Treatment of Optic Neuritis by Plasma Exchange (Add-On) in Neuromyelitis Optica

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Objective: To assess the contribution of plasma exchange (PE) in association (add-on) with pulsed intravenous corticosteroids in acute optic neuritis of neuromyelitis optica (NMO) and limited forms of NMO.

Methods: Thirty-six patients with optic neuritis were treated from January 1, 1995, through December 31, 2010, with pulsed intravenous corticosteroids and 16 with pulsed intravenous corticosteroids plus PE. The ophthalmologic examination was performed at least 6 months after optic neuritis treatment. Visual acuity and visual field assessed with the Snellen scale and the logarithmic scale of the Early Treatment Diabetic Retinopathy Study were measured using standard automated perimeter and frequency doubling technology perimetry. Retinal peripapillary fiber thickness was measured using optical coherence tomography.

Results: Final visual acuity was 20/400 in the corticosteroid group and 20/50 in the PE group (P = .04). The gain in visual acuity was 20/200 in the corticosteroid group and 20/30 in the PE group (P = .01). A poor final visual acuity outcome (≤20/200) was found in 19 of 36 patients (53%) in the corticosteroid group and 2 of 16 patients (13%) in the PE group (P = .008). Mean (SD) thickness of peripapillary retinal nervous fibers was 63.1 (20.4) µm in the corticosteroid group and 70.3 (20.3) µm in the PE group (P = .16). The mean (SD) thickness in the temporal quadrant was 38.5 (14.1) µm in the corticosteroid group and 44.5 (12.7) µm in the PE group (P = .02). In multivariate analysis, PE treatment was the only independent factor associated with a visual acuity greater than 20/200.

Conclusion: In optic neuritis associated with NMO, sequential treatment with pulsed intravenous corticosteroids and PE is more effective than standard monotherapy with corticosteroids on visual acuity outcome.

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EUROMYELITIS OPTICA

(NMO) is a demyelinating inflammatory disease of the central nervous system, affecting in a quasi-selective manner the optic nerves and the spinal cord.1,2 Some clinical, immunologic, and histologic characteristics distinguish NMO from multiple sclerosis (MS), such as brain sparing at the onset, frequent association (10%-40%) with autoimmune diseases, lesions in white and gray substances of the spinal cord, and presence of necrosis lesions.3,4 Optic neuritis disease is characterized by frequent recurrence and poor visual acuity.5 Median time from onset to blindness of the first eye is 2 years, onset to the attack of the second eye is 3 years, and onset to blindness of the second eye is 13 years. Two attacks are usually enough for definitive loss of vision.6 Percentage of bilateral blindness is as high as 41% in the series by Papais-Alvarenga et al.7 Currently, NMO is considered an autoimmune disease with regard to conditions of humoral immunity, as recently indicated by NMO-IgG directed against aquaporin 4.8 Lesions are characterized by the presence of perivascular deposits of IgM and C9.9

Although numerous studies9,10 have focused on prevention of attacks, such as with immunosuppressors (azathioprine and mitoxantrone) or intravenous γ-globulins, which reduce the frequency and severity of attacks, few data are available on the short-term treatment of optic neuritis and myelitis. High-dose intravenous corticosteroids for the treatment of optic neuritis associated with NMO are systemically used but poorly effective.11 By subtracting the antibody, plasma exchange (PE) showed effectiveness in several neurologic diseases that share similar physiopathologic mechanisms.12,13 Encouraging results were also described in several isolated observations of optic neuritis associated with NMO.14,15 The purpose of our study was to evaluate the contribution of PE in the treatment of acute optic neuritis observed in NMO and limited forms of NMO.
Our ambispective, nonrandomized study was performed at the University Hospital Center of Fort de France in Martinique.

**POPULATION STUDIED**

The study period was January 1, 1995, through December 31, 2010. Inclusion criteria were as follows: inaugural optic neuritis attack related to NMO or limited forms of NMO, treatment with corticosteroids or corticosteroids and PE, extensive eye examination at 6 months or more, and no recurrent optic neuritis between the selected attack and eye assessment. No patient had a previous history of familial, infectious, vascular, compressive, toxic, or nutritional deficiency optic neuritis. All patients tested negative for human immunodeficiency virus and human T-lymphotropic virus.

