Optic Neuritis in African Americans

Paul H. Phillips, MD; Nancy J. Newman, MD; Michael J. Lynn, MS

Objective: To describe the clinical profile of demyelinating optic neuritis in African Americans.

Methods: The medical records of all patients with a diagnosis of optic neuritis examined at the Neuro-Ophthalmology Unit at the Emory University Eye Center (Emory) and at the Grady Memorial Hospital Eye Clinic (Grady), Atlanta, Ga, between 1989 and 1996 were retrospectively reviewed.

Patients: African American and white patients, aged 15 through 55 years, with a single initial episode of acute optic neuritis of unknown or demyelinative origin were included in the study. Study patients included 23 African American patients and 56 white patients examined at Emory as well as 10 African American patients examined at Grady.

Results: There were no significant differences among the African American study patients, the white study patients, and patients from the Optic Neuritis Treatment Trial (ONTT) regarding sex (P=0.36), age (P=0.73), or the presence of disc edema (P=0.40), lesions found on magnetic resonance imaging (P=0.43), or multiple sclerosis (P=0.54) at the onset of an initial episode of optic neuritis. The Emory African American patients presented with more frequent severe visual loss (13 [93%] of 14 patients with a visual acuity ≤20/200) compared with Emory white patients (12 [39%] of 31 patients; P=0.002) and with ONTT patients (161 [36%] of 448 patients; P<0.001). At follow-up examination of at least 1 year, Emory African American patients had worse vision (9 [39%] of 23 patients <20/40, and 4 [17%] of 23 patients ≤20/200) compared with Emory white patients (5 [8%] of 63 patients <20/40, P=0.001; 3 [5%] of 63 patients ≤20/200, P=0.08), and with ONTT patients (29 [7%] of 409 patients <20/40, P=0.001; 12 [3%] of 409 patients ≤20/200, P=0.01). Compared with ONTT patients, the Emory African American patients combined with the Grady African American patients had more frequent severe visual loss (visual acuity ≤20/200) at presentation (18 [90%] of 20 patients vs 161 [36%] of 448 patients; P<0.001) and at follow-up examination of at least 1 year (6 [18%] of 33 patients vs 12 [3%] of 409 patients; P=0.002). Seven (58%) of 12 African American patients with multiple sclerosis had a “neuromyelitis optica” presentation defined by the presence of neurological deficits limited to the optic nerves and spinal cord.

Conclusions: The African American study patients with a single episode of demyelinating optic neuritis had visual acuities more severely affected at onset and after 1 year of follow-up compared with the white study patients and with patients in the ONTT. In the African American patients, multiple sclerosis occurred most frequently in a “neuromyelitis optica” form.

Arch Neurol. 1998;55:186-192

Demyelinating optic neuritis most frequently occurs in whites.1 The clinical profile of optic neuritis in African Americans has received little attention. Racial effects on the clinical profile of optic neuritis and demyelinating disease have been suggested in other countries. Although demyelinating optic neuritis is rare in South African blacks, when it occurs in this group it is frequently associated with severe visual loss.2 Similar studies, demyelinating disease occurs infrequently in Japan. However, Japanese patients with multiple sclerosis (MS) frequently have severe involvement of the optic nerves at presentation and during the course of the disease.3

The clinical profile of demyelinating optic neuritis specifically in African Americans is unknown. In the Optic Neuritis Treatment Trial4 (ONTT) there were 68 nonwhite patients, and within this group, 59 patients were African American. R. Beck, MD, written communication, November 8, 1995). The visual acuities of the white and nonwhite patients were not significantly different at the 6-month follow-up examination.2 No other
PATIENTS AND METHODS

The medical records of all patients with a diagnosis of optic neuritis examined at the Neuro-Ophthalmology Unit at the Emory University Eye Center (Emory) and at the Grady Memorial Hospital Eye Clinic (Grady), Atlanta, Ga, between 1989 and 1996 were reviewed. Inclusion criteria for further analysis included the presence of an acute optic neuritis of unknown or demyelinating origin; age range of 15 through 55 years; and the presence of decreased visual acuity, a visual field deficit, or a relative afferent pupillary defect either alone or in combination. Patients were excluded from further analysis if they had any ocular or systemic disease that would cause visual loss other than MS, ocular findings suggestive of a nondemyelinating cause of optic neuritis, positive results of a syphilis serological test, or an angiotensin-converting enzyme level above 60 U/L.

