Guillain-Barré syndrome (GBS) is the most frequent diagnosis in patients with acute flaccid palsy characterized by symmetrical motor and/or sensory deficits. In Europe, the underlying polyradiculoneuropathy is usually caused by demyelination and only rarely by axonal loss (5). GBS not only affects the limbs but can also damage the thoracic nerves and nerve roots, eventually necessitating mechanical ventilation. Next to the respiratory complications, cardiac arrhythmia due to vegetative deafferentation can be the greatest danger to the patients. Though the course of GBS can be ameliorated by treatment with immunoglobulins, plasmapheresis, or immunoadsorption, about 10% of patients suffer persistent neurological deficits (16).

GBS is considered an autoimmune disease. The medical history of GBS patients often reveals previous gastrointestinal or respiratory infections. Infections with Campylobacter jejuni and cytomegalovirus (CMV), and possibly also Epstein-Barr virus (EBV) and a mycoplasma, are thought to be the main triggering infectious agents (2, 4, 18, 31). Molecular mimicry of the infectious agent and neural ganglioside antigens (17, 33, 35) are thought to result in cross-reactive humoral and cytotoxic immune responses, leading to neural damage in these patients (15). Increased titers of antiganglioside (GM1, GD1b, GM2) serum antibodies in GBS patients (22, 30), histopathological data (9, 13), and animal studies (32, 34) support this pathogenic model. Nevertheless, in more than 40% of cases, the etiology of GBS remains unknown.

Campylobacter jejuni is an important cause of diarrheal disease in developing and industrialized countries (7), with an incidence second only to Salmonella enterica in the United States and Germany (3, 26).

The reported frequency of previous C. jejuni infections in GBS patients varies substantially (13 to 66%) (16). The association of C. jejuni with GBS seems to vary in different geographic regions. In northern China, the association reaches 66% (14), whereas in Europe, it may be as low as 15% (11).

Diagnoses of previous C. jejuni infections in GBS patients are mostly based on serological findings (16). However, Campylobacter serology is poorly standardized: various crude bacterial antigen preparations have been used for the detection of C. jejuni-specific antibodies in GBS patients (16). This lack of standardization most likely contributes to the discrepancies regarding the association of C. jejuni infections with GBS. The adjustment of these assays to acceptable levels of specificity results in low sensitivity, and we therefore assume that the importance of C. jejuni in triggering GBS has been underestimated. More-specific serological markers for C. jejuni infections are required to obtain reliable epidemiological data in this regard.

We have recently developed a serological assay for the diagnosis of C. jejuni infections that involves two purified recombinant C. jejuni antigens (24), thereby greatly improving Campylobacter serology. High specificity (99.0%) and sensitivity (91.9%) qualify this assay for the accurate assessment of previous C. jejuni infection in GBS patients. We here report a seroepidemiological study of 36 patients with acute GBS.

MATERIALS AND METHODS

Patients and sera. Patients with GBS who had been treated at the Department of Neurology at the University of Göttingen between 1993 and 2003 were included in the study. The inclusion criteria were those defined by the Guillain-Barré Syndrome Study Group (12): progressive motor weakness of more than one limb, areflexia, or at least marked hyporeflexia; cerebrospinal fluid showing albumin-cytologic dissociation with a leukocyte count of less than 20/µl; nerve conduction block; and/or pathological mean F-wave interval/vanished F-wave response. The nadir of clinical symptoms had to be reached within 8 weeks. Patients with fever, severe diabetic polyneuropathy, or alcoholism were excluded. The GBS scores according to van der Meche et al. (28) were obtained from the patients’ files.

The study and particularly the patient recruitment regimen were controlled and approved by the local ethics committee. Pretreatment serum samples were obtained on admission. Anonymous control serum samples were obtained from 57 healthy blood donors. The serum samples
from the healthy blood donors and from 37 subjects with culture-confirmed C. jejuni infections within the preceding 4 weeks (3 to 24 days) were used to define the cutoff values of the C. jejuni-specific P39/P18 enzyme-linked immunosorbent assay (ELISA). All sera were stored at −70°C until used.