The diagnosis of NMO was based on diagnostic criteria proposed by Wingerchuk et al. in 1999 and revised in 2006. The revised criteria imply the presence of 2 absolute criteria and 2 or 3 major criteria. The absolute criteria are the existence of unilateral or bilateral optic neuritis and acute myelitis. The major criteria are no abnormalities on the brain magnetic resonance image (MRI) at the onset of the disease, hypersignal extended to more than 3 vertebral segments on the spinal cord MRI, and NMO-IgG antibody positivity. The overall impairment of the disease was rated according to the 0- to 10-point Expanded Disability Status Scale (EDSS). The diagnosis of acute myelitis was defined by the presence of spinal cord symptoms, including sphincter disturbances, sensitivity disturbances, or motor signs. Severe optic neuritis was defined by visual acuity of 20/200 or less. Optic or spinal cord attack was defined by the appearance of new symptoms or the worsening of preexisting signs, lasting at least 24 hours. Limited forms of NMO were defined by a severe unilateral optic neuritis in a patient whose first eye was blind because of optic neuritis, by an inaugural severe bilateral optic neuritis, or by a unilateral optic neuritis characterized by the absence of light perception. All patients were tested for the NMO-IgG antibody.

**TREATMENT**

Both NMO and limited forms of NMO optic neuritis were treated by pulsed intravenous corticosteroids (2 g of methylprednisolone per day for 3-5 days). The treatment was started as soon as the patient was admitted to the hospital. The corticosteroid group refers to optic neuritis treated exclusively by intravenous corticosteroids. In 2006, PE (when available) was added to the corticosteroids. Five daily consecutive PEs were performed in the intensive care unit of our institution. During each cycle, a volume of plasma was exchanged with an albumin solution at 5%. Five exchanges are supposed to withdraw more than 90% of immunoglobulin. Treatment by anticoagulant was systematic.

**STATISTICAL ANALYSIS**

A complete eye examination, which included a precise determination of refraction, was performed at least 6 months after optic neuritis treatment. Visual acuity was measured with the Snellen scale. Optotypes were read at 5 m and were represented by capitalized letters of increasing size, including 12 values of acuity, ranging from 20/400 to 20/20. For calculation of the mean visual acuity, the visual acuity value was converted to logMAR. Visual acuity gain corresponds to the difference between observed visual acuity in the acute phase and visual acuity at 6 months. We also used the logarithmic scale of the Early Treatment Diabetic Retinopathy Study (ETDRS), which is made of black optotypes on white background, with contrast close to 100%.

Standard automated perimetry was performed (Humphrey Field Analyzer 750 II; Carl Zeiss Humphrey). We used the test of central threshold 24-2 (54 points tested) with the FASTPAC strategy (Carl Zeiss Humphrey). We also used frequency doubling technology perimetry. The frequency doubling technology perimetry machine (Carl Zeiss Humphrey) presents stimuli made of clear vertical bars alternating with dark bars under the influence of the phenomenon of doubling frequency. We used the N-30 threshold program. For each perimeter result, we recorded the total deviation corrected with age (mean deviation) and the standard derivation of differences between the threshold value and the expected value at each tested point (pattern standard deviation).

Sensitivity to spatial contrast was measured with the Pelli-Robson and Sloan tests. On the Pelli-Robson test, letters are organized in groups of 3 triplets of different contrast. The cards of weak contrast according to the Sloan test rely on the identification of gray letters with sizes that gradually decrease. The format is similar to that of the ETDRS, and 5 letters are displayed per line. The score is the sum of letters properly read (0-70). Color vision was assessed by the Farnsworth-Munsell 100 hue test. The square root of the score was used for the statistical analysis. The normal value of the score, depending on age, was obtained with Verriest tables.

Measurement of the thickness of peripapillary retinal nerve fibers was performed with optical coherence tomography (Stratus OCT, version 4; Carl Zeiss Meditec Inc). Data were obtained using the fast retinal nerve fiber layer protocol thickness. We measured the mean thickness of the layer of retinal nerve fibers and the mean thickness in temporal, superior, nasal, and inferior quadrants. The recorded scans had a quality of capture of 7 or higher. In the case of narrow pupils, optical coherence tomography was performed after administration of a drop of tropicamide.

For patients with optic neuritis associated with NMO, a poor outcome was defined as a final visual acuity of 20/200 or less (measured ≥6 months after optic neuritis treatment). Then, a logistic regression multivariate analysis, including significant variables ($P < .05$), was used to determine independent clinical factors associated with a poor visual acuity outcome.

