**Bilateral Simultaneous Anterior Ischemic Optic Neuropathy in a Young, Healthy Man**

While a small cup-disc ratio is the predominant risk factor in the development of anterior ischemic optic neuropathy (AION), other hemodynamic factors likely play a part in its occurrence. A case of bilateral AION in a young, healthy man is presented, in whom multiple factors may have contributed to vision loss.

**Report of a Case.** A 42-year-old male pilot underwent myopic photorefractive keratectomy (−2.00 OD and −2.25 OS). Five weeks postoperatively, he began taking 0.5% timolol, 2 times per day in each eye, for persistent steroid-induced ocular hypertension (30 mm Hg). Three days later, he commenced a 5-day backpacking trek, ascending from sea level to an elevation of 6000 ft. On the third day of hiking, he experienced headaches and bilateral, intermittent photopsias, which he described as slow-moving streaks of light in his inferior field of vision, lasting for several minutes. While his headaches subsided after completion of the hike, his photopsias persisted for 8 days, at which time, he underwent ophthalmic examination. His medications included timolol, 2 times per day in each eye, and 0.1% flurometholone acetate, 3 times per day in each eye. He did not have a significant medical history, history of migraine, or ocular history.

On examination, his visual acuity without correction was 20/20 OU. Intraocular pressure was 16 mm Hg OD and 17 mm Hg OS. He had no relative afferent pupillary defect. Slitlamp biomicroscopy disclosed a well-healed corneal ablation zone in each eye. Ophthalmoscopy revealed bilateral optic nerve swelling, with a cup-disc ratio of 0.1 OU ([Figure 1](#)). Automated static perimetry disclosed bilateral inferior altitudinal visual field defects ([Figure 2](#)). His blood pressure at that time (118/70 mm Hg) was unchanged in comparison with his blood pressure prior to the initiation of timolol (114/70 mm Hg). Findings on magnetic resonance imaging of the brain and orbits with contrast and lumbar puncture, including opening pressure, were normal. Results of laboratory evaluation including prothrombin time, partial thromboplastin time, international normalized ratio (for anticoagulant monitoring), protein C and S, viscosity, factor V Leiden, homocysteine levels, antithrombin III activity, anticardiolipin and lupus anticoagulant, erythrocyte sedimentation rate, C-reactive protein, antinuclear antibody, rapid plasma reagin, microhemagglutination-Treponema pallidum, Lyme antibody, complete blood cell count, and angiotensin-converting enzyme, were normal. A low positive antibody titer of *Bartonella henselae* and *B quintana* (immunoglobulin G, 1:64; immunoglobulin M, <1:20 for each antibody) by enzyme-linked immunosorbent assay was not confirmed by the more clinically validated immunofluorescent assay.

The patient was diagnosed as having bilateral AION and began taking aspirin, 325 mg/d, and 0.2% bromonidine tartrate, 3 times per day in each eye. Four months later, he had an uncorrected visual acuity of 20/20 OU, mild inferior visual field defects (Figure 2), and superior segmental disc pallor.

**Comment.** This patient’s signs and symptoms, work-up findings, course, and outcome are consistent with a diagnosis of AION. Multiple factors are likely to have played a role, each involving hemodynamic alterations at the optic nerve head. This patient had a small cup-disc ratio, a disc configuration regarded as a risk factor for anterior ischemic optic neuropathy.1,2

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**Figure 1.** Fundus photographs demonstrate bilateral optic nerve edema.
However, the bilateral, simultaneous onset suggests that additional risk factors may have played a role. The effects of systemically absorbed topical beta blockers include decreased cardiac output, heart rate, and blood pressure, as well as adverse effects of bronchospasm and arrhythmias. Hayreh et al suggest that, due to their potential ability to limit autoregulation during nocturnal hypotension, the use of topical beta blockers may be a risk factor for non-arteritic AION in susceptible individuals. Various compensatory adaptations to exertion and altitude occur, including increased heart rate and cardiac output. The recent introduction of a beta blocker may have impaired these critical autoregulatory mechanisms, leading to inadequate optic nerve head perfusion under these conditions. The transient headache while at a high altitude may have been another early manifestation of poor adaptation to altitude.

The development of bilateral AION in this patient was likely multifactorial. He had a “disc at risk,” underwent photorefractive keratectomy, and was subsequently exposed to multiple potentially contributing factors, including initiation of topical beta blocker medications followed by prolonged exertion at an elevated altitude, all of which might cause alterations in optic nerve head perfusion. While none of these factors individually may be regarded as causative for AION in this patient, their combined hemodynamic effect may have overwhelmed the autoregulatory capacity of the optic nerve vasculature, resulting in ischemia.

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