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Case Report

Preservation of Vision after Early Recognition of Anterior Ischemic Optic Neuropathy in a Patient with Sepsis

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Keywords

Sepsis · Optic neuropathy · Shock-induced anterior ischemic optic neuropathy · Non-arteritic ischemic optic neuropathy · Early recognition · Case report

Abstract

Non-arteritic ischemic optic neuropathy (NAION) can rarely occur in the setting of sudden vascular compromise, especially in patients with a "disk-at-risk" appearance. Anemia and hypotension are believed to be the main precipitators of shock-induced NAION. Early recognition of this phenomenon can prevent further visual loss and result in partial visual recovery. We here present a 56-year-old patient who developed NAION characterized by optic disc edema in both eyes and visual loss in the left eye secondary to hypotension in the setting of septic shock. He received aggressive blood pressure management (stopping all his anti-hypertensives, hydration, and midrodrine) which resulted in stabilization of vision in the right eye and likely prevented further visual loss in the left eye.

Introduction

Non-arteritic ischemic optic neuropathy (NAION) is the most common cause of optic neuropathy in adults [1]. It commonly presents with unilateral, sudden-onset visual loss and affects older patients with vascular risk factors [1]. The pathophysiology of NAION is still a subject of debate. It is believed to occur secondary to an acute precipitating event followed by

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the development of compartment syndrome in patients with "disc at risk" appearance – patients with a small cup to disc ratio [1]. An acute event can trigger hypoperfusion in these arteries which are susceptible due to their small size, resulting in ischemia and subsequent inflammation. In rare circumstances, generalized failure of the circulatory system to supply central and peripheral tissues, such as in the event of severe hemorrhage, may result in shock-induced anterior ischemic optic neuropathy (SIAION) [1]. SIAION is more commonly bilateral (as opposed to unilateral in NAION) and typically more severe (vision light perception or worse vs. rarely light perception in NAION). Hypotension, "disk-at-risk" appearance, and anemia may serve as possible risk factors for developing SIAION [1].

While SIAION is a well-known entity, very few cases of sepsis-induced NAION have been reported in the literature. We here report a 56-year-old patient who developed NAION secondary to septic shock due to a fungal infection.

Case Description

A 61-year-old man was referred urgently to ophthalmology for complete loss of vision in his left eye. He was admitted to hospital for sepsis due to *Candida albicans* and had a past medical history of hypertension, dyslipidemia, and type 2 diabetes. He also had a history of urinary retention due to phimosis from balanitis xerotica obliterans and urethral strictures. His medication included aspirin 81 mg, bisoprolol, ezetimibe, empagliflozin, gliclazide, metformin, ramipril. He presented to hospital 14 days prior to vision loss with vomiting, fever, and flank pain secondary to pyelonephritis and left hydronephrosis. A left nephrostomy tube was inserted, and he subsequently developed hypoxia and pulmonary edema, and blood cultures grew *C. albicans*. He was treated with fluconazole and caspofungin under the guidance of infectious disease.

He reported sudden vision loss in his left eye 14 days after his initial presentation to the hospital. A "Code Stroke" was called, and CT/CTA of the head and neck were normal. Hemoglobin was 103 g/L (normal 125–170 g/L in men), C-reactive protein was 47.3 mg/L (normal less than 5.0 mg/L), and sedimentation rate was 75 mm/h (normal 0–10 mm/h in men). Ophthalmology was consulted and found a visual acuity of 20/20 OD and NLP OS. There was a left RAPD, and dilated fundus examination showed mild right optic disc edema with multiple cotton wool spots in the right eye and left pallid optic disc edema (Fig. 1). OCT of the retinal nerve fiber layer revealed increased thickness of 127 μM OD and was not measurable due to a segmentation error OS (Fig. 2a). A review of his medical record revealed that his blood pressure was very low, measuring 98/40 immediately before the episode of vision loss. He had no symptoms of giant cell arteritis. A diagnosis of anterior ischemic optic neuropathy related to systemic hypotension and sepsis was made, and his blood pressure medications (ramipril and bisoprolol) were held. He was also encouraged to hydrate well and was prescribed salt tablets and midodrine 10 mg PO TID. His blood pressure subsequently improved, and his visual function stabilized. At the 1-month follow-up, the optic disc edema in both eyes was significantly improved and essentially resolved. OCT retinal nerve fiber layer revealed resolved edema OU and mild thinning OS (Fig. 2b). His right eye remained 20/20 with no visual field defect, and his left eye was NLP (Fig. 3). The CARE checklist [2] has been completed by the authors for this case report – attached supplementary material (for all online suppl. material, see https://doi.org/10.1159/000530326).

