Optic Neuropathy in Patients Using Amiodarone
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Objective: To refine the criteria for the diagnosis of amiodarone-related optic neuropathy by including a broader spectrum of clinical features, thus helping to differentiate this entity from nonarteritic anterior ischemic optic neuropathy coincidentally affecting a patient taking amiodarone.

Methods: A retrospective case review of 22 patients who developed optic neuropathy while taking amiodarone, in whom other systemic causes were excluded.

Results: We identified 3 groups of patients: those in whom a diagnosis of amiodarone-induced optic neuropathy seems probable (n=14), those in whom an association with amiodarone optic neuropathy is indeterminate (n=5), and those in whom the occurrence of nonarteritic anterior ischemic optic neuropathy seems to be coincidental (ie, unrelated to amiodarone) (n=3). We formulated specific diagnostic criteria for each of these categories.

Conclusions: We recommend a systematic approach that includes assessment of bilaterality, mode of onset, degree of optic nerve dysfunction, structure of the involved optic disc in unilateral cases, and systemic toxic effects. Such well-defined diagnostic criteria can help the clinician in the treatment of patients with this disorder.

Arch Ophthalmol. 2006;124:696-701

Amiodarone optic neuropathy was first reported in 1987.1,2 Additional detailed descriptions of this entity have appeared in several case reports and small case series.3-11 The clinical picture that has emerged is quite variable, including cases with both unilateral and bilateral optic nerve involvement. While most reported cases have been associated with mild optic nerve dysfunction, which may be reversible, others have experienced significant permanent visual loss. The diagnosis of amiodarone optic neuropathy is a clinical one and, therefore, this variability in presentation has sometimes caused diagnostic confusion. Because patients taking amiodarone often have risk factors for vascular disease, differentiating between amiodarone optic neuropathy and anterior ischemic optic neuropathy (AION) is particularly challenging. In some cases, the clinical features of the 2 disorders are sufficiently different to allow confident diagnosis, but others are more ambiguous. Determining the correct diagnosis is all the more critical when treatment of the patient is strongly influenced by the decision. We sought to further refine the clinical criteria for the diagnosis of amiodarone optic neuropathy.

We performed a retrospective medical record review of patients who developed optic neuropathy while taking amiodarone. Patients were drawn from the neuro-ophthalmology service of 3 institutions, and all were examined by one of us. Exclusion criteria included evidence of an alternative systemic disorder that could explain the optic neuropathy and insufficient clinical information. Because this was a retrospective study, informed consent was considered unnecessary.

The following information was recorded for each patient: age, sex, amiodarone dose, interval between initiation of treatment and onset of visual symptoms, examination findings (initial and final), treatment, outcome, and follow-up interval. The onset of symptoms was considered sudden if the patient could describe a particular day that visual loss began, and designated as insidious if either the onset was more gradual or the patient was unaware of the deficit. We recorded information regarding systemic symptoms consistent with amiodarone toxicity, specifically tremor, ataxia, nausea, and confusion. The onset of such symptoms shortly after starting the medication, resolution after discontinuing treatment, and exclusion of other causes were considered supportive of the designation of these symptoms as drug toxicity. We also recorded the results of laboratory and radiographic tests for all patients. Follow-up information was obtained by di-
We divided patients initially into 2 groups: those with bilateral and those with unilateral optic nerve involvement. All patients in the bilateral group had simultaneous bilateral optic disc edema. The group with unilateral optic nerve involvement was further separated into those with clinical features typical of nonarteritic AION (NAION) vs those whose features were atypical. Typical features of NAION included immediate onset of painless visual loss, significant optic nerve dysfunction, and a crowded fellow disc. Features considered atypical for NAION included insidious onset, relative preservation of optic nerve function, and a generous cup-disc ratio in the fellow eye. Optic nerve function was assessed based on visual acuity, visual field measured by Goldmann perimetry, and clinical quantification of the relative afferent pupillary defect (RAPD) with visual field measured by Goldmann perimetry, and clinical quantification of the relative afferent pupillary defect (RAPD).

Rect examination, telephone contact, and/or review of records from other physicians.