Data were computerized and analyzed strictly anonymously with the Excel (Microsoft Inc) and Statview (SAS Institute, Inc) software programs. Statistical analysis used the following tests: $\chi^2$ for frequency comparisons, corrected $\chi^2$ of Yates for small sizes, and $t$ test for mean comparisons. This study was approved by the Consulting Committee for the Protection of Individuals of Biomedical Research sponsored by the French Ministry of Health.

**RESULTS**

**DEMOGRAPHIC CHARACTERISTICS**

Thirty-two patients were selected, 23 with NMO and 9 with limited forms of NMO. Thirty were African-Caribbean, 1 was white, and 1 was Asian. Eleven patients (34%) had endocrinal disturbances of hypothalamic-hypophyseal origin. Two patients had acute disseminated erythematous lupus. Optic neuritis was the first symptom in 22 (69%), whereas myelitis was the inaugural attack in 10 patients (31%). The mean (SD) EDSS score at assessment was 4.8 (2.4).

Fifty-two patients with optic neuritis fit the inclusion criteria. Thirty-six (69%) were treated with intravenous
Table 1. Demographic Characteristics of the Study Patients

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>All (N=52)</th>
<th>Corticosteroid Group (n=36)</th>
<th>PE Group (n=16)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at onset, mean (SD) [range], y</td>
<td>35.6 (11.8) [13-61]</td>
<td>33.2 (9.5) [15-54]</td>
<td>40.5 (14.8) [13-61]</td>
<td>.06</td>
</tr>
<tr>
<td>Female sex</td>
<td>47 (90)</td>
<td>32 (89)</td>
<td>15 (94)</td>
<td>.50</td>
</tr>
<tr>
<td>Disease duration, mean (SD), y</td>
<td>9.2 (5)</td>
<td>10.8 (5)</td>
<td>5.8 (4)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Limited forms of NMO</td>
<td>14 (27)</td>
<td>7 (19)</td>
<td>7 (44)</td>
<td>.07</td>
</tr>
<tr>
<td>NMO-IgG positive</td>
<td>19 (36.53)</td>
<td>13 (36.11)</td>
<td>6 (16)</td>
<td>.86</td>
</tr>
<tr>
<td>Major criteria 1 and 2</td>
<td>35 (67)</td>
<td>29 (81)</td>
<td>6 (38)</td>
<td>.006</td>
</tr>
<tr>
<td>Major criteria 2 and NMO-IgG positive</td>
<td>19 (36)</td>
<td>13 (36)</td>
<td>6 (38)</td>
<td>.99</td>
</tr>
<tr>
<td>Major criteria 1 and 2 and NMO-IgG positive</td>
<td>18 (35)</td>
<td>12 (33)</td>
<td>6 (38)</td>
<td>.99</td>
</tr>
</tbody>
</table>

Abbreviations: NMO, neuromyelitis optica; PE, plasma exchange.

aData are presented as number (percentage) of patients unless otherwise indicated.

bMajor criteria 1 is negative brain magnetic resonance imaging result at onset. Major criteria 2 is spinal cord magnetic resonance image with signal abnormality extending over 3 or more vertebral segments.

corticosteroids and 16 (31%) with sequential corticosteroids and PE. Three patients were treated with corticosteroids alone for one eye and PE for the other eye. Demographic characteristics of all patients with optic neuritis attacks and comparison between the corticosteroid and PE groups are given in Table 1. Mean (SD) disease duration was significantly longer in the corticosteroid group than in the PE group (10.8 [5] vs 5.8 [4.3] years, P < .001). A significant difference was found for patients who met criteria 1 (negative brain MRI result at onset) and 2 (spinal cord MRI with signal abnormality extending over >3 segments). This difference was related to the higher proportion of patients with limited forms of NMO in the PE group than in the corticosteroid group (44% vs 19%). In most cases, patients with limited forms of NMO did not experience an extensive longitudinal myelitis. Actually, they were treated early in their disease history, and often spinal cord attack had not occurred yet.

