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Distinguishing acute-onset CIDP from fluctuating Guillain-Barré syndrome
A prospective study

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On behalf of the Dutch GBS Study Group

ABSTRACT

Objective: The aim of the study was to provide criteria that can help to distinguish between GBS-TRF and A-CIDP in the early phase of disease.

Background: The distinction between Guillain-Barré syndrome (GBS) with fluctuations shortly after start of treatment (treatment-related fluctuations, or GBS-TRF) and chronic inflammatory demyelinating polyneuropathy with acute onset (A-CIDP) is difficult but important because prognosis and treatment strategy largely differ.

Methods: Patients with GBS (n = 170) were included in a prospective longitudinal study. Patients with GBS-TRF (n = 16) and patients with A-CIDP (n = 8) were analyzed and compared. Extended clinical data, biologic material, and electrophysiologic data were collected during 1 year follow-up.

Results: The first TRF in the GBS-TRF group always occurred within 8 weeks (median 18 days; range 10–54 days) from onset of weakness. In the GBS-TRF group, 5 (31%) patients had a second TRF and none had more TRFs. At all timepoints, patients in the A-CIDP group were less severely affected than patients with GBS-TRF, did not need artificial ventilation, rarely had cranial nerve dysfunction, and tended to have more CIDP-like electrophysiologic abnormalities. More GBS-TRF patients were severely affected and more patients had sensory disturbances when compared to the GBS group without fluctuations.

Conclusions: The diagnosis of acute-onset chronic inflammatory demyelinating polyneuropathy (CIDP) should be considered when a patient thought to have Guillain-Barré syndrome deteriorates again after 8 weeks from onset or when deterioration occurs 3 times or more. Especially when the patient remains able to walk independently and has no cranial nerve dysfunction or electrophysiologic features likely to be compatible with CIDP, maintenance treatment for CIDP should be considered. Neurology® 2010;74:1680–1686

GLOSSARY

A-CIDP = acute-onset chronic inflammatory demyelinating polyneuropathy; CI = confidence interval; CIDP = chronic inflammatory demyelinating polyneuropathy; dCMAP = distal compound muscle action potential; DML = distal motor latency; GBS = Guillain-Barré syndrome; GBS-TRF = Guillain-Barré syndrome with treatment-related fluctuations; GRAPH = GBS Research about Pain and Heterogeneity study; IgG = immunoglobulin G; IgM = immunoglobulin M; IVIg = IV immunoglobulin; MFS = Miller Fisher syndrome; mNCV = motor nerve conduction velocity; MP = methylprednisolone; pCMAP = proximal compound muscle action potential; SIDP = subacute inflammatory demyelinating polyneuropathy; SNAP = sensory nerve action potential; sNCV = sensory nerve conduction velocity; TRF = treatment-related fluctuation.

Guillain-Barré syndrome (GBS) and chronic inflammatory demyelinating polyneuropathy (CIDP) are immune-mediated neuropathies, sharing many symptoms and signs in the acute phase of disease.1-3 To differentiate between GBS and CIDP in the early phase of disease, clinicians primarily use the time to reach maximum severity (nadir) and the subsequent course of the disease. GBS is a monophasic disease in which the time to reach nadir by definition is within 4 weeks.4,5 In CIDP, the initial progressive phase lasts more than 2 months, whereafter the course may be relapsing-remitting, steadily progressive, or monophasic.6

However, not all patients fulfill all diagnostic criteria for either GBS or CIDP. It has been reported that 16% of patients with CIDP have rapidly progressive weakness, with a nadir...
within 8 weeks from onset of disease, which is followed by a chronic course. These patients are considered to have acute-onset CIDP (A-CIDP). On the other hand, 8%–16% of patients with GBS have 1 or more deteriorations shortly after initial improvement or stabilization following plasma exchange or IV immunoglobulin (IVIg), described as treatment-related fluctuations (TRF). Additionally a group of patients with a progressive phase of 4–8 weeks and a monophasic course has been described as subacute inflammatory demyelinating polyneuropathy (SIDP). In clinical practice, it may be very difficult to distinguish a patient with GBS having a secondary deterioration after initial improvement or stabilization within the first weeks or months after onset of disease (GBS-TRF) from a patient having a second episode of weakness due to A-CIDP.

