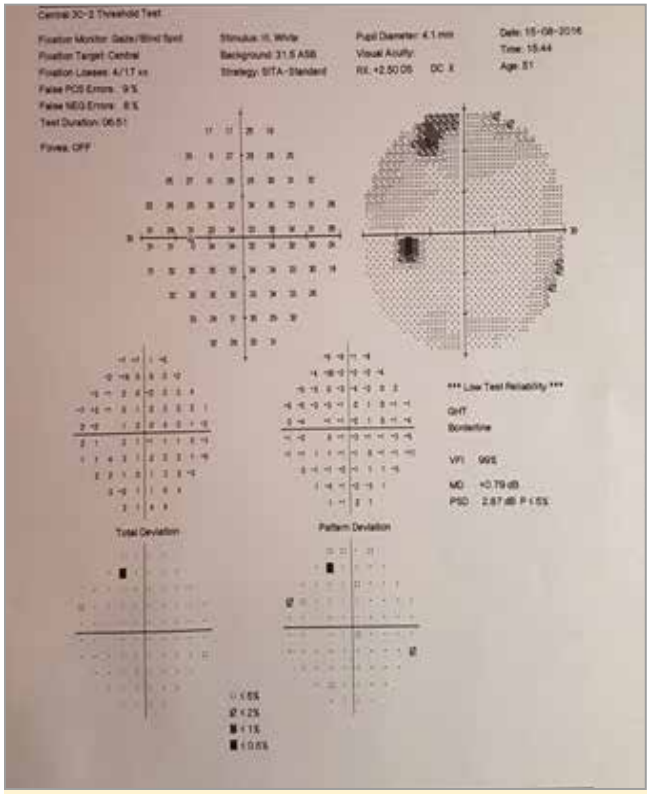

Figure 3b: *Visual field defects in the left eye 3 days after admission.*

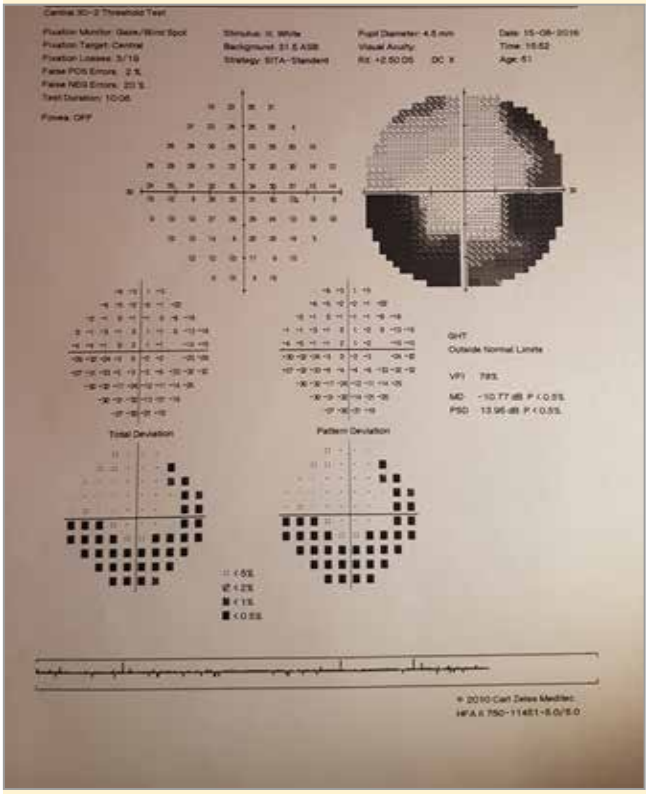

Figure 3c: *Visual field defects in right eye 7 days after admission.*

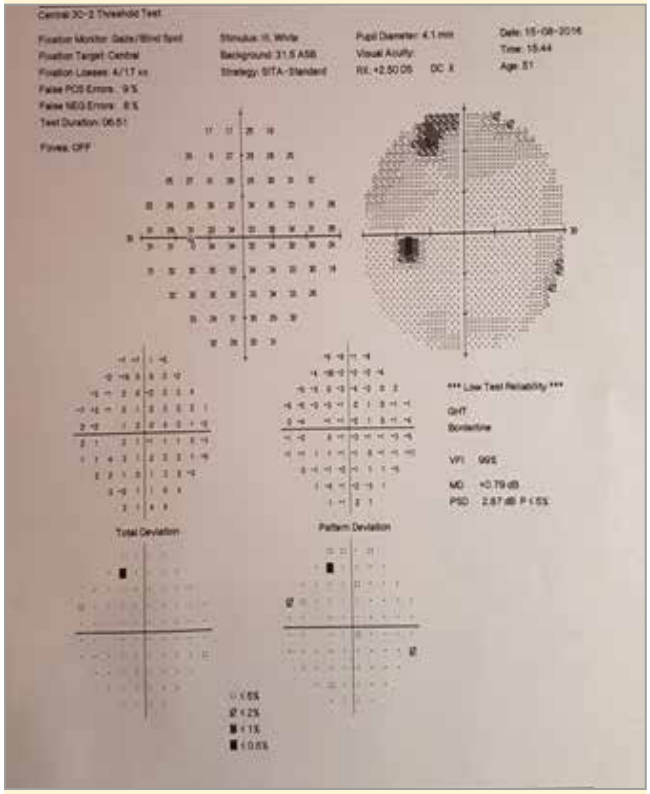

Figure 3d: *Visual field defects in the left eye 7 days after admission.*

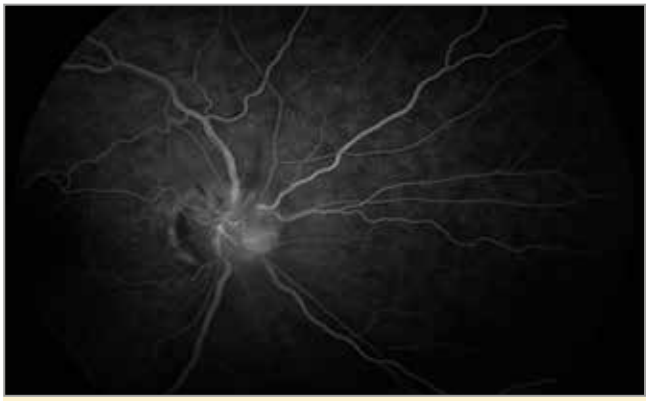

Figure 4a: *Initial fluorescein angiography with dye leakage from the optic disc in the right eye.*

Figure 4b: *Initial FA with dye leakage from the optic disc in the left eye.*

Figure 4c: *Fluorescein angiography 1.5 month after the first examination showed improvement in the right eye.*