Discussion

This is a rare case of NAION in the context of sepsis in an isolated manner as previously reported cases have additional significant risk factors such as renal failure on dialysis, extensive blood loss, and severe burns, which may induce hypotension and contribute to

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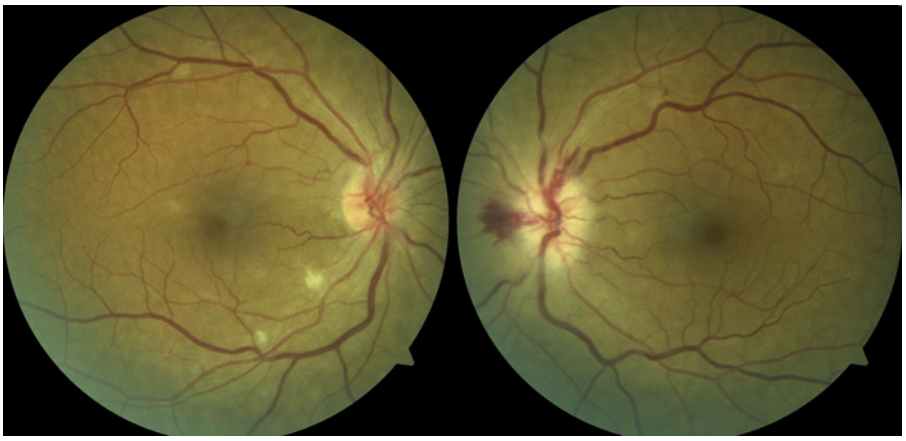

Fig. 1. Optic disc photos at presentation demonstrating very subtle right optic disc edema with cotton wool spots in the retina. The left optic nerve had pallid optic disc edema with peripapillary hemorrhages.

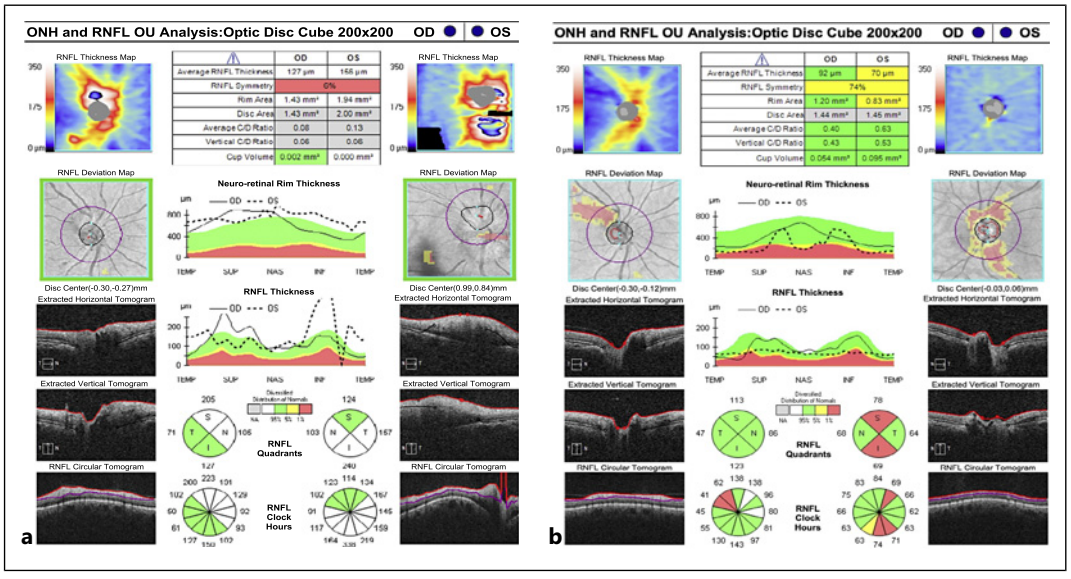

Fig. 2. Optical coherence tomography of the retinal nerve fiber layer at initial (**a**, left) and final presentation (**b**, right).

hypoperfusion of posterior ciliary arteries. Wortmann et al. [3] reported a 53-year-old female with dialysis-dependent renal failure who developed AION following sepsis. Vallejo et al. [4] reported a 27-year-old patient admitted to intensive care unit with 85% body surface area burns who had a complicated course with multiple episodes of septic shock, renal failure, and thrombocytopenia, requiring extensive fluid resuscitation. Cullinane et al. reported on nine patients who developed NAION after a traumatic event requiring extensive hemodynamic support. One patient in this cohort developed sepsis, which may have contributed to SIAION [5].