Table 1. Clinical Summary of Group 1 Patients

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>Amiodarone Dosage, mg/d</th>
<th>Interval, mo</th>
<th>Systemic Symptoms</th>
<th>Onset of Visual Symptoms</th>
<th>VA at Presentation*</th>
<th>Visual Field*</th>
<th>VA at Follow-up*</th>
<th>Follow-up Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/M/74</td>
<td>300</td>
<td>12</td>
<td>Ataxia</td>
<td>Insidious</td>
<td>20/20 Superior altitudinal</td>
<td>20/20</td>
<td>20/20</td>
<td>7 y</td>
</tr>
<tr>
<td>2/M/70</td>
<td>400</td>
<td>3</td>
<td>No</td>
<td>Superior acupuncture</td>
<td>20/20</td>
<td>20/25</td>
<td>20/25</td>
<td>7 mo</td>
</tr>
<tr>
<td>3/M/69</td>
<td>400</td>
<td>5</td>
<td>No</td>
<td>Superior acupuncture</td>
<td>20/20</td>
<td>20/20</td>
<td>20/20</td>
<td>2 y</td>
</tr>
<tr>
<td>4/M/61</td>
<td>200</td>
<td>22</td>
<td>Tremor</td>
<td>Superior acupuncture</td>
<td>20/20</td>
<td>20/20</td>
<td>20/20</td>
<td>6 y</td>
</tr>
<tr>
<td>5/F/70</td>
<td>400</td>
<td>3</td>
<td>No</td>
<td>Superior acupuncture</td>
<td>CF</td>
<td>20/20</td>
<td>20/20</td>
<td>4 y</td>
</tr>
<tr>
<td>6/M/63</td>
<td>300</td>
<td>12</td>
<td>No</td>
<td>Superior acupuncture</td>
<td>20/20</td>
<td>20/20</td>
<td>20/20</td>
<td>7 y</td>
</tr>
<tr>
<td>7/F/74</td>
<td>200</td>
<td>2½</td>
<td>Ataxia</td>
<td>Superior acupuncture</td>
<td>20/20</td>
<td>20/20</td>
<td>20/20</td>
<td>3 y</td>
</tr>
<tr>
<td>8/M/68</td>
<td>200</td>
<td>2</td>
<td>No</td>
<td>Superior acupuncture</td>
<td>20/20</td>
<td>20/20</td>
<td>20/20</td>
<td>6 mo</td>
</tr>
<tr>
<td>9/M/78</td>
<td>400</td>
<td>2½</td>
<td>Tremor, ataxia, and confusion</td>
<td>Superior acupuncture</td>
<td>20/20</td>
<td>20/20</td>
<td>20/20</td>
<td>3 mo</td>
</tr>
<tr>
<td>10/M/70</td>
<td>NA</td>
<td>2</td>
<td>No</td>
<td>Superior acupuncture</td>
<td>CF</td>
<td>20/20</td>
<td>20/20</td>
<td>4 mo</td>
</tr>
<tr>
<td>11/M/77</td>
<td>100</td>
<td>2</td>
<td>No</td>
<td>Superior acupuncture</td>
<td>20/20</td>
<td>20/20</td>
<td>20/20</td>
<td>4 y</td>
</tr>
<tr>
<td>12/F/83</td>
<td>200</td>
<td>NA</td>
<td>No</td>
<td>Superior acupuncture</td>
<td>20/20</td>
<td>20/20</td>
<td>20/20</td>
<td>4 y</td>
</tr>
<tr>
<td>13/M/71</td>
<td>200</td>
<td>1</td>
<td>No</td>
<td>Superior acupuncture</td>
<td>20/20</td>
<td>20/20</td>
<td>20/20</td>
<td>4 y</td>
</tr>
<tr>
<td>14/M/62</td>
<td>200</td>
<td>4</td>
<td>Headache and nausea</td>
<td>Superior acupuncture</td>
<td>20/15</td>
<td>20/15</td>
<td>20/15</td>
<td>5 y</td>
</tr>
</tbody>
</table>

Abbreviations: CF, counting fingers; NA, data not available; VA, visual acuity.

*Data are given for the right eye and then the left eye.

