CASE REPORT

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Unrecognized severe obstructive sleep apnea as a dominant risk factor for non-arteritic anterior ischemic optic neuropathy in an apparently healthy patient

Neotkrivena teška opstruktivna apneja u snu kao dominantan faktor rizika od nearteritične prednje ishemijske optičke neuropatije kod naizgled zdravog bolesnika

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Abstract

Introduction. Non-arteritic anterior ischemic optic neuropathy (NAION) is typically manifested by sudden, painless vision loss in one eye, often immediately upon waking up. The visual loss remains fairly stable over time but can be followed by similar manifestations in the fellow eye. One of the main causes for NAION is impaired hemodynamic regulation, followed by hypoperfusion of the optic nerve head. Recognized risk factors include arterial hypertension, hyperlipidemia, diabetes mellitus, smoking, etc. **Case report.** A 43-year-old man with a sudden painless right eye vision loss and typical symptoms and signs of NAION is presented. The ophthalmological examination, followed by fluorescein angiography and visual field testing, confirmed the diagnosis, while medical history had not revealed any of the standard risk factors. During hospitalization, the medical staff noted that the patient was snoring, waking up during the night while feeling short of breath, followed by excessive daytime sleepiness, which was later confirmed by family members as symptoms that had been present for several months. The patient was referred to a somnologist, who, after diagnostics, confirmed the presence of severe, untreated obstructive sleep apnea (OSA) and indicated treatment with a continuous positive air pressure (CPAP) device. The patient responded well to treatment, showing subjective and objective improvements in sleep quantity and quality. No progression of right eye NAION or vision impairments in the left eye has been noted after 6 months from diagnosis and CPAP introduction. **Conclusion.** OSA was probably the main underlying cause of NAION in this patient. We point out that OSA screening in high-risk patients can contribute to the early diagnosis and prevention of NAION.

Key words:
diagnosis; optic neuropathy, ischemic; risk factors; sleep apnea, obstructive.

Apstrakt

Uvod. Nearteritična prednja ishemijska optička neuropatija (*non-arteritic anterior ischemic optic neuropathy* – NAION) obično se manifestuje iznenadnim, bezbolnim gubitkom vida na jednom oku, često odmah po buđenju. Gubitak vida ostaje prilično stabilan tokom vremena, ali može biti praćen sličnim manifestacijama na drugom oku. Jedan od glavnih uzroka NAION-a je poremećena hemodinamska regulacija, praćena hipoperfuzijom prednjeg dela optičkog nerva. Prepoznati faktori rizika su arterijska hipertenzija, hiperlipidemija, dijabetes melitus, pušenje, itd. **Prikaz bolesnika**. Prikazan je 43-godišnji muškarac sa iznenadnim, bezbolnim gubitkom vida na desnom oku i tipičnim simptomima i znacima NAION-a. Pregled oftalmologa, praćen fluoresceinskom angiografijom i ispitivanjem vidnog polja, potvrdili su dijagnozu, dok u anamnezi nije otkriven nijedan od standardnih faktora rizika. Tokom hospitalizacije, medicinsko osoblje je primetilo da bolesnik hrče, budi se tokom noći sa osećajem nedostatka vazduha i da pokazuje dnevnu pospanost, što su kasnije članovi porodice potvrdili kao simptome prisutne već nekoliko meseci. Bolesnik je upućen specijalisti za poremećaje spavanja, koji je nakon dijagnostike konstatovao

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tešku nelečenu opstruktivnu apneju u snu (*obstructive sleep apnea* – OSA) i indikovao lečenje aparatom za kontinuirani pozitivni vazdušni pritisak (*continuous positive air pressure* – CPAP). Bolesnik je dobro reagovao na lečenje, pokazujući subjektivno i objektivno poboljšanje u dužini i kvalitetu sna. Nije zabeležena progresija NAION-a desnog oka ili oštećenja vida na levom oku posle 6 meseci od dijagnoze i uvođenja CPAP-a. **Zaključak**. Glavni i osnovni uzrok NAION-a kod prikazanog bolesnika je najverovatnije bila OSA. Ukazujemo na to da utvrđivanje OSA-e kod bolesnika sa visokim rizikom može doprineti ranoj dijagnozi i prevenciji NAION-a.