Table 2. Visual Function of the Study Patients

<table>
<thead>
<tr>
<th>Visual Function</th>
<th>Corticosteroid Group (n=36)</th>
<th>PE Group (n=16)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial visual acuity (Snellen)</td>
<td>20/400</td>
<td>20/400</td>
<td>.73</td>
</tr>
<tr>
<td>Final visual acuity (Snellen)</td>
<td>20/200</td>
<td>20/50</td>
<td>.04</td>
</tr>
<tr>
<td>Visual acuity gain (Snellen)</td>
<td>20/200</td>
<td>20/30</td>
<td>.01</td>
</tr>
<tr>
<td>Final visual acuity = 20/200, No. (%)</td>
<td>20 (56)</td>
<td>2 (13)</td>
<td>.01</td>
</tr>
<tr>
<td>No. (%)</td>
<td>2 (6)</td>
<td>2 (13)</td>
<td>.01</td>
</tr>
<tr>
<td>Final visual acuity &gt; 20/200 and ≤20/40, No. (%)</td>
<td>14 (39)</td>
<td>12 (75)</td>
<td>.01</td>
</tr>
<tr>
<td>Visual acuity ETDRS score</td>
<td>24.4 (12.3)</td>
<td>40.7 (23.4)</td>
<td>.05</td>
</tr>
<tr>
<td>Pelli-Robson test score</td>
<td>0.6 (0.7)</td>
<td>1.2 (0.6)</td>
<td>.01</td>
</tr>
<tr>
<td>Sloan 1.25% test score</td>
<td>2.8 (6.7)</td>
<td>2.5 (5.7)</td>
<td>.5</td>
</tr>
<tr>
<td>Sloan 2.5% test score</td>
<td>6.4 (11.5)</td>
<td>9.6 (12)</td>
<td>.49</td>
</tr>
<tr>
<td>MD SAP</td>
<td>-14.6 (7.1)</td>
<td>-9.6 (8.9)</td>
<td>.02</td>
</tr>
<tr>
<td>PSD SAP</td>
<td>8.1 (2.8)</td>
<td>5 (3.1)</td>
<td>.002</td>
</tr>
<tr>
<td>MD FDTIP</td>
<td>-10.5 (7.5)</td>
<td>-4.7 (5.5)</td>
<td>.01</td>
</tr>
<tr>
<td>PSD FDTIP</td>
<td>7.5 (3.4)</td>
<td>7.7 (6.5)</td>
<td>.54</td>
</tr>
<tr>
<td>Farnsworth-Munsell 100 hue test</td>
<td>108 (141)</td>
<td>152 (191)</td>
<td>.16</td>
</tr>
</tbody>
</table>

Abbreviations: ETDRS, Early Treatment Diabetic Retinopathy Study; FDT, frequency doubling technology perimetry; MD, mean deviation; PE, plasma exchange; PSD, pattern standard deviation; SAP, standard automated perimetry.

aData are presented as mean (SD) unless otherwise indicated.

Table 3. Clinical Characteristics in Poor (<20/200) and Good (≥20/200) Final Visual Acuity Outcome

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Final Visual Acuity ≤20/200 (n=21)</th>
<th>Final Visual Acuity &gt;20/200 (n=31)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at onset, mean (SD), y</td>
<td>33.2 (11.8)</td>
<td>37.2 (11.8)</td>
<td>.23</td>
</tr>
<tr>
<td>Female sex</td>
<td>19 (90)</td>
<td>29 (94)</td>
<td>.99</td>
</tr>
<tr>
<td>Disease duration, mean (SD), y</td>
<td>11 (5.3)</td>
<td>8 (5)</td>
<td>.04</td>
</tr>
<tr>
<td>NMO-IgG positive</td>
<td>11 (52)</td>
<td>8 (26)</td>
<td>.15</td>
</tr>
<tr>
<td>PE treatment</td>
<td>2 (10)</td>
<td>14 (45)</td>
<td>.008</td>
</tr>
</tbody>
</table>

Abbreviations: NMO, neuromyelitis optica; PE, plasma exchange.

aData are presented as number (percentage) of patients unless otherwise indicated.

VisuAl FUNCTION

Visual function between the 2 groups is detailed in Table 2. One significant difference was found between the 2 groups regarding the initial visual acuity (P=.73). The final visual acuity was better in the PE group than in the corticosteroid group (20/400 vs 20/50, P=.04). Visual acuity gain obtained by calculating the difference between final and initial visual acuity was 20/200 in the corticosteroid group and 20/30 in the PE group (P=.01). Seventy-five percent of eyes in the PE group had a final visual acuity superior to 20/40 vs 39% in the corticosteroid group (P=.01). A poor final visual acuity outcome (≤20/200) was noted in 56% of corticosteroid patients and in only 12% of PE patients (P=.01). Alterations of the visual field were also more significant in the corticosteroid group than in the PE group. The mean deviation standard automated perimetry was −14.62 in the corticosteroid group and −9.68 in the PE group. The ETDRS scores, sensitivity to spatial contrast (Pelli-Robson and Sloan 2.5% tests), and color vision were weaker in the corticosteroid group than in the PE group.

