The incubation period of West Nile virus ranges from 3 to 14 days. Two serosurveys have shown that approximately 1 in 150 infections resulted in meningitis or encephalitis, but most human infections remain subclinical. The reported symptoms and signs associated with West Nile virus infection include fever, malaise, anorexia, nausea, vomiting, headache, myalgia, rash, and lymphadenopathy. The frequencies of various symptoms and signs are poorly defined. Many patients with West Nile virus infection complain of severe muscle weakness. Acute flaccid paralysis similar to that associated with Guillian-Barré and poliomylitis-like syndromes has been reported. Although our patient had a long history of mitochondrial myopathy, an acute change in muscle pain and fatigue had occurred. This would not be typical for a mitochondrial cytopathy, which is characterized by slowly progressive myogenic weakness.

Nash and colleagues described the clinical characteristics of 59 patients hospitalized with West Nile virus infection in the New York City area in 1999. Fourteen percent of patients had symptoms of photophobia, and 3% of patients had conjunctivitis. Marberg et al. described the clinical characteristics of various symptoms and signs associated with West Nile virus infection including fever, malaise, anorexia, nausea, vomiting, headache, myalgia, rash, and lymphadenopathy. The frequencies of various symptoms and signs are poorly defined. Many patients with West Nile virus infection complain of severe muscle weakness. Acute flaccid paralysis similar to that associated with Guillian-Barré and poliomylitis-like syndromes has been reported. Although our patient had a long history of mitochondrial myopathy, an acute change in muscle pain and fatigue had occurred. This would not be typical for a mitochondrial cytopathy, which is characterized by slowly progressive myogenic weakness.

Nash and colleagues described the clinical characteristics of 59 patients hospitalized with West Nile virus infection in the New York City area in 1999. Fourteen percent of patients had symptoms of photophobia, and 3% of patients had conjunctivitis. Marberg et al. described 70 patients with West Nile fever. Forty-five percent reported ocular pain, and 60% had conjunctival hyperemia. Neither study commented on visual acuity or ophthalmologic examination findings. Although uveitis may have been the cause of the ocular findings, it was not specifically identified as such. Meningitis alone can cause photophobia and ocular pain. An Israeli patient developed signs and symptoms of meningitis, blurred vision, photophobia, and ocular pain. Ophthalmologic examination revealed visual dysfunction, bilateral optic nerve edema, and hemorrhages, but no uveitis. It is conceivable that the visual field defect in our patient was representative of a subclinical optic neuropathy secondary to the virus infection.

We postulate that some of the patients' photophobia, conjunctival hyperemia, and ocular pain may have had uveitis, but they were not examined by an ophthalmologist or with appropriate magnification. The patient described herein had anterior and posterior uveitis. Although acute hyperglycemia may cause uveitis, our patient had normal blood glucose levels throughout the observation period. Theoretically, our patient's uveitis could have occurred via human T-lymphotropic virus type 1 infection in the setting of Graves disease. This is unlikely to occur at the same time as her acute West Nile virus infection; furthermore, she does not live in an endemic region for human T-lymphotropic virus type 1 or have any risk factors for the infection. Although idiopathic bilateral uveitis can occur, the temporal relationship to the acute West Nile virus infection in our patient suggests a relationship to the viral infection. In addition, some flaviviruses can cause uveitis. Advanced age is the most significant risk factor for the development of severe neurologic disease, long-term morbidity, and death associated with these viruses. Diabetes mellitus is also associated with death in this infectious setting. A similar finding was noted during the 1996 Romanian outbreak of West Nile encephalitis. Our patient's medical conditions, including the presence of diabetes mellitus, may have predisposed the development of symptomatic West Nile virus infection, including uveitis.

Based on a MEDLINE search, we believe this represents the first report of uveitis associated with confirmed West Nile virus infection. Patients with a confirmed infection and ocular symptoms may warrant an ophthalmologic opinion. Patients with uveitis in endemic areas and with systemic symptoms may deserve West Nile virus testing.

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Exudative Complications After Photodynamic Therapy

Subfoveal choroidal neovascularization (CNV) caused by age-related macular degeneration is the leading cause of irreversible vision loss in Americans 65 years or older. Photodynamic therapy (PDT) with verteporfin (Visudyne; CIBA Vision Corp, Duluth, Ga) has been shown to retard vision loss compared with placebo in eyes with predominantly classic and purely occult CNV lesions. Verteporfin generates reactive oxygen species when illuminated with light at a wavelength of 689 nm, which results in occlusion of choroidal new.
vessels. Visual disturbances, including decreased visual acuity, visual field defect, or otherwise abnormal vision, are reported by 22% to 42% of patients after PDT. Severe vision loss, defined as the loss of at least 20 letters of visual acuity, has been reported in 4.4% (10/225) of patients with purely occult CNV. Eighty percent (8/10) of patients who suffered severe vision loss had occult lesions. Visual acuity improved to varying degrees in these patients. We report 2 cases of a marked exudation associated with vision loss within days after PDT.