Infection serology. Pretreatment serum samples from GBS patients were tested for serological evidence of recent infections with C. jejuni, CMV, and EBV.

The assays for CMV and EBV were performed using commercially available assays with previously established criteria for positivity. CMV infection was defined by the presence of immunoglobulin M (IgM) antibodies in a CE-certified ELISA (Virion/Serion, Würzburg, Germany). EBV infection was defined by the presence of IgM antibodies against the virus capsid antigen (by ELISA; Virion/Serion) in combination with the detection of viral DNA in the serum with an EBV-specific PCR (Artus, Hamburg, Germany). C. jejuni infections were defined by the presence of IgA or IgG in the P39/P18 ELISA previously described (24). Briefly, 96-well ELISA microtiter plates (Greiner, Frickenhausen, Germany) were coated with recombinant affinity-purified P39 and P18, which had been pooled in coating buffer at equal amounts to a final concentration of 0.3 μg/ml. Sera were tested in duplicate at a dilution of 1:100 for the presence of P39/P18-specific antibodies. Specific IgG and IgA antibodies were detected photometrically (optical density at 490 nm [OD490]) using horseradish peroxidase-P39/P18-specific antibodies. Specific IgG and IgA antibodies were detected photometrically (optical density at 490 nm [OD490]) using horseradish peroxidase-conjugated secondary antibodies. OD490 values were adjusted for interassay variation by using the same control sera in each assay. The cutoff OD490 values for the IgA- and IgG-specific ELISAs were established with sera from healthy control sera from 57 healthy blood donors (mean age, 46.9, (18.2 ± 11.9 years) were obtained from the Department of Transfusion Medicine of the University of Göttingen.

The characteristics of the patients included in this study are shown in Table 1. The age of the patients was 60.0 ± 18.2 (mean ± standard deviation [SD]) years. The mean time interval ± SD between the onset of neurological symptoms and serum sampling for the 36 patients was 20.1 ± 16.3 days. Control sera from 57 healthy blood donors (mean age, 46.9, ± 11.9 years) were obtained from the Department of Transfusion Medicine of the University of Göttingen. The male/female ratios were 1.0 in both study groups.

Our findings showed that 29 (80.6%) of the GBS patients had serological evidence of a preceding C. jejuni infection, compared to 3.5% of the healthy controls (χ² test; P < 0.001) (Fig. 1; Table 1). In 23 (63.9%) patients, but in none of the controls, we detected P39/P18-specific IgA antibodies, indicating that a C. jejuni infection had occurred in these GBS patients within weeks preceding the serum sampling (24). In a separate analysis of a subgroup including only GBS patients that were younger than 70 years (n = 25; mean age ± SD, 51.7 ± 14.7 years), serological evidence of a previous C. jejuni infection was found in 88.0%. The Campylobacter-specific complement fixation assay results were positive for 19.4% of all the GBS patients and 1.8% of the controls. Testing for an association between C. jejuni-positive serology and either GBS score or gender did not reveal a statistically significant difference.

A recent CMV infection was detected in two GBS patients (patient G36 and patient G46) (Table 1) and in none of the controls. Recent infections with EBV were not detected in patients or controls.

### RESULTS

Pretreatment sera from 36 patients who fulfilled the criteria of acute GBS were available for serological investigation. All of these patients were Caucasians and had been treated between 1993 and 2003 at the Department of Neurology of the University of Göttingen.

The patients included in this study are shown in Table 1. The age of the patients was 60.0 ± 18.2 (mean ± standard deviation [SD]) years. The mean time interval ± SD between the onset of neurological symptoms and serum sampling for the 36 patients was 20.1 ± 16.3 days. Control sera from 57 healthy blood donors (mean age, 46.9, ± 11.9 years) were obtained from the Department of Transfusion Medicine of the University of Göttingen. The male/female ratios were 1.0 in both study groups.