RESULTS

Twenty-two patients (18 men and 4 women) were studied. Ages ranged from 58 to 83 years (mean, 70 years). The maintenance dosage of amiodarone ranged from 100 to 600 mg/d. The interval between initiation of amiodarone and onset of visual symptoms ranged from 1 to 22 months (mean, 6 months). Ancillary testing included computed tomography and/or magnetic resonance imaging in 17 patients, lumbar puncture in 5, Westergren erythrocyte sedimentation rate in 21, and temporal artery biopsy in 3. One patient with chronic renal failure had an erythrocyte sedimentation rate of 82 mm/h, prompting a temporal artery biopsy that showed no inflammation. He had no symptoms of giant cell arteritis at presentation or during the next 2 years. Other than an asymptomatic cavernous sinus meningioma in one patient and a mildly elevated cerebrospinal fluid protein level in another, all other test results were normal. Some follow-up information was available for all 22 patients. The follow-up interval ranged from 2 months to 7 years (mean, 2.5 years; median, 3 years).

Group 1 (those with simultaneous bilateral disc edema) consisted of 14 patients whose characteristics are summarized in Table 1. The onset of visual symptoms was insidious in 6 patients and immediate in 8. The visual acuity was worse than 20/200 in 2 patients. Optic nerve function was normal in 4 patients (5 eyes). The duration of disc edema (from initial discovery to documented resolution) was longer than 2 months in 11 patients and 2 months or less in 3 patients.

Groups 2 and 3 (those with unilateral disc edema) consisted of 5 and 3 patients, respectively. The clinical features of these patients are summarized in Table 2 and
Amiodarone was discontinued, and the patient began to take digoxin. One month later, optic disc edema was less pronounced in both eyes and there was some improvement of left optic nerve function. Reexamination 6 weeks later revealed a visual acuity of 20/25 OD and 20/100 OS, with mild dyschromatopsia in the left eye and a +1 RAPD. Goldmann perimetry in the right eye showed a normal field. In the left eye, there was a centrocecal scotoma and mild inferonasal depression. A fundus examination revealed bilateral optic disc edema, most prominent inferiorly.

The result of a computed tomographic scan of the head and orbits, with and without contrast, was normal, and the Westergren erythrocyte sedimentation rate was 19 mm/h. Amiodarone was discontinued, and the patient began to take digoxin. One month later, optic disc edema was less pronounced in both eyes and there was some improvement of left optic nerve function. Reexamination 6 weeks after discontinuation of amiodarone showed a visual acuity of 20/25 OD and 20/30 OS, with a +1 RAPD and an RAPD of 0.9 log units or less in 4.

A 69-year-old man began to receive amiodarone in April 1989 for newly diagnosed atrial fibrillation. Five months later, he developed a gray spot temporal to fixation in the left eye, unassociated with eye pain. A neuroophthalmic examination 10 days later revealed a visual acuity of 20/25 OD and 20/100 + 2 OS, with mild dyschromatopsia in the left eye and a +1 RAPD. Goldmann perimetry in the right eye showed a normal field. In the left eye, there was a centrocecal scotoma and mild inferonasal depression. A fundus examination revealed bilateral optic disc edema, most prominent inferiorly. The result of a computed tomographic scan of the head and orbits, with and without contrast, was normal, and the Westergren erythrocyte sedimentation rate was 19 mm/h. Amiodarone was discontinued, and the patient began to take digoxin. One month later, optic disc edema was less pronounced in both eyes and there was some improvement of left optic nerve function. Reexamination 6 weeks after discontinuation of amiodarone showed a visual acuity of 20/25 OD and 20/30 OS, with a +1 RAPD and an RAPD of 0.9 log units or less in 4.
later showed some further improvement, and at a fol-
low-up visit 6 months later, the optic discs were flat. The
patient’s visual acuity improved to 20/60 OS, and there was
only a small scotoma inferotemporal to fixation, with mild
inferior depression. His vision remained stable and car-
diac arrhythmia well controlled with digoxin 2 years later.

Although this patient reported unilateral visual loss,
examination in fact revealed swelling of both optic discs.
Despite moderate disc edema, optic nerve function in the
asymptomatic fellow eye was normal. There was no clini-
cal evidence of increased intracranial pressure or giant
cell arteritis. The combination of bilateral optic nerve in-
volvement and disc edema with preserved optic nerve
function is consistent with a diagnosis of amiodarone op-
tic neuropathy.