Ključne reči:
dijagnoza; neuropatija, optička, ishemička; faktori rizika; apneja u snu, opstruktivna.

Introduction

Non-arteritic anterior ischemic optic neuropathy (NAION) is a major cause of blindness or severely impaired vision in adults. It is the most common cause of acute optic neuropathy in patients older than 50 years and the second most frequent form of optic neuropathy after glaucoma [1]. The typical clinical presentation of NAION includes a sudden, unilateral, and painless loss of vision. The rapid development indicates that NAION is caused by the sudden disruption of blood flow to the optic nerve, further supported by the fact that it is almost always unilateral. The main pathogenic mechanism is believed to be ischemic damage of the laminar and retrolaminar regions in the anterior part of the optic nerve, mostly due to hypoperfusion of short posterior ciliary arteries (SPCAs). Several risk factors have been linked to NAION, such as diabetes mellitus (DM), atherosclerosis, arterial hypertension, hyperlipidemia, and the use of some medications such as phosphodiesterase type 5 inhibitors. NAION is common in older individuals, given the increasing prevalence of a number of risk factors in this population [2]. NAION is a naturally progressive disease, and the contralateral eye involvement rate is 15–20% in the following 5 years [3].

Exposure to brief hypoxic episodes has been anecdotally linked to NAION in young and healthy people. Combat airplane pilots have reported visual field defects while performing high-G-force maneuvers [4, 5]. Given that neurons cannot rely on glycolysis as a source of energy, the nervous system, including the optic nerve and retina, is critically dependent on an uninterrupted oxygen supply for energy production *via* oxidative phosphorylation. Prolonged hypoxia or repeated hypoxic episodes have been shown to exhaust the nervous system's capacity for repair, resulting in more permanent structural damage.

Obstructive sleep apnea (OSA) is a chronic progressive disease with repetitive interruptions in ventilation during sleep due to complete or partial collapse of the pharyngeal part of the airway. This cessation of breathing is followed by a drop in oxygen saturation and/or waking [6]. OSA is the most common disorder in the spectrum of sleep-related breathing disorders, affecting more than 900 million people worldwide [7]. OSA is characterized by specific symptoms and signs, including excessive daytime sleepiness, pronounced snoring, and acknowledged episodes of breathing interruptions during sleep or upon waking. The diagnosis requires the occurrence of a minimum of five obstructive respiratory events, such as apneas, hypopneas, or waking associated with respiratory effort *per* hour of sleep, as measured by the apnea-hypopnea index (AHI) [8]. OSA is considered mild if AHI is 5–15 events *per* hour of sleep (5–15/hr), moderate if AHI is 15–30/hr, and severe if AHI is ≥ 30/hr [9].

Screening for OSA includes a detailed medical history of daily and nocturnal symptoms, physical examination and the use of standardized questionnaires, such as Epworth Sleepiness Scale (ESS) [10] and STOP-BANG scoring model [acronym stands for: snoring, tiredness, observed apnea, high blood pressure, body mass index (BMI), age, neck circumference, and gender] [11]. Definitive diagnosis is established by performing a full night polysomnography with monitoring of at least the respiratory flow, respiratory effort, pulse oximetry, snoring, and body position [9].

OSA has been associated with several ocular disorders, such as floppy eyelid syndrome, keratoconus, glaucoma, central serous chorioretinopathy, and retinal vein occlusion [12]. Recent studies have indicated that OSA can play an important role in NAION pathogenesis due to recurring hypoxia and hypoxemia, as well as endothelial dysfunction [13, 14]. Patients with NION have been found to have a greater prevalence of OSA and *vice versa*, and some studies showed that untreated OSA can lead to unilateral NAION progression or damage of the temporal peripapillary retinal nerve fiber layer in the contralateral optic nerve [15, 16].