Age at onset of symptoms, ocular symptoms, visual acuity with best refractive correction, color vision, optic nerve appearance, and treatment interventions at presentation and for all follow-up visits were recorded for each initial attack of optic neuritis within a given eye. Data were collected for both eyes in patients with bilateral nonsimultaneous optic neuritis but not for recurrent episodes of optic neuritis within the same eye.

Eighteen African American patients examined at Grady between 1989 and 1996 were diagnosed as having optic neuritis. Eight patients were discontinued from further analyses because of the concurrent presence of the acquired immunodeficiency syndrome (n=2), neurosyphilis (n=1), sarcoidosis (n=1), elevated intracranial pressure (n=1), an orbital mass (n=1), a pituitary mass (n=1), and an angiotensin-converting enzyme level of 93 U/L (n=1). The 10 remaining patients had 20 episodes of optic neuritis, including 14 initial episodes and 6 recurrent episodes of optic neuritis. Four of the 10 patients had bilateral nonsimultaneous episodes of optic neuritis.

Thirty-two African American patients examined at Emory between 1989 and 1996 were diagnosed as having optic neuritis. Nine patients were discontinued from further analyses because of the concurrent presence of severe uveitis (n=2), glaucoma (n=2), herpes zoster ophthalmicus (n=1), functional visual loss (n=1), sarcoidosis (n=1), recent sinus surgery (n=1), and an angiotensin-converting enzyme level of 84 U/L (n=1). The 23 remaining patients had 39 episodes of optic neuritis, including 30 initial episodes and 9 recurrent episodes of optic neuritis. Seven of the 23 patients had bilateral nonsimultaneous episodes of optic neuritis.

Eighty-two white patients examined at Emory between 1989 and 1996 were diagnosed as having optic neuritis. Twenty-six patients were discontinued from further analyses because of the concurrent presence of severe uveitis (n=8), ocular trauma (n=1), systemic lupus erythematosus (n=4), elevated intracranial pressure (n=1), sarcoidosis (n=1), mononucleosis (n=1), meningitis (n=1), and an age outside the acceptable range (n=9). The remaining 56 patients had 97 episodes of optic neuritis, including 76 initial episodes and 21 recurrent episodes of optic neuritis. Twenty of these 56 patients had bilateral nonsimultaneous episodes of optic neuritis.

Table 1 shows patient demographics and clinical characteristics of optic neuritis in this nonwhite group. We describe the clinical profile of demyelinating optic neuritis in a group of African American patients at an academic medical center that includes a tertiary referral center and an inner-city hospital.

RESULTS

Eighteen African American patients examined at Grady between 1989 and 1996 were diagnosed as having optic neuritis. Seven of the 23 patients had bilateral nonsimultaneous episodes of optic neuritis. The remaining 56 patients had 97 episodes of optic neuritis, including 76 initial episodes and 21 recurrent episodes of optic neuritis. Twenty of these 56 patients had bilateral nonsimultaneous episodes of optic neuritis.

Initial visual acuity was defined as the visual acuity first obtained within 2 weeks after onset of symptoms. The most recent visual acuity obtained at least 1 year after onset of a single initial episode of optic neuritis was considered the follow-up visual acuity. If the patient did not have a follow-up examination more than 1 year after onset of an episode, the most recent visual acuity was recorded as the 1-year follow-up visual acuity only if the patient had achieved a visual acuity of at least 20/40.

Laboratory results, magnetic resonance imaging (MRI) results, and MS status were recorded for each patient. Patients were considered to have MS if they had probable or definite MS according to the clinical criteria of Poser et al.8

The mean age at presentation was compared among the 3 study groups and the ONTT1 using a 1-way analysis of variance. All other data were analyzed as categorical variables. Proportions were compared among the groups using either a χ² test or Fisher exact test. An α level of .05 was used to assess statistical significance. For variables in which some data were missing, all available data were included in the analysis. Sex, laboratory test results, presence of lesions on MRI scans, and MS at initial presentation were analyzed on a per patient basis. All other variables were analyzed on a per eye basis.