Our findings showed that 29 (80.6%) of the GBS patients had serological evidence of a preceding C. jejuni infection, compared to 3.5% of the healthy controls (χ² test; P < 0.001) (Fig. 1; Table 1). In 23 (63.9%) patients, but in none of the controls, we detected P39/P18-specific IgA antibodies, indicating that a C. jejuni infection had occurred in these GBS patients within weeks preceding the serum sampling (24). In a separate analysis of a subgroup including only GBS patients that were younger than 70 years (n = 25; mean age ± SD, 51.7 ± 14.7 years), serological evidence of a previous C. jejuni infection was found in 88.0%. The Campylobacter-specific complement fixation assay results were positive for 19.4% of all the GBS patients and 1.8% of the controls. Testing for an association between C. jejuni-positive serology and either GBS score or gender did not reveal a statistically significant difference.

A recent CMV infection was detected in two GBS patients (patient G36 and patient G46) (Table 1) and in none of the controls. Recent infections with EBV were not detected in patients or controls.

### DISCUSSION

In our study, the specific antibody responses to C. jejuni, CMV, and EBV were measured in sera from patients with acute GBS and from healthy controls.

GBS is considered to be a postinfectious autoimmune disease. Infections with C. jejuni and CMV—possibly also with EBV and Mycoplasma pneumoniae—are specifically related to GBS (16, 18). Antecedent C. jejuni and CMV infections have been consistently reported in GBS patients, with frequencies

### TABLE 1. Patient data and Campylobacter serology

| Patient | Gender | Age (yr) | GBS score<sup>b</sup> | Clinical indication(s) of GBS<sup>d</sup> | ELISA | CFA | IgA | IgG |
|---------|--------|---------|-----------------------|-----------------------------------------|-------|-----|-----|-----|
| G16     | M      | 69      | 5                     | 2                                       | Pos   | Pos | Neg |
| G18     | M      | 71      | 5                     | 3                                       | Neg   | Neg | Neg |
| G26     | F      | 80      | 5                     | 3                                       | Neg   | Neg | Pos |
| G27     | M      | 82      | 5                     | 3                                       | Pos   | Neg | Neg |
| G36     | M      | 31      | 5                     | 3                                       | Pos   | Neg | Neg |
| G39     | F      | 75      | 5                     | 3                                       | Pos   | Neg | Neg |
| G44     | F      | 86      | 5                     | 3                                       | Pos   | Neg | Neg |
| G49     | F      | 54      | 5                     | 3                                       | Neg   | Neg | Neg |
| G52     | M      | 66      | 5                     | 3                                       | Pos   | Pos | Neg |
| G54     | M      | 54      | 3                     | 3                                       | Neg   | Pos | Pos |
| G1      | F      | 43      | 4                     | 1                                       | Neg   | Neg | Neg |
| G6      | F      | 61      | 4                     | 3                                       | Pos   | Pos | Neg |
| G12     | F      | 86      | 4                     | 3                                       | Neg   | Neg | Neg |
| G14     | F      | 40      | 4                     | 3                                       | Pos   | Neg | Pos |
| G15     | F      | 60      | 4                     | 3                                       | Pos   | Neg | Neg |
| G22     | M      | 69      | 4                     | 3                                       | Pos   | Pos | Neg |
| G34     | F      | 84      | 4                     | 3                                       | Pos   | Neg | Neg |
| G50     | F      | 72      | 4                     | 3                                       | Pos   | Neg | Neg |
| G51     | F      | 35      | 4                     | 3                                       | Neg   | Neg | Neg |
| G55     | M      | 20      | 4                     | 3                                       | Neg   | Neg | Pos |
| G48     | F      | 52      | 3                     | 3                                       | Neg   | Neg | Pos |
| G5      | M      | 60      | 2                     | 3                                       | Pos   | Pos | Neg |
| G7      | M      | 76      | 2                     | 3                                       | Pos   | Neg | Pos |
| G8      | F      | 58      | 2                     | 3                                       | Pos   | Neg | Pos |
| G10     | F      | 66      | 2                     | 3                                       | Neg   | Neg | Neg |
| G20     | M      | 63      | 2                     | 3                                       | Pos   | Pos | Neg |
| G21     | M      | 37      | 2                     | 3                                       | Pos   | Pos | Neg |
| G24     | M      | 63      | 2                     | 1                                       | Pos   | Neg | Neg |
| G29     | M      | 75      | 2                     | 3                                       | Pos   | Neg | Pos |
| G37     | F      | 81      | 2                     | 3                                       | Neg   | Neg | Pos |
| G46     | M      | 23      | 2                     | 1                                       | Pos   | Pos | Neg |
| G57     | M      | 59      | 2                     | 3                                       | Pos   | Neg | Neg |
| G28     | F      | 31      | 1                     | NA<sup>a</sup>                          | Neg   | Pos | Pos |
| G31     | M      | 47      | 1                     | 1                                       | Pos   | Pos | Neg |
| G53     | M      | 46      | 1                     | 3                                       | Pos   | Pos | Neg |
| G47     | F      | 65      | NA                    | 3                                       | Pos   | Neg | Neg |