Case report

A 43-year-old man presented to the ophthalmology outpatient clinic with unilateral (right) vision loss without pain or other symptoms. Ophthalmological examination showed that the patient had visual acuity (VA) of 1/60 in the right eye and 1.0 in the left eye. Intraocular pressure for both eyes was 14 mm Hg. A dilated fundus examination demonstrated optic disc edema in the right eye, and initial edema in the left eye was detected (Figure 1). In the left optic disc, a small cup-to-disc ratio of 0.3 was observed, consistent with the so-called 'disk-at-risk' (Figure 1). Fluorescein angiography was performed, confirming the suspicion of right NAION. Octopus (Haag-Strait) G Standard top visual field testing showed a right inferior altitudinal defect and an initial inferior altitudinal visual field defect in the left eye (Figure 2). He had a normal peripheral retinal examination. Ocular motility, alignment, and cranial nerve function testing appeared normal. The patient used corrective lenses for myopia (-6.0 Dsph).

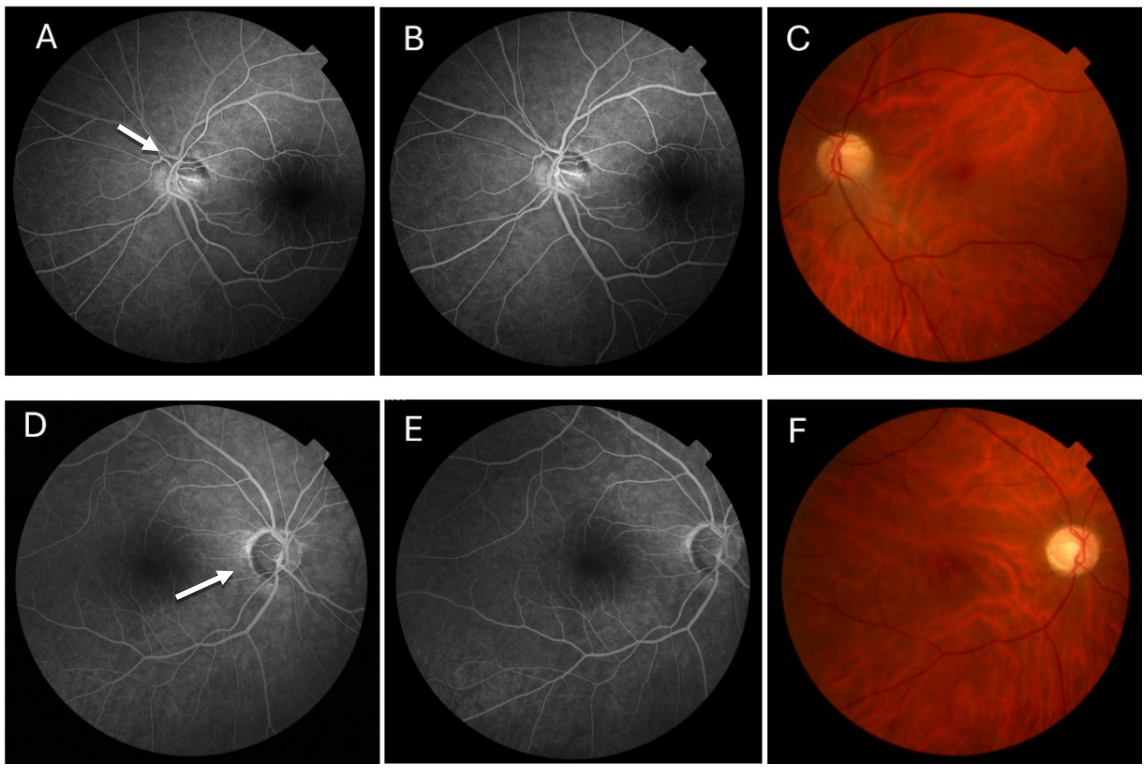

Fig. 1 – Fluorescence angiography (FA) in a 43-year-old patient with non-arteritic ischemic optic neuropathy (NAION) one month after the onset of symptoms. A, B) Partial NAION in the left eye. In the early A-V phase (0:23.7), on the optic nerve head, segmental (clockwise from 10.30 to 14.30 hrs) lesion of the papilla affected by an infarction, with extravasation of the contrast seen at the end of examination. The arrows in images A and D indicate optic disc involvement due to infarction. C) Left eye fundus. D, E) The first image of the right eye, taken at the beginning of the second minute, shows the optic disc as hypofluorescent temporally, becoming relatively fluorescent by the 11th minute, and fully fluorescent at the end of the FA – a consequence of NAION on the right eye. F) Right eye fundus.

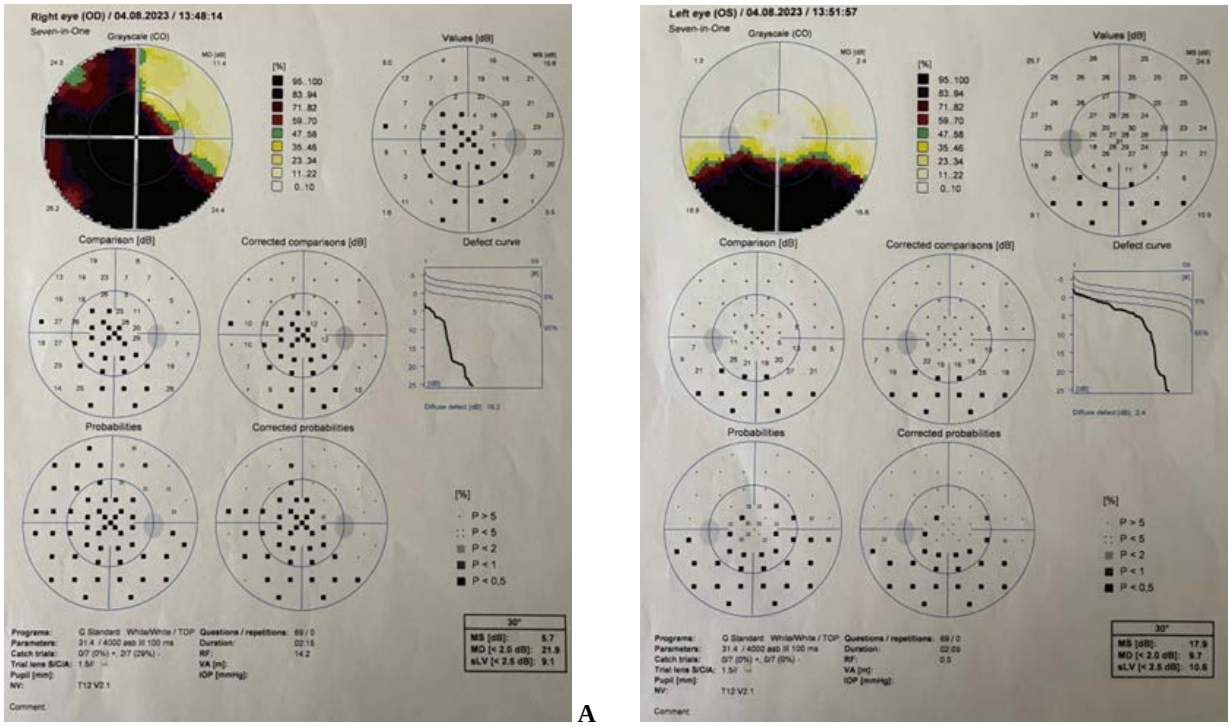

Fig. 2 – A) Right inferior altitudinal defect. B) Left eye initial inferior altitudinal visual field defect.