Eighty-two white patients examined at Emory between 1989 and 1996 were diagnosed as having optic neuritis. Twenty-six patients were discontinued from further analyses because of the concurrent presence of severe uveitis (n=8), ocular trauma (n=1), systemic lupus erythematosus (n=4), elevated intracranial pressure (n=1), sarcoidosis (n=1), mononucleosis (n=1), meningitis (n=1), and an age outside the acceptable range (n=9). The remaining 56 patients had 97 episodes of optic neuritis, including 76 initial episodes and 21 recurrent episodes of optic neuritis. Twenty of these 56 patients had bilateral nonsimultaneous episodes of optic neuritis.
Figure 2 shows the distribution of visual acuities on examination at least 1 year after onset of a single initial episode of optic neuritis for our 3 study groups. Table 3 shows that the Emory African American patients failed to recover a visual acuity of at least 20/40 significantly more frequently compared with Emory white patients (39% vs 8%; \( P = .001 \)) and with ONTT\(^7 \) patients (39% vs 7%; \( P = .0001 \)). In addition, the Emory African American patients failed to recover a visual acuity greater than 20/200 more frequently compared with Emory white patients (17% vs 5%; \( P = .08 \)) and with ONTT\(^7 \) patients (17% vs 3%; \( P = .01 \)). Compared with ONTT\(^7 \) patients, the Emory African American patients combined with the Grady African American patients had significantly more frequent follow-up visual acuities of less than 20/40 (36% vs 7%; \( P < .0001 \)) and less than or equal to 20/200 (18% vs 3%; \( P = .002 \)).

One of the problems with retrospective studies is that patients who recover may fail to attend follow-up appointments, thus biasing the results toward a poorer outcome. Follow-up visual acuities were not available for 11 African American patients—7 patients in the Emory African American group and 4 patients in the Grady African American group. Four of these patients had a visual acuity of less than 20/40 but were not yet 1 year from the onset of their episode of optic neuritis. Three patients had a visual acuity of less than 20/40 but had recurrent episodes of optic neuritis before the 1-year follow-up visit for their initial episode of optic neuritis. Only 4 patients were actually unavailable for follow-up. If all the African American patients without 1-year follow-up visual acuities are assumed to have achieved a visual acuity of 20/20 and are incorporated into the analyses, the African American study patients still have significantly worse visual acuity at follow-up compared with the ONTT\(^7 \) patients (Table 4).

Including patients who developed MS during follow-up, 8 Emory African American patients and 4 Grady African American patients were diagnosed as having probable or definite MS. Seven (58%) of these 12 patients had a “neuromyelitis optica” (or “Devic’s-like”) presentation. For the purposes of this study, neuromyelitis optica was defined by the presence of signs and symptoms limited primarily to the optic nerves and spinal cord. Of the 13 Emory white patients who ultimately developed probable or definite MS, 3 (23%) had a Devic’s-like presentation. All the patients with a Devic’s-like presentation had a polyphasic illness with remissions and exacerbations. None had bilateral, simultaneous optic neuritis. The visual acuities and MRI results of the African American patients with a Devic’s-like presentation are shown in Table 5.

Results of laboratory tests of the study patients did not reveal a disease other than MS that would cause visual loss or other neurological deficits. Results of magnetic resonance imaging (obtained in 28 [85%] of 33 African American study patients and in 44 [79%] of 56 Emory white patients) were either normal or revealed lesions consistent with MS. Among patients with a final visual acuity of less than 20/40, MRI was performed in 9 (90%) of 10 African American study patients and in 4 (80%) of 5 Emory white patients.