Because treatment strategy and prognosis for GBS-TRF and A-CIDP differ considerably, it is relevant to distinguish between these 2 variants early in the course of disease. A patient with GBS-TRF generally requires a repeated IVIg course or plasma exchanges, whereas A-CIDP patients require long-term maintenance treatment with steroids, IVIg, or plasma exchange with or without immunosuppressive agents. In a retrospective study, we suggested that the diagnosis of A-CIDP should be considered when a patient with GBS deteriorates after 9 weeks from onset, or when deterioration occurs 3 times or more. There currently is no prospective study that provides robust criteria that can help to distinguish between GBS-TRF and A-CIDP in the early phase of disease.

Regarding electrophysiologic patterns, a direct comparison between GBS-TRF and A-CIDP in the literature is also lacking. However, patients with A-CIDP seem to have some distinct electrophysiologic features when compared to patients with CIDP with a more chronic onset of disease. Patients with GBS-TRF more frequently have sensory disturbances, but otherwise no distinct electrophysiologic characteristics when compared to patients with GBS.

In this study we prospectively investigated a large number of patients initially diagnosed with GBS. Detailed clinical, biologic, and electrophysiologic characteristics were analyzed in more detail. We aimed to provide more criteria that can help to distinguish between GBS-TRF and A-CIDP in the early phase of disease.

**METHODS**

**Patients.** A total of 170 patients diagnosed with GBS or MFS were prospectively included in the GBS Research about Pain and Heterogeneity (GRAPH) study. During follow-up, part of the patients showed 1 or more TRFs. Some patients initially diagnosed and included in the GRAPH study as having GBS finally were revealed to have a chronic relapsing and remitting course. These patients were reclassified as A-CIDP. Because we aimed to differentiate between GBS-TRF and A-CIDP, we only analyzed these 2 groups of patients.

**Study design.** Between February 2005 and October 2008, patients admitted to one of the 55 participating Dutch centers were included in the GRAPH study. Exclusion criteria were age below 12 and significant comorbidity with a worse prognosis (less than 1 year survival). The protocol was approved by the ethics committee of the Erasmus MC and subsequently by the other participating centers. Clinical data, biologic material, and electrophysiologic data were collected systematically during 1-year follow-up after obtaining written informed consent for participating in the study.

Questionnaires were filled in by the neurologist twice a week during hospital stay and once after 6 months. If the patient, due to deterioration after hospital discharge, visited the hospital again during 1-year follow-up, an additional questionnaire was filled in by the neurologist.

When the patient was discharged from the hospital, additional questionnaires were filled in by the patient or relatives at 3, 6, 9, and 12 months after inclusion. After receiving the questionnaires back, the research coordinator phoned the patient when questions were not filled in.

**Questionnaires.** Baseline characteristics and data about medical history were obtained.

Neurologic symptoms and signs, disability scale (GBS disability scale ranging from 0 "no symptoms or signs" to 6 "paralysis" to 60 "normal strength"), impairment scale (MRC sumscore ranging from 0 “paralysis” to 60 “normal strength”), treatment, and course of disease were obtained from the questionnaire filled in by the neurologist. After hospital discharge, the GBS disability score and course of disease were obtained from the questionnaire filled in by the patient.

To determine nadir, improvement, deterioration, or stabilization during 1-year follow-up, the GBS disability score and MRC sumscore were used. The first progressive phase needs to have its nadir within 4 weeks, in accordance with the criteria for GBS. Thereafter, TRFs (in case of GBS-TRF) and exacerbations (in case of A-CIDP) occurred with their own nadir. Because only part of the exacerbations in A-CIDP is treatment related (especially during the later phase of disease), here we used the term exacerbations instead of TRFs. In every questionnaire, information on improvement, stabilization, or deterioration was obtained and we questioned if there was a new hospital visit or any retreatment. A TRF or exacerbation was defined as follows: 1) improvement in GBS disability score of at least 1 grade or improvement in MRC sumscore of more than 5 points after completion of therapy, followed by a worsening in GBS disabil-
Clinical characteristics, preceding infections, and laboratory findings in the acute phase in patients with GBS-TRF and patients with A-CIDP