Testing for the three primary mutations associated with Leber's hereditary optic neuropathy (LHON) was conducted, and the results were negative. Next-generation sequencing of mitochondrial DNA was also performed and yielded negative results. Screening for the most common mutations in the *DNAJC30* gene associated with autosomal recessive LHON was also negative.

The patient was diagnosed with right NAION, based on his symptoms, visual field loss, and segmental optic disk edema typical for the condition.

The patient's medical record, examination, and autoanamnestic data did not show the presence of traditional risk factors for NAION, such as older age, arterial hypertension, DM, elevated blood lipids, or medication use, so the patient was admitted to the neurology department in order to perform further diagnostic procedures and exclude other reasons for sudden unilateral vision loss.

Brain magnetic resonance imaging, including orbits, was performed to exclude potential infiltrative processes and showed two unrelated chronic micro-ischaemic brain lesions (Figure 3).

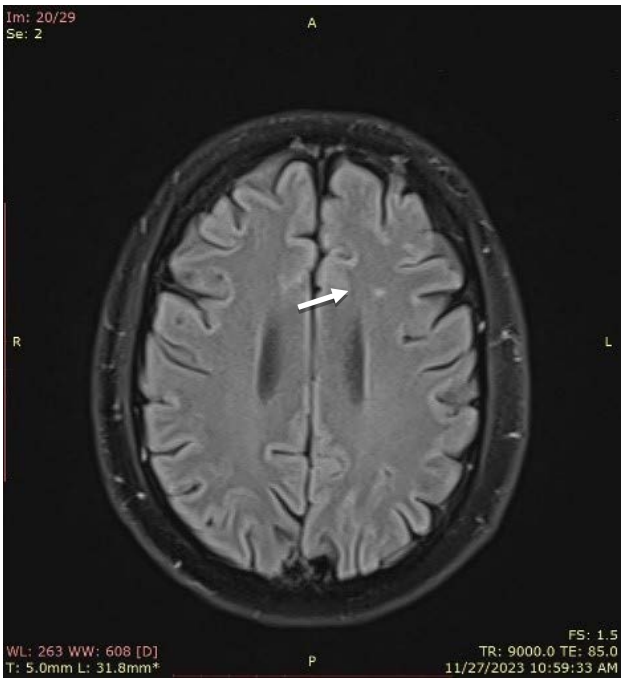

Fig. 3 – Magnetic resonance imaging shows two isolated chronic micro-ischaemic brain lesions.

During hospitalization, healthcare personnel in charge of patient care witnessed the patient's loud snoring, followed by breathing pauses, gasping for air, and waking during the night, including excessive daytime sleepiness (falling asleep while waiting for the examination, frequent napping during the day). Further interviews with the patient's family members confirmed that they had seen the patient exhibiting similar symptoms several months prior to vision loss.

Given that the patient exhibited a significant history of OSA symptoms, he was referred to a somnologist for further diagnostic procedures. Detailed anamnesis, examination, and the use of standardized questionnaires revealed a high probability of OSA (STOP-BANG score 5/8) with excessive daytime sleepiness (ESS score 14/24). Since no significant comorbidities or signs of other sleep-related disorders were present, a limited attended polysomnography was indicated and performed in hospital settings.

The patient underwent a single-night attended cardiorespiratory polysomnography using a type 3 polygraphy system (Alice Night One, Philips Respironics, the Netherlands). The analysis of all-night limited polysomnography records (Figure 4) revealed the presence of OSA syndrome with a very severe degree, with an AHI of 52.6/hr. Respiratory events were associated with a drop in peripheral arterial oxygen saturation to 79% (the lowest saturation values were just 79%). The oxygen desaturation index was 45/hr. Saturation with less than 90% of oxygen was present at 25% of the recording time. Respiratory events (apnea, hypopnea) were dominantly present when the patient was sleeping on his back.