By our selection criteria, results of syphilis serological tests were negative and the angiotensin-converting enzyme level was lower than 60 U/L when these tests were performed. Results of syphilis serological tests were obtained in 23 (70%) of 33 African American study patients and in 26 (46%) of 56 Emory white patients. Among patients with a final visual acuity of less than 20/40, results of syphilis serological tests were obtained in 7 (70%) of 10 African American study patients and in 1 (20%) of 5 Emory white patients. An angiotensin-converting en-

### Table 1. Patient Demographics and Clinical Characteristics*

<table>
<thead>
<tr>
<th>Patient Characteristics</th>
<th>Emory Blacks</th>
<th>Grady Blacks</th>
<th>Emory Whites</th>
<th>ONTT</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean±SD), y</td>
<td>32.7±7.3</td>
<td>31.2±7.9</td>
<td>32.6±9.1</td>
<td>31.8±6.7</td>
<td>.73</td>
</tr>
<tr>
<td>Sex, % F</td>
<td>87 (n=23)</td>
<td>90 (n=10)</td>
<td>71 (n=56)</td>
<td>77 (n=448)</td>
<td>.36</td>
</tr>
<tr>
<td>% Present</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>82 (n=22)</td>
<td>89 (n=9)</td>
<td>71 (n=51)</td>
<td>92 (n=448)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Disc edema</td>
<td>44 (n=16)</td>
<td>63 (n=8)</td>
<td>35 (n=37)</td>
<td>35 (n=448)</td>
<td>.40</td>
</tr>
<tr>
<td>MRI lesions†</td>
<td>53 (n=15)</td>
<td>67 (n=6)</td>
<td>38 (n=42)</td>
<td>49 (n=448)</td>
<td>.43</td>
</tr>
<tr>
<td>MS at presentation</td>
<td>22 (n=23)</td>
<td>20 (n=10)</td>
<td>17 (n=52)</td>
<td>13 (n=448)</td>
<td>.54</td>
</tr>
</tbody>
</table>

*Emory indicates the Neuro-Ophthalmology Unit at the Emory University Eye Center and Grady, Grady Memorial Hospital Eye Clinic in Atlanta, Ga; ONTT, data from the Optic Neuritis Treatment Trial\(^1 \); MRI, magnetic resonance imaging; MS, multiple sclerosis; and \( n \), number of patients for sex, MRI lesions, and MS at presentation and number of eyes for pain and disc edema.

†The MRI category of grades 2 to 4 in the Optic Neuritis Treatment Trial.\(^1 \)

#### Figure 1

*Figure 1. Distribution of visual acuities at presentation. Grady indicates the Grady Memorial Hospital Eye Clinic and Emory, the Neuro-Ophthalmology Unit at the Emory University Eye Center, Atlanta, Ga; CF, counting fingers; HM, hand motion; LP, light perception; and NLP, no light perception.*

<table>
<thead>
<tr>
<th>Range of Visual Acuity</th>
<th>% of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;20/200</td>
<td>0</td>
</tr>
<tr>
<td>20/200-20/400</td>
<td>10</td>
</tr>
<tr>
<td>20/50-20/100</td>
<td>90</td>
</tr>
<tr>
<td>20/200</td>
<td>0</td>
</tr>
<tr>
<td>20/100</td>
<td>10</td>
</tr>
<tr>
<td>20/800</td>
<td>0</td>
</tr>
<tr>
<td>CF</td>
<td>100</td>
</tr>
<tr>
<td>HM</td>
<td>0</td>
</tr>
<tr>
<td>LP</td>
<td>0</td>
</tr>
<tr>
<td>NLP</td>
<td>0</td>
</tr>
</tbody>
</table>

#### Table 3

| Table 2. Visual Acuities at Presentation and Number of Eyes for Pain and Disc Edema. |
|-----------------------------------|------------|------------|------------|----------|----------|
| Visual Acuity Range               | Grady Blacks | Emory Blacks | Emory Whites | ONTT     |
| >20/200                            | 0          | 0          | 0          | 0        |
| 20/200-20/400                      | 10         | 9          | 71         | 92       |
| 20/50-20/100                       | 90         | 89         | 71         | 92       |
| 20/200                             | 0          | 0          | 0          | 0        |
| 20/100                             | 10         | 10         | 71         | 92       |
| 20/800                             | 0          | 0          | 0          | 0        |
| CF                                 | 100        | 100        | 100        | 100      |
| HM                                 | 0          | 0          | 0          | 0        |
| LP                                 | 0          | 0          | 0          | 0        |
| NLP                                | 0          | 0          | 0          | 0        |

*Emory indicates the Neuro-Ophthalmology Unit at the Emory University Eye Center and Grady, Grady Memorial Hospital Eye Clinic in Atlanta, Ga; ONTT, data from the Optic Neuritis Treatment Trial\(^1 \); MRI, magnetic resonance imaging; MS, multiple sclerosis; and \( n \), number of patients for sex, MRI lesions, and MS at presentation and number of eyes for pain and disc edema.