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Clinical characteristics, preceding infections, and laboratory findings in the acute phase in patients with GBS-TRF and patients with A-CIDP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GBS-TRF (n = 16)</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>12 (75)</td>
</tr>
<tr>
<td>Age at onset, y, mean ± SD</td>
<td>54 ± 17</td>
</tr>
<tr>
<td>Previous GBS-like episode in medical history, n (%)</td>
<td>1 (6)</td>
</tr>
<tr>
<td>Paresthetic/hypesthetic sensations, n (%)</td>
<td>14 (88)</td>
</tr>
<tr>
<td>Pure motor, n (%)</td>
<td>1 (6)</td>
</tr>
<tr>
<td>Pain before onset of weakness, n (%)</td>
<td>6 (38)</td>
</tr>
<tr>
<td>Pain in acute phase, n (%)</td>
<td>13 (81)</td>
</tr>
<tr>
<td>Cranial nerve dysfunction, n (%)</td>
<td>11 (69)</td>
</tr>
<tr>
<td>III, IV, or VI</td>
<td>6 (38)</td>
</tr>
<tr>
<td>VII</td>
<td>10 (63)</td>
</tr>
<tr>
<td>IX, X, or XII</td>
<td>4 (25)</td>
</tr>
</tbody>
</table>

**Clinical preceding infections**

| Respiratory tract/influenza-like, n (%) | 5 (31)                                                                 | 2 (25)                                                                     | 1.0     |
| Gastroenteritis/diarrhea, n (%) | 4 (25)                                                                 | 2 (25)                                                                     | 1.0     |
| CSF | 2 | 2 (0–5)                                                                                  | 0.30   |
| Protein, g/L, median (95% CI) | 0.9 (0.4–1.8)                                                                  | 0.7 (0.5–1.6)*                                                            | 0.68 |
| Increased protein, >0.55 g/L, n (%) | 10 (63)                                                                 | 4 (57)*                                                                    | 1.0     |
| Antiganglioside antibodies |  |  |  |
| IgM reactivity against GM1, GM2, GD1a, GD1b, or GQ1b | 2 (13)                                                                 | 1 (13)                                                                     | 1.0     |
| IgG reactivity against GM1, GM2, GD1a, GD1b, or GQ1b | 3 (19)                                                                 | 0                                                                           | 0.53    |

**Abbreviations:** A-CIDP = acute-onset chronic inflammatory demyelinating polyneuropathy; CI = confidence interval; GBS-TRF = Guillain-Barré syndrome with treatment-related fluctuations; IgG = immunoglobulin G; IgM = immunoglobulin M.

* n = 7.
* n = 15.
* n = 6.
same items listed in table 1 were also compared between GBS (n = 140) and GBS-TRF (n = 16) patients. The only significant difference we found was a lower percentage of pure motor patients in the GBS-TRF group compared to the GBS group without fluctuations (6% vs 39%; p < 0.05).

**TRFs and exacerbations.** The course of disease during follow-up is indicated in table 2. There was a significant difference in the median time to reach nadir, first TRF/exacerbation, and second TRF/exacerbation between GBS-TRF and A-CIDP. All patients with GBS-TRF had their nadir within 16 days and the patients with A-CIDP within 22 days. The median time to reach nadir in the GBS group without fluctuations was 8 days which was very much comparable with the GBS-TRF group.

The first TRF in the GBS-TRF group was always within 8 weeks (median 18 days; range 10–54 days), and 14 of the 16 patients with GBS-TRF had their first TRF within 4 weeks. Five (31%) patients with GBS-TRF also had a second TRF and none of these patients had more than 2 TRFs. All patients with A-CIDP had their exacerbations after 4.5 weeks (median 51 days; range first exacerbation: 31–63 days). The patients with A-CIDP had 2 to 5 exacerbations until intermittent treatment was started. At all time-points there was a significant difference in level of weakness and severity between GBS-TRF and A-CIDP (table 2). The GBS-TRF group, in comparison with the GBS group without fluctuations, was more severely affected (100% vs 79%; p < 0.05) and contained more ventilated patients (44% vs 15%; p < 0.05) at nadir.

**Laboratory findings.** Table 1 shows the results from the laboratory findings. There were no differences in CSF protein level and number of cells in CSF between GBS-TRF and A-CIDP. In 1 patient with GBS-TRF (6%) and none of the patients with A-CIDP, serologic evidence was found for recent infection with *C. jejuni*. One patient with GBS-TRF had IgG and IgM reactivity against GM1 and GD1b. In 1 patient with GBS-TRF, IgG reactivity against GD1b and GQ1b was found; in another patient with GBS-TRF, IgG reactivity against GD1b was found. In 1 patient with GBS-TRF and 1 patient with A-CIDP, IgM reactivity against GM1 was found.