GROUP 2 (PATIENT 16)

A 65-year-old chemist began to receive amiodarone in
February 1998. In September, he began to experience gen-
eralized weakness, tremor, and ataxia. Two weeks later,
he developed halos around lights. His visual acuity was
20/20 OU. He missed 1 color plate in the right eye and
identified all 15 in the left eye. There was a small (0.6–
log unit) right RAPD. Goldmann perimetry in the right
eye showed a mild inferior altitudinal defect; the field in
the left eye was full. There was marked diffuse right op-
tic disc edema; the left disc was flat, with a 0.3 cup-disc
ratio. Corneal verticillate were seen bilaterally. The re-
result of a computed tomographic scan of the head was nor-
mal, the erythrocyte sedimentation rate was 10 mm/h,
and the complete blood cell count was normal.

Amiodarone was discontinued, leading to progressive
resolution of tremor and ataxia. A follow-up exami-
nation 1 month later showed much reduced optic disc
edema, normal color vision, and only a 0.3–log unit RAPD
in the right eye. There was only minimal depression of
the inferior visual field.

Diagnosis in this patient was more problematic be-
cause only 1 eye was affected, raising the possibility of
NAION. Features considered atypical for NAION in this
patient included insidious onset (actually asymptom-
atic from his optic neuropathy), relatively mild visual loss,
and a noncrowded disc in the fellow eye. In addition, this
patient experienced symptoms of systemic amiodarone
toxicity. The degree of visual recovery that he eventu-
ally enjoyed was also unusual for NAION.

GROUP 3 (PATIENT 21)

A 62-year-old physician experienced sudden painless loss
of vision inferiorly in the left eye 1 year after starting amio-
darone therapy. He had a history of hypertension and hy-
percholesterolemia. A neuro-ophthalmic examination 5
days after onset revealed a visual acuity of 20/20 OD and
20/25 OS, with a left RAPD. There was moderate infe-
rior field loss and segmental disc edema superiorly in the
left eye. The right disc was crowded, with no physi-
ologic cup. For the next 2 weeks, the inferior field de-
fect became more pronounced and the lower half of the

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<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Amiodarone Therapy</th>
<th>Time to Resolution of Disc Edema</th>
<th>Follow-up Interval</th>
<th>Cardiac Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Continued</td>
<td>3 mo</td>
<td>7 y</td>
<td>Recommended d/c amiodarone, but not done; pacemaker placed 5 y later</td>
</tr>
<tr>
<td>2</td>
<td>Reduced for 5 mo, then d/c</td>
<td>&gt;6 mo</td>
<td>1 y</td>
<td>No new medication started; died of MI 1 y later</td>
</tr>
<tr>
<td>3</td>
<td>d/c</td>
<td>6 mo</td>
<td>2 y</td>
<td>Doing well taking digoxin</td>
</tr>
<tr>
<td>4</td>
<td>d/c</td>
<td>&gt;2 mo</td>
<td>5 y</td>
<td>Doing well with a pacemaker and taking warfarin sodium (Coumadin)</td>
</tr>
<tr>
<td>5</td>
<td>d/c</td>
<td>3 mo</td>
<td>5 y</td>
<td>Doing well taking warfarin</td>
</tr>
<tr>
<td>6</td>
<td>d/c</td>
<td>3 mo</td>
<td>8 y</td>
<td>Sotalol therapy failed; amiodarone therapy restarted 1 y later at a reduced dose (200 mg), did well for 7 y</td>
</tr>
<tr>
<td>7</td>
<td>d/c</td>
<td>3 mo</td>
<td>3 y</td>
<td>Doing well taking warfarin and digoxin</td>
</tr>
<tr>
<td>8</td>
<td>d/c</td>
<td>2 mo</td>
<td>2 y</td>
<td>Doing well taking sotalol plus receiving ablation therapy</td>
</tr>
<tr>
<td>9</td>
<td>d/c</td>
<td>3 mo</td>
<td>6 y</td>
<td>Doing well taking warfarin and with a pacemaker placed, then died 6 y later</td>
</tr>
<tr>
<td>10</td>
<td>d/c</td>
<td>4 mo</td>
<td>4 mo</td>
<td>NA</td>
</tr>
<tr>
<td>11</td>
<td>d/c</td>
<td>2 mo</td>
<td>4 y</td>
<td>Doing well taking digoxin and warfarin</td>
</tr>
<tr>
<td>12</td>
<td>d/c</td>
<td>&gt;2 mo</td>
<td>4 y</td>
<td>Doing well taking aspirin</td>
</tr>
<tr>
<td>13</td>
<td>d/c</td>
<td>2 mo</td>
<td>4 y</td>
<td>Doing well taking warfarin</td>
</tr>
<tr>
<td>14</td>
<td>d/c</td>
<td>6 mo</td>
<td>4 y</td>
<td>NA</td>
</tr>
<tr>
<td>Group 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>d/c</td>
<td>2 mo</td>
<td>3 y</td>
<td>Died 3 y later</td>
</tr>
<tr>
<td>16</td>
<td>d/c</td>
<td>2 mo</td>
<td>5 y</td>
<td>Doing okay without treatment; atrial fibrillation used only with exertion</td>
</tr>
<tr>
<td>17</td>
<td>d/c</td>
<td>NA</td>
<td>4 mo</td>
<td>Digoxin therapy started; died of a stroke 2½ mo later</td>
</tr>
<tr>
<td>18</td>
<td>Reduced</td>
<td>1 mo</td>
<td>4 mo</td>
<td>Doing well taking amiodarone (200 mg)</td>
</tr>
<tr>
<td>19</td>
<td>d/c 6 mo later</td>
<td>NA</td>
<td>2 y</td>
<td>Doing well taking digoxin</td>
</tr>
<tr>
<td>Group 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>d/c</td>
<td>6 wk</td>
<td>7 y</td>
<td>Doing well taking propafenone, plus using a pacemaker</td>
</tr>
<tr>
<td>21</td>
<td>Continued</td>
<td>6 wk</td>
<td>10 mo</td>
<td>Doing well taking amiodarone</td>
</tr>
<tr>
<td>22</td>
<td>Continued</td>
<td>NA</td>
<td>2 y</td>
<td>Died of meningitis 2 y later</td>
</tr>
</tbody>
</table>