†The MRI category of grades 2 to 4 in the Optic Neuritis Treatment Trial.\(^1 \)
zyme level was obtained in 19 (58%) of 33 African American study patients and in 8 (14%) of 56 Emory white patients. Among patients with a final visual acuity of less than 20/40, an angiotensin-converting enzyme level was obtained in 8 (80%) of 10 African American study patients and in 1 (20%) of 5 Emory white patients.

Chest radiographs always showed no abnormalities and were performed in 21 (64%) of 33 African American study patients and in 6 (11%) of 56 Emory white patients. Among patients with a final visual acuity of less than 20/40, chest radiographs were obtained in 6 (60%) of 10 African American study patients and in 1 (20%) of 5 Emory white patients.

Results of lumbar punctures were either normal or revealed abnormalities consistent with demyelinating disease, such as increased protein levels, elevated immunoglobulin levels, oligoclonal bands, or a lymphocytic cellular response. A lumbar puncture was obtained in 12 (36%) of 33 African American study patients and in 10 (18%) of 56 Emory white patients. Among patients with a final visual acuity of less than 20/40, a lumbar puncture was performed in 4 (40%) of 10 African American study patients and in none of the Emory white patients.

An antinuclear antibody (ANA) titer was obtained in 17 (52%) of 33 African American study patients. Eight of these patients had positive titers. The highest titer was 1:160. These 8 patients had no clinical evidence of a collagen-vascular disease and 7 of them had further rheumatological workup results that were unremarkable. Among patients with a final visual acuity of less than 20/40, an ANA titer was obtained in 5 (50%) of 10 patients. The ANA titer was normal in 4 of these patients and 1 patient had positive titers of 1:80. This patient had no clinical evidence of collagen-vascular disease and further results of rheumatological workup were unremarkable.

An ANA titer was obtained in 23 (41%) of 56 Emory white patients. Four of these patients had positive titers. The highest titer was 1:160. These 4 patients had no clinical evidence of a collagen-vascular disease and 1 of them had further rheumatological test results that were unremarkable. Among patients with a final visual acuity of less than 20/40, an ANA titer was obtained in 2 (40%) of 5 patients and was normal in both patients.

An ANA titer was obtained in 3 (43%) of 7 African American study patients with a Devic’s-like presentation. Two of these patients had positive titers measuring 1:40. Neither patient had clinical evidence of a collagen-vascular disease and results of further rheumatological workup were unremarkable for both patients. Among all the study patients, positive ANA titers did not significantly correlate with severe vision loss (visual acuity ≤20/200) either at presentation (P=.42) or at 1 year of follow-up (P=.36).

The study patients were observed either with no treatment or treated with oral or intravenous steroids. None of the study patients had a steroid-dependent optic neuropathy with a decrease in visual acuity as use of steroids was discontinued.

**Table 2. Visual Acuity at Presentation**

<table>
<thead>
<tr>
<th>Visual Acuity</th>
<th>Emory Blacks, % (n=14)</th>
<th>Emory Whites, % (n=31)</th>
<th>ONTT, % (n=448)</th>
<th>Emory Blacks and Grady Blacks, % (n=20)</th>
<th>ONTT, % (n=448)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;20/40</td>
<td>93</td>
<td>52 (P=.008)†</td>
<td>65 (P=.04)‡</td>
<td>90</td>
<td>65 (P=.04)§</td>
</tr>
<tr>
<td>≤20/200</td>
<td>93</td>
<td>39 (P=.002)‡</td>
<td>36 (P&lt;.001)†</td>
<td>90</td>
<td>36 (P&lt;.001)§</td>
</tr>
</tbody>
</table>

*Emory indicates the Neuro-Ophthalmology Unit at the Emory University Eye Center and Grady, the Grady Memorial Hospital Eye Clinic in Atlanta, Ga; ONTT, data from the Optic Neuritis Treatment Trial; and n, number of eyes.
†P value comparing Emory blacks with Emory whites.
‡P value comparing Emory blacks with ONTT patients.
§P value comparing Emory blacks and Grady blacks with ONTT patients.