A poor final visual acuity was found in 21 eyes (40%). Long disease duration and absence of PE treatment were associated with poor prognosis in univariate analysis (Table 3), whereas PE treatment was the only independent factor related to a final visual acuity greater than 20/200 (odds ratio, 6.8; 95% CI, 1.2-37.4; P=.02).
THICKNESS OF PERIPAPILLARY NERVOUS FIBERS

Thickness of peripapillary nerve fibers is presented in Table 4 for both groups. Although the mean thickness of peripapillary nerve fibers was higher (70.3 µm) in the PE group than in the corticosteroid group (63.1 µm), the difference did not reach significance. It was also higher in each quadrant, and the difference was significant for the temporal quadrant, which was the host of the papillomacular fibers (38.5 vs 44.5 µm, P = .02).

Table 5 gives the principal results obtained in NMO-IgG—positive and NMO-IgG—negative patients. No difference in NMO-IgG status was found.

We report the largest series of optic neuritis associated with NMO and treated at the acute phase by PE. It is the first study, to our knowledge, that compares PE with the conventional treatment of acute optic neuritis by pulsed intravenous corticosteroids. Plasma exchange is effective in improving visual acuity and thickness of nerve fibers of the optic nerve. Plasma exchange leads to a larger gain in visual acuity than corticosteroid standard treatment (20/30 vs 20/200) and is an independent factor of final visual acuity greater than 20/200 after an optic neuritis attack.

In central nervous system demyelinating diseases, PE is often used for severe attacks that were previously resistant to intravenous corticosteroids. Good results were obtained by Weinschenker et al25 in a double-blind, randomized study among 22 patients. Neurologic improvement, measured with the EDSS, was noted in 42% of patients. The study by Keegan et al26 included 59 cases of demyelinating diseases, 10 of which were NMO. Significant improvement of the EDSS score was noted in 60% of NMO patients vs 40% of those with the recurrent remitting form of MS. The earlier PE was performed, the higher the effectiveness. Good results were also observed when PE was performed later than the 60th day after attack onset. Plasma exchange was more effective among males. Bonnan et al24 reported 96 severe medullar attacks, isolated or associated with NMO. Twenty-nine were treated with PE and intravenous corticosteroids. The worsening EDSS score measured after the attacks was lower among patients treated with PE and corticosteroids than among patients treated with corticosteroids alone (1.2 vs 2.6).

Few therapeutic studies took optic neuritis into account. They mostly concerned MS and optic neuritis of unknown causes, and none compared PE and corticosteroids. Llufriu et al25 report 4 cases of severe optic neuritis (visual acuity ≤20/200), 2 associated with MS and 2 idiopathic, treated by PE and intravenous corticosteroids. At 6 months, an amelioration of visual acuity of more than 2 lines was observed for 3 patients. In the study by Ruprecht et al,25 PE was realized with a mean onset-to-treatment delay of 34 days. These investigators report 10 cases of severe optic neuritis (+associated with relapsing-remitting MS and 6 clinically isolated) treated with PE and intravenous corticosteroids. A gain of 2 lines of visual acuity was observed at 10 days in 7 cases. Poor results were noted among patients treated later.

Weinshenker et al23 in a double-blind, randomised study, among 22 patients, noted that PE treatment was associated with a better improvement of visual acuity and thickness of nerve fibers of the optic nerve. Plasma exchange leads to a larger gain in visual acuity than corticosteroid standard treatment (20/30 vs 20/200) and is an independent factor of final visual acuity greater than 20/200 after an optic neuritis attack.