Abbreviations: d/c, discontinued; MI, myocardial infarction; NA, data not available.
disc also became swollen. By 6 weeks after onset, disc edema had resolved, leaving the top half of the disc pale. His inferior altitudinal field remained stable 1 year later.

This patient experienced acute unilateral visual loss with segmental disc edema and moderate loss of optic nerve function. He had typical vascular risk factors for NAION, the characteristic cupless appearance of the fellow optic disc, and no symptoms of systemic amiodarone toxicity. These clinical features are all most consistent with a diagnosis of NAION that by coincidence occurred while taking amiodarone. The relatively rapid subsequent resolution of disc edema constitutes additional supportive evidence.

**COMMENT**

The mechanism of amiodarone neuropathy is not completely understood. A histopathologic study of the optic nerve in a patient without visual symptoms who was treated with amiodarone demonstrated multiple lamellar inclusion bodies within large axons, unaccompanied by axonal loss. Experimental studies of mice treated with amiodarone have shown similar inclusions in glial cells as well. Intraneuronal accumulation of this material may lead directly to axonal swelling or, alternatively, deposition in glial cells with subsequent swelling of these cells may secondarily obstruct axonal transport. Either process could produce the optic disc edema observed in amiodarone optic neuropathy. In some cases, these changes lead to impairment of visual function, and in these cases, the resulting clinical picture may lead to confusion between amiodarone optic neuropathy and NAION.

This challenge was addressed by Macaluso et al, who reviewed 16 previously reported cases plus 57 others reported to the National Registry of Drug-Induced Ocular Side Effects, the US Federal Drug Administration, and the World Health Organization. Based on these data, the researchers proposed criteria for distinguishing amiodarone optic neuropathy from NAION as follows. Amiodarone optic neuropathy is characterized by insidious onset of visual loss, protracted disc edema (for months), and bilateral (usually simultaneous) involvement. In contrast, NAION is characterized by acute unilateral visual loss with resolution of disc edema over several weeks. While these features are useful for diagnosis in some cases, we have found that several patients share features of each category.