**Electrophysiologic findings.** Electrophysiologic investigations of 14 patients with GBS-TRF and 8 patients with A-CIDP were performed after 13 days (median; 95% CI 0–16 days). In 18 patients, the electrophysiologic investigations were performed within 3 weeks after inclusion (as was formulated in the protocol). Due to clinical conditions, 4 patients had their electrophysiologic investigation 1 or 2 weeks later. In 6 patients with A-CIDP, a second electrophysiologic investigation was performed (median 67 days, 95% CI 15–187 days). Of 2 patients with GBS-TRF, the electrophysiologic investigations could not be retrieved. The A-CIDP group tended to have more CIDP-like abnormalities (table 3). A higher percentage of patients with A-CIDP showed decreased mNCVs compared to the GBS-TRF group (p = 0.04). The A-CIDP group showed a higher percentage of other demyelinating features, more sensory abnormalities, and a lower percentage of patients showed active denervation. However, these differences did not reach significance. Only 2 patients in the A-CIDP group fulfilled the electrophysiologic criteria for CIDP. Yet, also in the GBS-TRF group, 2 patients fulfilled these criteria. In the second EMG, the demyelinating features of the A-CIDP group were more pronounced, though still only 2 patients fulfilled the strict electrophysiologic criteria for CIDP.
DISCUSSION  Because prognosis and treatment strategy in patients with GBS-TRF and patients with A-CIDP differ, it is important to distinguish these 2 entities in an early phase of disease. We prospectively investigated the differences between patients with GBS-TRF and patients with A-CIDP.

In the current study, 5% of the patients initially diagnosed with GBS were revealed to have A-CIDP. This is the first study that prospectively investigated the development of A-CIDP in a large group of patients initially diagnosed with GBS. By definition, patients with CIDP should have their nadir beyond 8 weeks. In this study, all patients with A-CIDP had their nadir already within 4 weeks, being the reason that they initially were diagnosed with GBS; however, active disease exceeded 8 weeks in all patients with A-CIDP.6,5 In our retrospective study on this issue for which we used our CIDP database, it appeared that over half of the patients with A-CIDP already reached their nadir within 4 weeks.11 The fact that nadir for A-CIDP often already is reached within 4 weeks underscores the diagnostic difficulties between GBS-TRF and A-CIDP. In this study, 10% of the patients with GBS had at least 1 TRF. This percentage is comparable with the percentages described before.9,11,17

This prospective study showed different clinical, biological, and electrophysiologic characteristics of patients with A-CIDP compared to patients with GBS-TRF. The median time to reach nadir, first exacerbation, and second exacerbation was significantly longer in the A-CIDP group compared to the GBS-TRF group. In contrast to patients with A-CIDP, none of the patients with GBS-TRF deteriorated after 8 weeks. Most patients with GBS-TRF had their first deterioration within 4 weeks and none of the patients with GBS-TRF had more than 2 TRFs. At least half of the patients with A-CIDP were able to walk independently at nadir of the different deteriorations and none of the patients with A-CIDP needed artificial ventilation. This is significantly different from the patients with GBS-TRF, where none of the patients were able to walk independently and 44% needed artificial ventilation at nadir of the different deteriorations. In line with the differences in severity based on the GBS disability score, the MRC sumscore was significantly lower in the GBS-TRF group compared to the A-CIDP group. In counting the number of, time to, and severity during the deteriorations it should be considered that therapy is a confounder in both groups. Therefore, we only counted the exacerbations in the A-CIDP group before the start of intermittent treatment.

Patients with A-CIDP had significantly less cranial nerve dysfunction compared with the group of patients having GBS-TRF, which is in line with our previous retrospective study.11 The percentage of patients with cranial nerve involvement and the level of disability and weakness in the A-CIDP group are in line with the clinical characteristics usually found in CIDP.7 There were no differences in preceding infections between the GBS-TRF and A-CIDP group. In GBS-TRF, preceding infections have been described before in a similar percentage.17 There are no studies known about preceding infections in A-CIDP; however, the percentage of preceding infections found in the group of patients with A-CIDP are comparable with the preceding infections found in CIDP.7 None of the patients with A-CIDP had a positive C jejuni serology. In the GBS-TRF group, there was only 1 patient with a positive C jejuni serology and the pure motor form. In a previous study, none of the patients with GBS-TRF had the pure motor form.17