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Our results are in line with 2 series describing corticosteroid and PE treatment in NMO optic neuritis. Nevertheless, our study stands out because of the delay in PE implementation after the patient was admitted to the hospital and intravenous corticosteroids had been administered. Those series have few optic neuritis cases (2 and 3 cases). Plasma exchange was performed approximately 2 weeks after attack onset in response to intravenous corticosteroid failure (between 3 and 6 g of methylprednisolone). Visual acuity improvement was observed soon after the implementation of PE, sometimes even after the first session.14,26 Garcia-Martín et al27 described a patient with NMO who presented with severe optic neuritis and experienced a quasi-complete recovery of visual function 6 months after treatment with intravenous injection of corticosteroids, PE, monoclonal antibodies, and immunosuppressors. Visual acuity recovery was also obtained by using double-filtration plasmapheresis. In the patient with NMO who was treated for bilateral optic neuritis, the PE had to be interrupted because of anaphylactic shock.15 Beside this observation, in most studies, the secondary effects associated with PE are not significant. Nevertheless, complications can be metabolic (hypocalcemia), infectious, allergic, central catheter related, or anticoagulation related (thrombotic and bleeding risk). Minor complications include those that are quickly reversible and easily treatable, such as arterial hypotension, fainting, or paresthesia. Major complications are anaphylactic reactions, respiratory arrest, and myocardial infarction. Plasma exchange requires

### Table 4. Retinal Nerve Fiber Layer Measurements

<table>
<thead>
<tr>
<th>Measurement</th>
<th>PE Group (n = 16)</th>
<th>Corticosteroid Group (n = 36)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall mean RNFL thickness</td>
<td>70.3 (20.3)</td>
<td>63.1 (20.4)</td>
<td>.16</td>
</tr>
<tr>
<td>Temporal quadrant RNFL</td>
<td>44.5 (12.7)</td>
<td>38.5 (14.1)</td>
<td>.02</td>
</tr>
<tr>
<td>Inferior quadrant RNFL</td>
<td>89 (28.4)</td>
<td>77.1 (29.0)</td>
<td>.19</td>
</tr>
<tr>
<td>Nasal quadrant RNFL</td>
<td>55.9 (21.5)</td>
<td>54.4 (17.7)</td>
<td>.98</td>
</tr>
<tr>
<td>Superior quadrant RNFL</td>
<td>89.1 (30.0)</td>
<td>77.3 (33.0)</td>
<td>.24</td>
</tr>
</tbody>
</table>

### Table 5. Visual Function and RNFL Thickness Comparison Between the NMO-IgG—Positive and NMO-IgG—Negative Groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>NMO-IgG Positive (n = 19)</th>
<th>NMO-IgG Negative (n = 33)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Final visual acuity (Snellen, mean (SD))</td>
<td>23 (25.25)</td>
<td>32.58 (25.11)</td>
<td>.22</td>
</tr>
<tr>
<td>Visual acuity ETDRS, mean (SD)</td>
<td>11 (58)</td>
<td>10 (30)</td>
<td>.09</td>
</tr>
<tr>
<td>Overall RNFL thickness (mean (SD), µm)</td>
<td>64.95 (19.95)</td>
<td>63.23 (24.34)</td>
<td>.79</td>
</tr>
<tr>
<td>Temporal quadrant RNFL, mean (SD), µm</td>
<td>40.57 (12.05)</td>
<td>36.23 (13.75)</td>
<td>.17</td>
</tr>
</tbody>
</table>

Abbreviations: PE, plasma exchange; RNFL, retinal nerve fiber layer.
extended surveillance, an appropriate infrastructure, and personnel trained in how to perform this technique. The action mechanism of PE relies on the elimination of responsible agents of the inflammation, such as the antibodies, fractions of the complement, and cytokines. Plasma exchange appears to be more effective the sooner it is used, suggesting that a prolongation of the inflammation would worsen the demyelination and the damages to axons. According to Miyamoto and Kusunoki, regular use of PE leads to the diminution of optic or medullar recurrence. If these factors contribute to the diagnosis, the NMO-IgG antibodies are not a required criterion. The sensitivity of their detection is 60% to 70%. These antibodies are directed against aquaporin 4, a protein of water ca-
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ies are directed against aquaporin 4, a protein of water ca-
NMO-IgG antibodies are not a required criterion. The sen-
ables to PE leads to the diminution of optic or medullar
ment might differ between patients who test positive and
mains incomplete. No current data suggest that treat-
emergent, multicenter study is needed for definitive valida-
Our study reveals that sequential treatment with corticosteroids and PE is more effective than intravenous corticosteroids alone in optic neuritis of NMO. Regarding the poor visual acuity outcome after optic nerve attack in patients with NMO or limited forms of NMO, our center currently uses systematically sequential treatment with pulsed intravenous corticosteroids and PE. Plasma exchange is an effective technique that has been used for several decades. Plasma exchange has become more reliable, and complication rates have decreased. A randomized, multicenter study is needed for definitive validation of PE in the treatment of NMO optic neuritis.

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