Figure 4d: *Fluorescein angiography 1.5 months after the first examination showed exacerbation in the left eye.*

of homocysteine and thyroid-stimulating hormone (11,82 mIU/L).

A tuberculosis skin test, chest X-ray , head computed tomography and head magnetic resonance imaging and magnetic resonance angiography were all normal. The patient was examined by an internist, who diagnosed latent hypothirosis.

Amiodarone treatment was terminated on the 7th day

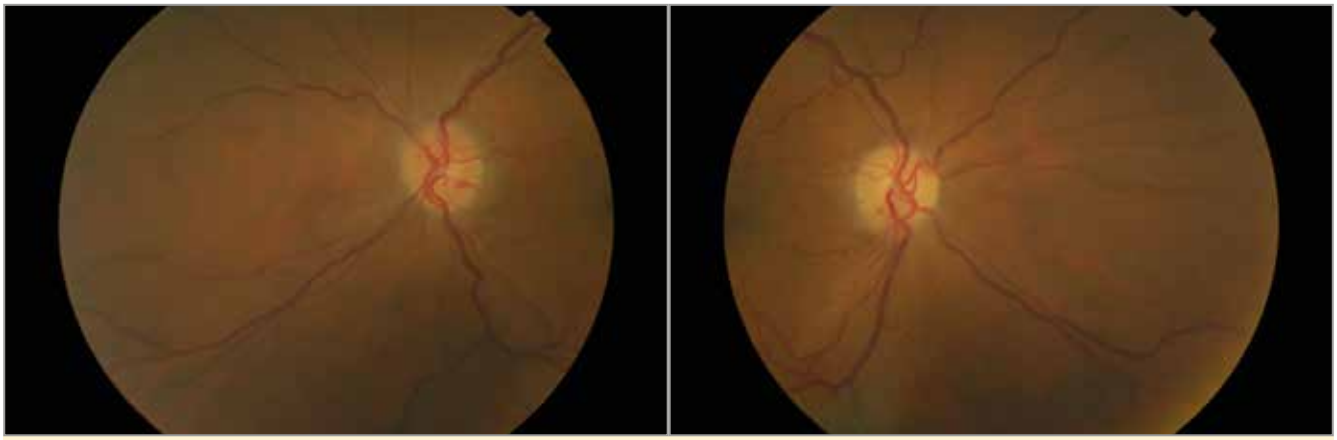

Figure 5: *Disappearance of optic disc edema in the right eye (left) and the left eye (right).*

of hospitalization. In subsequent months, the patient reported a decrease in blurred vision. A fundus examination showed normal optic discs without edema in both eyes (Figure 5).