<sup>a</sup> M, male; F, female.
<sup>b</sup> See Materials and Methods for the determination of GBS score.
<sup>c</sup> 1, motor defect; 2, sensory defect; 3, motor and sensory defects.
<sup>d</sup> The ELISA was specific for P18/P39. Pos, positive; neg, negative.

NA, data not available.
FIG. 1. Scattergrams showing IgG- and IgA-specific OD_{490} values measured with the *Campylobacter*-specific P18/P39 ELISA in sera from healthy controls and from GBS patients. OD_{490} values were adjusted for interassay variation by using the same control serum sample in each assay. The cutoff values defining a positive result are indicated by dotted lines. As a reference, results from serum samples from 37 patients with recent culture-confirmed *C. jejuni* enteritis are shown.
ranging between 13% and 66% and between 11% and 22%, respectively.

Most of the data supporting the association of antecedent infections with GBS result from serological studies (2, 6, 8, 10, 14, 18, 19, 21, 23, 25, 29, 31). Routinely used serological assays for the diagnosis of CMV and EBV infections are of high quality, with sensitivities and specificities of >90%. C. jejuni-specific serological assays commonly use crude antigenic preparations, such as acid-glycine extracts (1, 18), whole-cell sonicates (2), outer membrane protein preparations, or heat-stable antigens (19). Cross-reactivities with antigens from other bacterial species compromise the quality of these assays. Since the late eighties, intravenous administration of human immunoglobulin (IVIg) has been used for the treatment of GBS (20). IVIg preparations contain antibodies of various specificities. Therefore, the validity of serological results critically depends on the exclusion of posttreatment serum samples. In this regard, it is noteworthy that of 12 published serological studies that investigated antecedent infections in GBS patients, only 2 studies gave information on the time point of serum sampling (18, 23) relative to the beginning of treatment. We think that poor standardization of serological assays for C. jejuni infections and the possible inclusion of posttreatment serum samples in previous studies might explain the various results regarding the association of antecedent infections with GBS, in particular of C. jejuni infections.