Early recognition and treatment of SIAION are essential to prevent further visual loss and may partially restore vision in the effected eye. Connolly et al. [6] reported 3 patients who developed hypotension-induced NAION. Case 1 resulted from aggressive blood pressure control and was treated with stopping anti-hypertensive agents, IV dopamine, and norepinephrine

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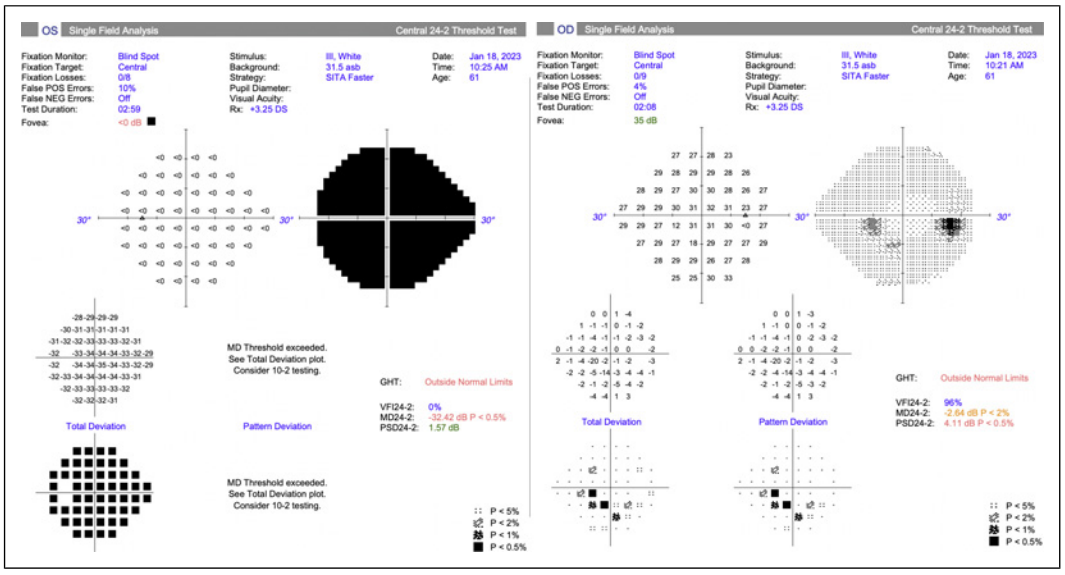

Fig. 3. Humphrey visual fields demonstrating severe depressed points in the right eye and complete defect in the left eye.

infusions. This patient regained a visual acuity of 20/40 OD and 20/70 OS from NLP OD and LP OS at nadir. Case 2 resulted from hypotension due to urinary excretion of pancreatic enzymes following kidney-pancreas transplantation and was treated with albumin and IV saline infusions. This patient regained a visual acuity of 20/40 OD and 20/70 OS, compared to NLP OD and LP OS at nadir. Case 3 developed NAION following hemodialysis and was found to be hypotensive and severely anemic. His visual acuity recovered to 20/100 (from 20/800 nadir) following blood transfusion and IV saline infusions. Jaben et al. reported a patient with bilateral SIAION due to hypovolemia and anemia following coronary artery bypass surgery. The patient was treated with multiple blood transfusions and recovered a visual recovery of 20/20 OD and 20/30 OS, from 20/400 OD and 20/200 OS at nadir [7]. In our patient, hypotension was treated by stopping anti-hypertensive agents, salt tablets, midodrine, and encouraging oral fluid intake. Without aggressive treatment, both eyes may have developed SIAION as indicated by the presence of ischemic changes in the eye with the intact visual acuity (Fig. 1). Previous cases and the case presented here emphasize the importance of aggressive measures to correct hypotension as well as other risk factors which may precipitate SIAION.

SIAION is likely the main culprit in our case given the patient's age, vascular risk factors, temporal relationship with hypotension and anemia, and unremarkable ocular exam apart from optic disc edema. However, it is important to maintain a broad differential diagnosis in patients with bilateral optic disc edema, which includes increased intracranial pressure, hypertensive crisis, infection/inflammation, toxic/nutritional conditions, and demyelinating conditions. Therefore, a detailed history and ocular exam should be obtained for all patients with bilateral optic disc edema.

In summary, this is a rare case of NAION due to septic shock in absence of other significant risk factors. It is likely mediated by hypotension resulting in hypoperfusion of the optic disc head in susceptible individuals with vascular risk factors or a "disk-at-risk" appearance. Early recognition and aggressive blood pressure control can prevent further visual loss and, in certain cases, may result in partial visual recovery.

Statement of Ethics

This research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. Ethical approval is not required for this study in accordance with local or national guidelines. Written and informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images.

Conflict of Interest Statement

The authors have no conflict of interest to disclose.

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Author Contributions

Conception and design, acquisition of data, analysis and interpretation of data, drafting the manuscript, revising it for intellectual content, and final approval of the completed manuscript: Amir R. Vosoughi and Jonathan A. Micieli.

Data Availability Statement

All available data are included in the case description. Further inquiries can be directed to the corresponding author.

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