DISCUSSION

Amiodarone-induced toxic optic neuropathy is still an underdiagnosed disorder (6). A reason for this underdiagnosis may be that the symptoms are similar to NA-ION (7, 8). Although past reports have suggested many criteria for distinguishing toxic optic neuropathy from NA-AION, such as an insidious onset, a milder degree of visual loss, a longer duration of disc edema, and being more bilateral than NA-AION, diagnosis is usually made by exclusion (9, 10). The precise medical history and all environmental risk factors should be considered when a patient presents with these clinical symptoms. Special attention should also be taken with younger patients, where NA-AION is less common (11, 12).

Our patient had an acute onset of symptoms, with a mild visual loss with visual field defects and bilateral optic disc edema. With the exception of arterial hypertension, there were no other risk symptoms of NAION in his medical history. The results of laboratory tests and image testing excluded other possible diagnoses. After discontinuation of amiodarone, the optic disc edema resolved in 5 weeks in the RE and in 3.5 weeks in the LE, and the patient reported a decrease of blurred vision.

Rapid diagnosis and prompt discontinuation with amiodarone is important for a good prognosis of visual acuity. We concur with other studies that suggested regular ophthalmic examinations, especially for visual acuity, as well as visual field and fundus examinations for patients who are treated with amiodarone. However, further studies with a larger number of patients are necessary before establishing proper guidelines for amiodarone-induced optic neuropathy screening.

REFERENCES

1. Vassallo P, Trohman RG. Prescribing amiodarone: an evidence-based review of clinical indications. JAMA. 2007;: p. 1312-22.
2. Orr CF, Ahlskog JE. Frequency, characteristics, and risk factors for amiodarone neurotoxicity. Arch Neurol. 2009;: p. 865-9.
3. Kervinen M, Falck A, Hurskainen M, Hautala N. Bilateral optic neuropathy and permanent loss of vision after treatment with amiodarone. J Cardiovasc Pharmacol. 2013;: p. 394-6.
4. Mansour AM, Puklin JE, O'Grady R. Optic nerve ultrastructure following amiodarone therapy. J Clin Neuroophthalmol. 1988;: p. 231-7.
5. Rod SP, Charles LB, Joseph P, Rashmi K, Lenworth NJ, Dennis WR, et al. Amiodarone-Associated Optic Neuropathy: A Critical Review. Am J Med. 2012;: p. 447-53.
6. Sharma P, Sharma R. Toxic optic neuropathy. Indian J Ophthalmol. 2011;: p. 137-41.
7. Feiner LA, Younge BR, Kazmier FJ. Optic neuropathy and amiodarone therapy. In Mayo Clin Proc; 1987. p. 702-17.
8. Leifert D, Hansen LL, Gerling J. Amiodarone-associated optic neuropathy: an independent syndrome? Three patients with bilateral optic neuropathy. Klinische Monatsblatter fur Augenheilkunde. 2000;: p. 171-7.
9. Nagra PK, Foroozan R, Savino PJ, Castillo I, Sergott RT. Amiodarone induced optic neuropathy. Br J Ophthalmol. 2003;: p. 420-2.
10. Macaluso DC, Shults WT, Fraunfelder FT. Features of amiodarone-induced optic neuropathy. Am J Ophthalmol. 1999;: p. 610-2.
11. Pahor A, Pahor D. Klinische Befunde bei Patienten mit nicht arteriitischeranteriorer ischämischer Optikusneuropathie (NAION) unter 50 Jahren. Klin Monatsbl Augenheilkd. 2016; 233(1): 66-71.
12. Pahor D, Pahor A. Gesichtsfelddefekte bei der Erstuntersuchung von Patienten mit nichtarteriitischer anterioren ischämischen Optikusneuropathie. Spektrum Augenheilkd. 2015; 29(4):130-6.