In this study, only pretreatment serum samples were included. For the serological diagnosis of a recent C. jejuni infection, a highly specific and highly sensitive ELISA using affinity-purified recombinant outer antigens from C. jejuni (24) was used. Our data showed that the great majority (80.6%) of the GBS patients had serological evidence of a previous C. jejuni infection, compared to 3.5% of the healthy controls. These findings exceeded by far the results obtained with a commercially available Campylobacter-specific CFA, which was used as the reference method, as well as previously reported data (2, 6, 8, 10, 14, 18, 19, 21, 23, 25, 29, 31). Confirmatory data from fecal sample cultures for C. jejuni isolation were not available. However, cultural diagnosis of C. jejuni after cessation of diarrhea is rarely successful (27) and therefore not a reliable method for the diagnosis of antecedent C. jejuni infection in GBS patients. The age difference between GBS patients (mean age ± SD, 60.0 ± 18.2 years) and controls (46.9 ± 11.9 years) does not explain the higher prevalence of C. jejuni-specific antibodies in GBS patients. This is shown in the separate analysis that included only GBS patients who were younger than 70 years. In this subgroup, serological evidence of a preceding C. jejuni infection was even higher (88.0%). This could indicate that the etiology of GBS differs with age. An alternative explanation is the well-known reduced capacity of the elderly to mount a pronounced antibody response. During their hospitalization, most GBS patients included in this study received IVIg, which precluded a confirmatory follow-up of the C. jejuni-specific antibody titers. However, the detection of short-lived C. jejuni-specific IgA (24) in most GBS patients (63.8%) strongly indicates that the C. jejuni infection took place within the previous weeks. Together, our results support our assumption that the frequency of C. jejuni infections in GBS patients has so far been greatly underestimated.

The IgG- and IgA-specific OD490 values in the P39/P18 ELISA tended to be lower in GBS patients than in the C. jejuni enteritis control group (Fig. 1). We have shown in our previous paper (24) that IgA and IgG antibody titers decline during the weeks following infection. The time interval between C. jejuni infection and serum sampling is likely to be much longer in the GBS patients than in the enteritis control group (≤24 days): GBS is a late-onset complication of C. jejuni infections, occurring up to 6 weeks after onset of intestinal symptoms (15), and in this study there was an additional time interval between the onset of neurological symptoms and serum sampling. This suggests that the difference in OD490 values between these two groups is due to the different time intervals between the C. jejuni infection and serum sampling.

CMV infections were detected in 5.6% of the GBS patients compared to none in the controls, and our data do not support an association of EBV infections with GBS. The association of GBS with these herpesvirus infections in our study is lower than that previously reported (18). One explanation for this might be the strict exclusion of posttreatment serum samples in our study, reducing false-positive results in this regard, which might have biased previous studies. Alternatively, there might be differences in the sensitivities of the CMV- and EBV-specific assays.

Additional control sera from patients with other neurological diseases were not available. However, previous serological investigations did not show a significant difference in the prevalence of C. jejuni-specific antibodies between healthy blood donors and patients with other neurological diseases used as controls (18), and we therefore do not think that this additional control group is essential for our study.

This study presents strong evidence that prior C. jejuni infection in GBS patients is far more frequent than previously reported.

Acknowledgments

We thank Michael Köhler from the Department of Transfusion Medicine of the University of Göttingen for providing sera from healthy blood donors. We thank Urs Schmidt-Ott and Friederike Fisher for the critical reading of the manuscript.