Report of Cases. Case 1. An 83-year-old patient, diagnosed as having age-related macular degeneration associated with occult subfoveal CNV in the right eye (Figure 1), underwent PDT according to a standard protocol with the use of a laser (Coherent Opal Photoactivator; Coherent, Inc, Santa Clara, Calif). Pretreatment best-corrected visual acuity was 20/60 in the right eye. The patient reported decreased visual acuity immediately after PDT. When the patient was examined 5 days after treatment, the best-corrected visual acuity was 20/400. Fundus examination demonstrated a marked increase in submacular fluid. Fluorescein angiography demonstrated early hypofluorescence of the treated area with late patchy hyperfluorescence corresponding to the entire area of treatment (Figure 2). The retinal circulation appeared normal throughout the study. Indocyanine green angiography demonstrated diffuse hyperfluorescence in the late phase throughout the treated area (Figure 3). A course of observation was undertaken. Six weeks after treatment, the best-corrected visual acuity was 20/50 with almost complete resorption of the subretinal fluid. Three months after treatment, the best-corrected visual acuity was 20/50 and fundus examination showed a dry subfoveal retinal pigment epithelial (RPE) scar. Fluorescein angiography showed recovery of normal fluorescence and slightly less leakage from the neovascularization (Figure 4).

Case 2. A 68-year-old patient with age-related macular degeneration developed metamorphopsia and vision loss in the right eye. Pretreatment best-corrected visual acuity was 20/60 in the right eye. Examination showed central subretinal fluid, exudate, and hemorrhage. Fluorescein angiography demonstrated an occult subfoveal CNV in the right eye. The patient underwent PDT with verteporfin according to the standard protocol with the use of a laser (Coherent Opal Photoactivator). He noted severely decreased vision 2 days after PDT. On examination 3 days after treatment, the best-corrected visual acuity was 4/200. There was a marked increase in subretinal fluid with no change in subretinal hemorrhage. Angiography showed partial hypofluorescence of the CNV lesion with late patchy hyperfluorescence of the entire treated area. The retinal circulation was intact, with a notable absence of retinal vascular stain-
ing. One week after treatment, there was an improvement in the subreti-
nal fluid and hemorrhage. Improvement continued for 2 months after
 treatment, with only scant subreti-
nal hemorrhage and fluid remain-
ing and a best-corrected visual acu-
yty of 20/70.

Comment. The sudden accumula-
tion of fluid in the subneurosen-
sory retinal space after PDT may
arise from several sources. Retinal
vascular injury or subsequent in-
flammation could cause dysfunc-
tion of retinal vascular endothelial
tight junctions, resulting in retinal
edema and accumulation of subreti-
nal fluid. In our cases, however, an-
giography demonstrated no occlu-
sion or staining of retinal vessels. A
second possible cause is increased
leakage from the CNV. Angiogra-
phy in this series demonstrated less
hyperfluorescence from the CNV
and a diffuse area of hyperfluores-
cence corresponding to the entire
treated area.

Animal models have demon-
strated damage to the RPE and cho-
riocapillaris after PDT with vertepor-
fin.4 We postulate that sudden fluid
accumulation after PDT occurs as a
result of dysfunction of the RPE
pump and/or increased permeabil-
ity of the choroid. Studies have dem-
onstrated that RPE cells possess low-
density lipoprotein receptors that are
targeted by the liposomal prepara-
tions of verteporfin.4,5 Reactive oxy-
gen species can be generated by verte-
porfin within RPE cells, resulting in
cell damage and impaired fluid trans-
port. Damage of the RPE in pri-
mates was described after PDT with
verteporfin.4 It is interesting to note
that both of these cases were occult
lesions. Neovascularization under the
RPE could predispose the RPE cells
to unusual damage by causing up-
regulation in low-density lipopro-
tein receptors or simply by making
the cells less resistant to oxidative
stress. Another possible mechanism
may be that with predominantly clas-
ic CNV, the lesion itself may cast a
shadow on the RPE and thereby
lower the irradiance of the RPE com-
pared with occult lesions.

Both cases in our series were
observed. The subretinal fluid re-
solved slowly during 2 to 3 months,
with return of vision to near pre-
treatment levels. Because neither eye
developed a geographic area of at-
rophy, a temporary RPE dysfunc-
tion that resolved over time with-
out significant RPE cell death may
have been involved.

Photodynamic therapy has be-
come a mainstay in treatment of the
subfoveal form of age-related macu-
lar degeneration. Although PDT has
very few vision-threatening compli-
cations, the clinician should be
aware of the possibility of sudden vi-
sion loss caused by a marked exu-
dative response, particularly when
dealing with occult lesions.

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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.

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

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% fluoromethalone 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 immuno-fluorescent 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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