While not significant, patients with GBS-TRF more frequently had IgM and IgG reactivity against antigangliosides as compared to the patients with A-CIDP. IgM anti-GM1 reactivity has been described before.9,11,17

| Table 3 Electrophysiologic findings in patients with GBS-TRF and patients with A-CIDP |
|-------------------------------------------------|-----------------|-----------------|-----------------|
| Demyelinating features, n (%)                  | GBS-TRF (n = 14)| A-CIDP (n = 8)  | p Value*        | A-CIDP second EMG (n = 6) |
| Prolonged DML                                   | 9 (64)          | 6 (75)          | 0.86            | 6 (100)           |
| Decreased mNCV                                  | 4 (29)          | 6 (75)          | 0.04            | 4 (67)            |
| Conduction block and/or temporal dispersion     | 4 (29)          | 3 (38)          | 0.67            | 2 (33)            |
| Increased latency F-wave                        | 5 (50)b         | 5 (83)c         | 0.18            | 5 (100)d          |
| Axonal features, n (%)                          | 7 (54)a         | 6 (75)          | 0.06            | 1 (20)d           |
| Sensory abnormality arms, n (%)                 | 7 (50)          | 0 (0)           | 0.08            | 5 (83)            |
| Classification, n (%)                           |                |                | 0.53            |                  |
| Demyelinating                                  | 9 (64)          | 6 (75)          | 0.83            |                  |
| Axonal                                         | 2 (14)          | 0               | 0               |                  |
| Equivocal                                      | 3 (21)          | 2 (25)          | 1 (17)          |                  |
| Normal                                         | 0               | 0               | 0               |                  |
| CIDP criteria fulfilled                        | 2 (14)          | 2 (25)          | 0.90            | 2 (33)            |

Abbreviations: A-CIDP = acute-onset chronic inflammatory demyelinating polyneuropathy; conduction block or temporal dispersion — with proximal compound muscle action potential/distal compound muscle action potential ratio of < 50% (distal compound muscle action potential =20% lower limit of normal); decreased mNCV = mNCV <90% lower limit of normal (85% if dCMAP <50% lower limit of normal); DML = distal motor latency; F-wave abnormality = F-wave latency >120% upper limit of normal or absent F-wave; GBS-TRF = Guillain-Barre syndrome with treatment-related fluctuations; mNCV = motor nerve conduction velocity; prolonged DML = DML >110% of upper limit of normal (120% if dCMAP <100% of lower limit of normal); sensory abnormality = sensory nerve action potential <50% lower limit of normal or absent.

*p Value of differences between first EMGs of GBS-TRF group and A-CIDP group.

b n = 10.

a n = 6.

c n = 5.

d n = 13.

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scribed before in CIDP and other chronic neuropathies, but in lower percentages than in GBS, comparable with our previous findings. 11

Although for most individual electrophysiologic variables there was no statistical significance, the A-CIDP group displayed a trend toward a more CIDP-like electrophysiologic investigation. 26 Signs of axonal damage (denervation potentials) are rare in the A-CIDP group, while more than half of the patients with GBS-TRF showed signs of axonal damage in the acute phase. Probably the numbers of patients per group were too small to reach statistical significance.

None of the 18 patients with Miller Fisher syndrome (MFS) enrolled in this study developed TRFs. This is a remarkable observation because recurrences of MFS are more frequent compared to GBS. 29

Compared to the group of patients with GBS without TRFs, this study additionally showed that the more severely affected patients with GBS with sensory disturbances are at risk for developing TRFs.

This prospective study confirmed the results of our retrospective study and added more robust factors and refined the results that can help to distinguish more accurately between these variants of inflammatory polyneuropathy in the early phase of disease. 11 These results and our experience indicate that the diagnosis of A-CIDP should be considered when a patient thought to have GBS deteriorates again beyond 8 weeks from onset or when deterioration occurs 3 times or more. Patients with A-CIDP generally are less severely disabled compared to patients with GBS-TRF. Patients remaining able to walk independently at nadir of different deteriorations, having no cranial dysfunction, and showing electrophysiologic features likely to be compatible with CIDP are more likely to have A-CIDP. In these patients, maintenance treatment should be considered.

AUTHOR CONTRIBUTIONS

Statistical analysis was conducted by Dr. L. Ruts.

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