References

1. Blaser, M. J., and D. J. Duncan. 1984. Human serum antibody response to Campylobacter jejuni infection as measured in an enzyme-linked immunob:assay. Infect. Immun. 44:292–298.
2. Bouquey, D., C. J. Sindic, M. Lamy, M. Delmee, J. P. Tomasi, and E. C. Laterre. 1991. Clinical and serological studies in a series of 45 patients with Guillain-Barre syndrome. J. Neur. Sci. 104:56–63.
3. Centers for Disease Control and Prevention. 2003. Preliminary FoodNet data on the incidence of foodborne illnesses—selected states, United States, 2002. Morb. Mortal. Wkly. Rep. 52:340–343.
4. Dowling, P. C., and S. D. Cook. 1981. Role of infection in Guillain-Barre syndrome: laboratory confirmation of herpesviruses in 41 cases. Ann. Neur. 9(Suppl.):44–55.
5. Durand, M. C., F. Lofaso, P. J. Lefaucheur, S. Chevet, P. Gajdos, J. C. Raphaël, and T. Sharshar. 2003. Electrophysiology to predict mechanical ventilation in Guillain-Barre syndrome. Eur. J. Neur. 10:39–44.
6. Enders, U., H. Karch, K. V. Toyka, M. Michels, J. Zielske, M. Pette, J. Reesemann, and H. P. Hartung. 1993. The spectrum of immune responses to Campylobacter jejuni and glycoconjugates in Guillain-Barre syndrome and in other neuroimmunological disorders. Ann. Neur. 34:136–144.
7. Friedman, C. R., J. Neimann, H. C. Wegener, and R. V. Tauxe. 2000. Epidemiology of Campylobacter jejuni infections in the United States and other industrialized nations, p. 121–138. In I. Nachamkin and M. J. Blaser, (ed.), Campylobacter, 2nd ed. American Society for Microbiology, Washington, D.C.
8. Gregson, N. A., S. Kohlar, and R. A. Hughes. 1993. Antibodies to ganglio-
sides in Guillain-Barre syndrome: specificity and relationship to clinical features. Q. J. Med. 86:111–117.
9. Griffin, J. W., C. Y. Li, T. W. Ho, M. Tian, C. Y. Gao, P. Xue, B. Mishu, D. R. Cornblath, C. Macko, G. M. McKhann, and A. K. Asbury. 1996. Pathology of the motor-sensory axonal Guillain-Barre syndrome. Ann. Neurol. 39:17–28.
10. Gruenewald, R., A. H. Ropper, H. Lior, J. Chan, R. Lee, and V. S. Molinario. 1991. Serologic evidence of Campylobacter jejuni/coli enteritis in patients with Guillain-Barre syndrome. Arch. Neurol. 48:1080–1082.
11. Guarino, M., M. Casmiro, R. D’Alessandro, et al. 1993. Campylobacter jejuni infection and Guillain-Barre syndrome: a case-control study. Neuroepidemiology 12:296–302.
12. Guillain-Barre Syndrome Study Group. 1985. Plasmapheresis and acute Guillain-Barre syndrome. Neurology 35:1096–1104.
13. Hafer-Macko, C. E., K. A. Sheikh, C. Y. Li, T. W. Ho, D. R. Cornblath, G. M. McKhann, A. K. Asbury, and J. W. Griffin. 1996. Immune attack on the Schwann cell surface in acute inflammatory demyelinating polyneuropathy. Ann. Neurol. 39:625–635.
14. Ho, T. W., B. Mishu, C. Y. Li, C. Y. Gao, D. R. Cornblath, J. W. Griffin, A. K. Asbury, M. J. Blaser, and G. M. McKhann. 1995. Guillain-Barre syndrome in northern China. Relationship to Campylobacter jejuni infection and anti-ganglioside antibodies. Brain 118:597–605.
15. Hughes, R. A., and D. R. Cornblath. 2005. Guillain-Barre syndrome. Lancet 366:1653–1666.
16. Hughes, R. A., and J. H. Rees. 1997. Clinical and epidemiologic features of Guillain-Barre syndrome. J. Infect. Dis. 176(Suppl. 2):S92–S98.
17. Irie, S., T. Saito, K. Nakamura, K. Nanazawa, M. Ogino, T. Nakazawa, H. Ito, Y. Tamai, and H. Kowa. 1996. Association of anti-GM2 antibodies in Guillain-Barre syndrome with acute cytomegalovirus infection. J. Neuroimmunol. 68:49–59.