**Figure 2. Distribution of visual acuities at least 1 year after presentation.** Grady indicates the Grady Memorial Hospital Eye Clinic and Emory, the Neuro-Ophthalmology Unit at the Emory University Eye Center, Atlanta, Ga; CF, counting fingers; HM, hand motion; LP, light perception; and NLP, no light perception.

**COMMENT**

The clinical profile of demyelinating optic neuritis in African Americans has received little attention. Racial effects on the clinical profile of demyelinating optic neuritis have been suggested in several groups from other countries. Although demyelinating optic neuritis is rarely described in South African blacks, optic neuritis in this group is often associated with severe visual loss. Dean et al described 12 black patients with MS from South Africa and Zimbabwe. Six of these patients became bilaterally “blind, or nearly so, from severe optic neuritis.” Ames and Louw reported 7 black South African patients with MS. All 7 patients had a history of optic neu-
ritis, with 6 affected bilaterally. Six of their patients had a visual acuity of less than 20/100 in at least 1 eye. Four patients had simultaneous bilateral optic neuritis and 4 patients had optic neuritis as the presenting manifestation of their disease.

A similar racial effect on the clinical course of demyelinating optic neuritis has been demonstrated in Japanese patients with MS, another group with a low prevalence of demyelinating disease. Kuroiwa et al analyzed 1084 Japanese patients with MS in a nationwide survey. These patients had more frequent visual impairment, especially bilaterally, at the onset of the illness, and more frequent and severe involvement of the optic nerves during the course of their illness compared with patients with MS in western countries.

The only source of published data on demyelinating optic neuritis in African Americans is the ONTT. The ONTT patients included 68 nonwhites of whom 59 were African American (R. Beck, written communication, November 8, 1995). The visual acuities of the nonwhite and white groups did not differ significantly at the 6-month follow-up examination.

More has been written on the effects of race on the clinical profile of MS. Most of this information reports the effect of race on the prevalence of the disease. Within a given location, the black population generally has a lower prevalence of MS compared with the white population. The extensive epidemiological study by Kurtzke et al and by Kurtzke on US veterans showed that, in America, African Americans and Asians have lower rates of MS than whites. Oh and Calhoun also showed that African Americans had decreased prevalence rates of MS compared with whites within the same city.

In South African blacks, MS frequently occurs in the form of neuromyelitis optica, accounting for up to 98% of cases. Ames and Louw described 4 black South African patients with MS with severe optic nerve and spinal cord involvement. Cosnett described 6 African blacks with neuromyelitis optica. A similar pattern occurs in Japanese patients. In the nationwide survey by Kuroiwa et al of MS in Japan, Japanese patients had frequent involvement of the optic nerves and spinal cord during the course of their illness, with neuromyelitis optica occurring in 7.6% of these patients.

Few studies have examined the clinical profile of MS in African Americans. Haeret performed a retrospective analysis of the natural history of MS in Mississippi blacks and whites. He reported that Mississippi black patients with MS had increased disability, increased mortality rates, more frequent chronic progressive disease, and fewer years from onset of disease to death compared with Mississippi white patients. Retrospective studies by Morariu and Linden and Alter both conclude that, overall, the clinical profile of MS in African Americans is not significantly different from that of whites. However, southern-born African Americans had more frequent chronic progressive disease and were older at presentation compared with northern-born African Americans.

Compared with ONTT patients and with white patients in our practice, the African American patients with optic neuritis in our study had significantly worse visual acuities at onset and after at least 1 year of follow-up from a single initial episode of optic neuritis. The
clinical profile of optic neuritis among all the groups was otherwise similar. This difference in our results from those of the ONTT may be the result of a variety of factors, including genetic or environmental influences and referral bias.

The African American patients in our study were derived from 2 medical centers within a single southern American city, whereas the African American patients in the ONTT were derived from multiple centers throughout the United States. The African American patients in our study may represent a subgroup of patients genetically at risk for severe optic neuritis. Several reports suggest a genetic influence on the prevalence and expression of MS. Kira et al showed immunogenetic differences between Japanese patients with MS with disseminated central nervous system involvement and those with selective involvement of the optic nerve and spinal cord.