The somnologist indicated the use of an auto-continuous positive airway pressure (CPAP) device with a nasal mask, which was very well accepted by the patient after initial titration (pressure range 4–10 cm H_2O). Further follow-ups after one week, one month, and six months of use showed adequate adherence and compliance (use more than 4 hrs for more than 70% of nights), with average AHI of less than 3/hr (normal range), and resolution of sleep related symptoms (snoring, apnea, non-restorative sleep) as well as excessive daytime sleepiness.

The patient was initially treated with corticosteroid therapy, antiplatelet therapy, and neuroprotective therapy. Subsequently, the patient was treated with a CPAP device. The follow-up ophthalmological examination after 6 months of the initial diagnosis showed no change in VA and no signs of NAION progression.

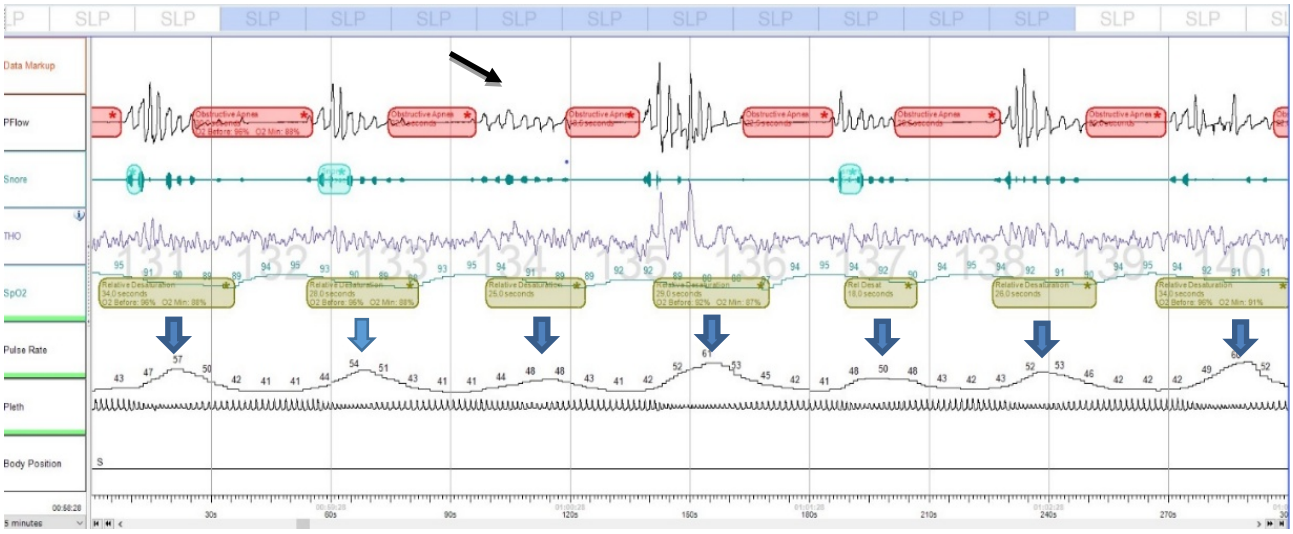

Fig. 4 – A 5-min excerpt of the limited polysomnography recording showing repetitive obstructive apneas (red field), followed by oxygen desaturation events (green field) and elevated heart rate (blue arrow).

Discusion

We presented an unusual case of NAION in a 43-year-old patient who, on admission, had no obvious associated risk factors for this disease. However, with careful observation and clinical history, followed by specialist examination and diagnostic procedures, the presence of unrecognized severe OSA was confirmed as a potential main risk factor for NAION.

NAION is a complex and multifactorial disease. Although the mechanisms underlying the pathogenesis of NAION have not been elucidated, it is generally accepted that it is intimately linked to impairments of microvascular regulation of the preliminary zone of the head of the optic nerve. The most widely cited hypothesis concerning the sequence of events that precede manifestations of NAION is that transient hypoperfusion (such as in cardiac dysfunction or pulmonary embolism) within the densely packed optic disc leads to infarction, followed by edema and consequent compression of the microvasculature in already constricted space of the optic canal, resulting in hypoperfusion. Thereby, the vicious circle infarct-edema-compression-infarct underlies the neuronal dysfunction with vision loss as its consequence [17].