18. Jacobs, B. C., P. H. Rothbarth, F. G. van der Meche, P. Herbrink, P. I. Schmitz, M. A. de Klerk, and P. A. van Doorn. 1998. The spectrum of antecedent infections in Guillain-Barre syndrome: a case-control study. Neurology 51:1110–1115.
19. Kaldor, J., and B. R. Speed. 1984. Guillain-Barre syndrome and Campylobacter jejuni: a serological study. Br. Med. J. Clin. Res. 288:1867–1870.
20. Kleyweg, R. P., F. G. van der Meche, and J. Meulstee. 1988. Treatment of Guillain-Barre syndrome with high-dose gammaglobulin. Neurology 38:1639–1641.
21. Mishu, B., A. A. Ilyas, C. L. Koski, F. Vriesendorp, S. D. Cook, F. A. Mithen, and M. J. Blaser. 1993. Serologic evidence of previous Campylobacter jejuni infection in patients with the Guillain-Barre syndrome. Ann. Intern. Med. 118:947–953.
22. Rees, J. H., N. A. Gregson, and R. A. Hughes. 1995. Anti-ganglioside GM1 antibodies in Guillain-Barre syndrome and their relationship to Campylobacter jejuni infection. Ann. Neurol. 38:809–816.
23. Rees, J. H., S. E. Soudain, N. A. Gregson, and R. A. Hughes. 1995. Campylobacter jejuni infection and Guillain-Barre syndrome. N. Engl. J. Med. 333:1374–1379.
24. Schmidt-Ott, R., F. Brass, C. Scholz, C. Werner, and U. Gross. 2005. Improved serodiagnosis of Campylobacter jejuni infections using recombinant antigens. J. Med. Microbiol. 54:761–767.
25. Speed, B. R., J. Kaldor, J. Watson, H. Newton-John, W. Tee, D. Noonan, and R. W. Dwyer. 1987. Campylobacter jejuni/Campylobacter coli-associated Guillain-Barre syndrome. Immunoblot confirmation of the serological response. Med. J. Aust. 147:13–16.
26. Stark, K., and K. Alpers. 2004. Bakterielle Gastroenteritiden: Situationsbericht 2003. Epidemiol. Bulletin 31:251–254.
27. Svedhem, A., and B. Kajiser. 1980. Campylobacter fetus subspecies jejuni: a common cause of diarrhea in Sweden. J. Infect. Dis. 142:353–359.
28. van der Meche, F. G., P. I. Schmitz, et al. 1992. A randomized trial comparing intravenous immune globulin and plasma exchange in Guillain-Barre syndrome. N. Engl. J. Med. 326:1123–1129.
29. Vriesendorp, F. J., B. Mishu, M. J. Blaser, and C. L. Koski. 1993. Serum antibodies to GM1, GD1b, peripheral nerve myelin, and Campylobacter jejuni in patients with Guillain-Barre syndrome and control: correlation and prognosis. Ann. Neurol. 34:133–135.
30. Willison, H. J., and N. Yuki. 2002. Peripheral neuropathies and anti-glycolipid antibodies. Brain 125:2591–2625.
31. Winer, J. B., R. A. Hughes, M. J. Anderson, D. M. Jones, H. Kangro, and R. P. Watkins. 1988. A prospective study of acute idiopathic neuropathy. II. Antecedent events. J. Neurol. Neurosurg. Psychiatry 51:613–618.
32. Yuki, N. 2001. Infectious origins of, and molecular mimicry in, Guillain-Barre and Fisher syndromes. Lancet Infect. Dis. 1:29–37.
33. Yuki, N. 1994. Pathogenesis of axonal Guillain-Barre syndrome: hypothesis. Muscle Nerve 17:680–682.
34. Yuki, N., K. Susuki, M. Koga, Y. Nishimoto, M. Odaka, K. Hirata, K. Taguchi, T. Miyatake, K. Furukawa, T. Kobata, and M. Yamada. 2004. Carbohydrate mimicry between human ganglioside GM1 and Campylobacter jejuni lipoooligosaccharide causes Guillain-Barre syndrome. Proc. Natl. Acad. Sci. USA 101:11404–11409.
35. Yuki, N., T. Taki, F. Inagaki, T. Kasama, M. Takahashi, K. Saito, S. Handa, and T. Miyatake. 1993. A bacterium lipopolysaccharide that elicits Guillain-Barre syndrome has a GM1 ganglioside-like structure. J. Exp. Med. 178:1771–1775.
36. Zhou, X., D. K. McClish, and N. A. Obuchowski. 2002. Statistical methods in diagnostic medicine. Wiley, New York, N.Y.