Alternatively, environmental factors may account for the different results. Many of the ONTT patients were from the northern United States. As noted earlier, Morariu and Linden and Alter described differences in the clinical expression of MS in northern vs southern African Americans, with the latter having more severe, chronic progressive disease.

Referral bias may account for the worse visual acuities observed in our African American patients. The African American patients in our study were derived from 2 different sources. One group was from Emory, a private neuro-ophthalmology referral practice. The other group was from Grady, an inner-city facility with primary eye care. Patients with severely affected vision may be more likely referred to a tertiary care center such as Emory. Alternatively, patients of low socioeconomic means may not seek medical care unless they are devastated by their disease. In general, African American patients seen at Emory are not socioeconomically different from whites seen at this center. However, the Emory African American patients had significantly worse vision compared with the Emory white patients. Since both these groups are governed by similar referral patterns, referral bias is an unlikely explanation for the differences observed between these 2 groups. In addition, the visual acuities were significantly worse in our African American patients when the Emory and Grady patients together were compared with the ONTT patients. The inclusion of African American patients from the primary eye care clinic at Grady should reduce the amount of referral bias.

One of the problems with retrospective studies is that patients who are doing well may not follow up, thus biasing the results toward a worse outcome. This was not a significant source of bias in this study. Most of the patients without 1 year follow-up visual acuities were either not yet 1 year out from the onset of their episode of optic neuritis or had a recurrent attack of optic neuritis before the 1 year follow-up visit. In addition, even if it is assumed that all these patients had achieved a final visual acuity of 20/20, the African American study patients still had significantly worse vision at 1-year follow-up compared with the ONTT patients (Table 4).

Finally, the inadvertent inclusion of nondemyelinating optic neuritis secondary to diseases such as sarcoidosis, syphilis, or systemic lupus erythematosus could account for the worse visual acuities observed in the African American study patients. This is unlikely since most of the study patients with poor visual acuity underwent an extensive workup to rule out other causes of optic neuritis. Furthermore, except for age, all the study patients would have fulfilled the rigorous inclusion criteria of the ONTT. Several reports have described patients with an “autoimmune retrobulbar optic neuritis” with a poor visual outcome. Some of these reported patients had severe optic neuritis associated with a transverse myelitis similar to our African American study patients. However, most of these patients had either definite clinical evidence of a collagen-vascular disease, extremely elevated ANA titers, or other results of serological tests indicative of a collagen vascular disease, or a steroid-dependent optic neuropathy with decreased vision with tapering of steroid therapy. Although several of our study patients had positive ANA titers, none had clinical evidence of a collagen-vascular disease, the titers were low, results of further rheumatological and serological workup were unremarkable, and none of the study patients had a steroid-dependent optic neuropathy. Furthermore, the presence of positive ANA titers was not significantly correlated with poor vision. Similarly, the ONTT patients with positive ANA test results (16%) did not have a worse visual outcome.

Although the primary purpose of this study was not to examine the clinical profile of MS, our data showed that 7 (58%) of 12 African American patients with MS had a neuromyelitis optica form of disease, with the most prominent clinical manifestations occurring from optic nerve and spinal cord lesions. Whether neuromyelitis optica represents a variation of MS or a distinct clinicopathological entity is a source of controversy. Whitham and Brey described 2 groups of patients with neuromyelitis optica. One group had a limited monophasic disease, consistent with a postinfectious encephalomyelitis. The other group had a recurrent remitting course consistent with a diagnosis of MS. None of our study patients with neuromyelitis optica had a monophasic course with the simultaneous onset of bilateral optic neuritis and transverse myelitis. All our patients had at least 2 lesions separated in time and space consistent with a diagnosis of MS. As noted above, none of the study patients had evidence of a collagen-vascular disease.

The clinical profile of severe optic neuritis and a “neuromyelitis optica” form of MS in our African American patients is similar to the clinical profile of demyelinating disease described in black South African and Japanese patients. The factors responsible for this similarity are unknown, but genetic and environmental factors must be considered.


©1998 American Medical Association. All rights reserved.