Typical systemic vascular risk factors such as male gender, hypertension, DM, hyperlipidaemia, smoking, migraines, etc., are also the risk factors for NAION [18]. Most of these factors are not recognized in our patient, except for the male gender. Further diagnostic procedures have been performed to exclude other causes for sudden unilateral vision loss [19, 20]. Whole-genome sequencing has not been performed; therefore, genetic diseases cannot be excluded as the cause of vision loss in this patient.

A recent meta-analysis has found that patients with OSA have a four times higher risk of NAION [12]. Initial relations between OSA and NAION were noticed in a case report published in 1988, where the patient with severe OSA and normal visual function had bilateral optic disk edema, which resolved with the use of tracheostomy (treatment of choice for OSA before CPAP) [21]. Later studies have found that most of the NAION symptoms appeared in the morning, suggesting that some nocturnal events, such as blood pressure or sympathetic activity variations, could lead to or function as a trigger for NAION [22, 23]. OSA has been recognized as causing repetitive intermittent nocturnal hypoxia, followed by significant variations in sympathetic activity, with manifestations like cardiac arrhythmias, arterial hypertension, and hypotension episodes, so OSA gained interest in NAION research [24, 25].

Several studies have further researched the potential relationship between these two disorders, analyzing not only OSA-induced hypoxia and the effect on catecholamines but also the elevated cranial pressure on the optic nerve during apneic events, variations of ocular perfusion pressure, and the effects of OSA on other risk factors [15, 26]. Stein et al. [27] found that patients with diagnosed and untreated OSA have a 16% greater chance of NAION compared to subjects without OSA.

Although there is a strong association between OSA and NAION, OSA is rarely assumed by the ophthalmologist or other medical specialists. Even with the obvious symptoms and signs, it is estimated that 80–90% of OSA patients are still unrecognized and, therefore, untreated [14, 28].

Due to the estimated high prevalence of OSA in NAION patients, it has been suggested that patients with confirmed NAION should be screened for OSA and *vice versa* [26].

In the presented case, OSA was not previously diagnosed, but the educated clinical observation followed by extended anamnesis and heteroanamnesis led to proper diagnostics and treatment.

In their recent study, Li et al. [16] have proven that untreated OSA has the potential to cause subclinical nerve damage in the contralateral optic nerve in patients with NAION, followed by temporal quadrant thinning of the peri-

papillary retinal nerve fiber layer, probably because the papillomacular bundle located there has the highest oxygen deficit sensitivity.

Chang et al. [29] have shown that one of the main risk factors for second eye involvement in NAION patients with moderate to severe OSA was noncompliance with CPAP treatment, raising the chances by more than four times. In our case, the patient showed continuous adherence and compliance to CPAP treatment, which led to the elimination of his OSA signs and symptoms. He also had no signs of NAION progression in his right eye, nor signs of NAION in his left eye after 6 months of follow-up.

Conclusion

The case we present hereby supports the inclusion of OSA in the risk factors for NAION. Moreover, this case emphasizes the importance of screening for OSA in all patients in the high-risk groups for NAION and patients already diagnosed with NAION. Given the reported association between NAION and OSA, a thorough history should be screened for OSA when NAION is diagnosed. Furthermore, perhaps most importantly, screening for and diagnosing OSA should alert clinicians to conduct an ophthalmological examination. Such screening could easily be implemented in primary care, reducing the time from presentation to diagnosis of NAION. Importantly, OSA is a modifiable risk factor and can be successfully treated with CPAP. Implementation of this simple and effective algorithm has the potential to significantly reduce the risk of optic disk lesions, as well as other microvascular complications precipitated by hypoxic damage. Together, this will result in early diagnosis and prevention of irreversible degenerative changes of the optic nerve.

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