In applying the criteria proposed by Macaluso et al to our 22 patients, we found that only 7 fit all of the criteria for classification (4 as amiodarone optic neuropathy and 3 as NAION). In other words, 15 of our patients (two thirds) would remain unclassified by the recommendations. We, therefore, sought to further refine the criteria to help with ambiguous cases whose clinical features overlap the 2 diagnostic categories. Ultimately, this classification should aid in treatment decisions for these patients.

We propose the following system for classifying patients with optic neuropathy while taking amiodarone. Patients are divided initially into those with simultaneous bilateral and those with unilateral disc edema. In the group with bilateral disc edema, it is imperative to obtain appropriate ancillary test results to exclude other potential causes, including increased intracranial pressure and giant cell arteritis. Those with unilateral disc edema are then further subdivided into those with clinical features typical of NAION and those with atypical features. We identified atypical features as the following: insidious onset of symptoms, relatively mild optic nerve dysfunction, and a generous cup-disc ratio in the fellow eye. The presence of a crowded fellow disc is so characteristic of NAION that its absence should cast some doubt on the diagnosis. Because of the broad range of visual acuities in ischemic damage, we used qualitative assessment of the visual field and quantitative measure of pupil function (the RAPD) as our measurements of optic nerve function. Furthermore, we considered the presence of systemic symptoms consistent with amiodarone toxicity to increase the suspicion that the patient’s optic neuropathy was related to amiodarone. For patients who continue to pose a diagnostic challenge some weeks after onset, prolonged duration of disc edema may also be considered an atypical feature. We used this system to classify our 22 patients into 3 groups (Tables 1-3).

Group 1 consisted of 14 patients with simultaneous bilateral disc edema. Six experienced insidious and 8 had immediate onset of visual symptoms. Initial visual acuities ranged from 20/15 to counting fingers. Eleven patients had protracted disc edema, 3 had more prompt resolution. Despite this variability in clinical features, we believe this group most likely represents optic neuropathy secondary to amiodarone. The spectrum of amiodarone optic neuropathy may be broader than previously defined, particularly in regard to the degree of optic nerve dysfunction. The severity of visual loss in this condition may be influenced by underlying risk factors for secondary disc ischemia and the direct effects of amiodarone toxicity. The duration of disc edema has limited value in patient treatment because this information only becomes available later in the course of the disease.

Our 3 group 3 patients had immediate onset of disc edema. In addition, each had a crowded contralateral disc, moderate to substantial deficits of optic nerve function, and no systemic symptoms of amiodarone toxicity. We believe this group most likely represents NAION; in other words, the optic neuropathy in these patients is coincidental, unrelated to amiodarone.

In rigorously defining the criteria for the diagnosis of NAION, we identified 5 patients (group 2) with unilateral disc edema who had 1 or more features considered atypical for NAION. Three of these patients experienced insidious onset of their visual symptoms. Optic nerve function was relatively preserved in all. The fellow disc was noncrowded in 4 fellow eyes and could not be assessed because of preexisting optic atrophy in the remaining patient. In addition, 2 patients reported systemic symptoms of amiodarone toxicity. We believe the mechanism of optic neuropathy in these patients is best considered indeterminate. Their clinical features are atypical for either amiodarone toxicity or NAION and, thus, for a condition that is defined by clinical features they cannot be firmly classified.

Based on the previously described classification, we would recommend that discontinuation of amiodarone be strongly considered for patients in group 1 (ie, those with probable amiodarone optic neuropathy) and considered...
as well for patients in group 2. In our series, of 16 patients in whom amiodarone was discontinued and for whom adequate follow-up information was available, 1 experienced a fatal stroke 2½ months after discontinuing treatment and 1 died of a myocardial infarction 1 year later. Thirteen patients continued to do well with alternative treatments (medical or surgical) for their cardiac arrhythmia, from 2 to 7 years later. For group 3 patients (ie, those classified as having NAION), continuation of amiodarone therapy may be appropriate. Decisions regarding treatment for all 3 patient groups must take into consideration the potential risks and benefits of treatment with amiodarone, a process that will involve a collaboration between the treating cardiologist and ophthalmologist and must be individualized in each case.

Submitted for Publication: March 9, 2005; final revision received June 10, 2005; accepted June 21, 2005.

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Author Contributions: The authors had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

Financial Disclosure: None.

REFERENCES