Neurology® Clinical Practice

Research

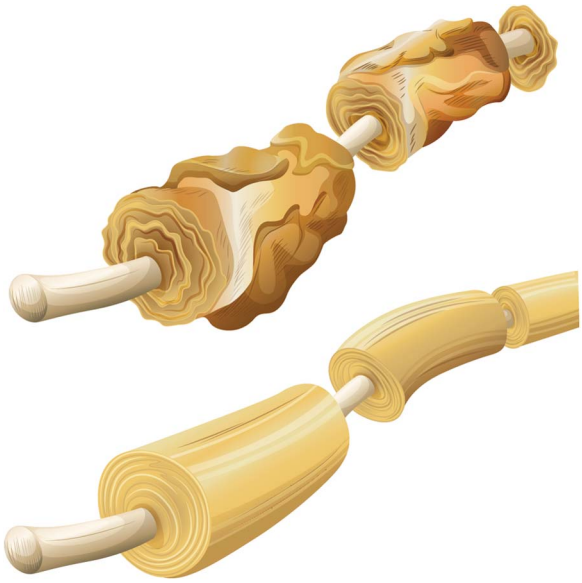
Guillain-Barré syndrome after surgical procedures

Predisposing factors and outcome

Elanagan Nagarajan, MD; Mark Rubin, MD; Eelco F.M. Wijdicks, MD; Sara E. Hocker, MD

Abstract

Background: We sought to identify clinical associations and potential triggers of Guillain-Barré syndrome (GBS) within 8 weeks of surgical procedures. **Methods:** We retrospectively reviewed consecutive patients diagnosed with GBS within 8 weeks of a surgical procedure between January 1995 and June 2014 at Mayo Clinic. Postsurgical GBS was defined as symptom onset within 8 weeks of a surgical procedure. Patients with postsurgical GBS were compared with patients who did not have a surgery or procedure prior to GBS onset to determine differences between groups. **Results:** A total of 208 patients with GBS, median age 55 years (interquartile range [IQR] 41–68), were included. Thirty-one patients (15%) developed postsurgical GBS. Median duration from the surgery or procedure to onset of first GBS symptom was 19 days (IQR 11.1–37.5). The main types of surgeries/procedures preceding GBS were gastrointestinal, cardiac, and orthopedic. General anesthesia was used in 18 (58%) and conscious sedation in 13 (42%) patients. Among the 31 patients with postsurgical GBS, 19 (61%) had a known diagnosis of malignancy. Autoimmune conditions were present in 9 (29%) patients. Additional triggering factors were identified in 11 (35.5%) patients. On univariate analysis, the factors that showed an association with postsurgical GBS were age ($p = 0.003$), malignancy ($p < 0.0001$), active malignancy ($p = 0.05$), preexisting autoimmune disorder ($p = 0.001$), and duration of hospital stay ($p = 0.015$). On multivariate analysis, age ($p = 0.045$), malignancy ($p < 0.0001$), and preexisting autoimmune disorder ($p = 0.004$) remained associated. **Conclusions:** Surgical procedures antedated GBS in 15% of patients, which is unexpectedly high. History of malignancy or autoimmune disease may predispose to development of postsurgical GBS. ***Neurol Clin Pract* 2017;7:1–9**



Department of Neurology (EN, EFMW, SEH), Mayo Clinic, Rochester, MN; Department of Neurology (MR), Northshore University Health System, Glenview, IL; and Department of Neurology, Mayo Clinic, Scottsdale, AZ.

Funding information and disclosures are provided at the end of the article. Full disclosure form information provided by the authors is available with the **full text of this article at Neurology.org/cp**.

Correspondence to: hocker.sara@mayo.edu

Patients with postsurgical GBS were compared with those with GBS and no prior surgery/procedure to determine the differences between groups.

Patients with Guillain-Barré syndrome (GBS) may recall a flu-like syndrome weeks before the onset of symptoms. Other antecedent events have been reported, including trauma, vaccination, blood transfusion, administration of chemotherapy, or surgery. Two publications from Mayo Clinic and Massachusetts General Hospital have reported surgical procedures as a trigger for GBS.[1,2] Since these observations were published, 2 retrospective series noted an incidence of GBS after surgery of 5% and 9.5%, and multiple subsequent case reports have been published, most commonly in patients undergoing cardiovascular, gastrointestinal, or neurosurgical procedures.[1–8] The first detailed study on postoperative polyneuritis reported that postsurgical GBS occurred as a result of the release of nerve antigen during the course of surgery. Further, the authors hypothesized an association with other host factors that caused a vigorous immunologic response, culminating in the development of polyneuritis.[1] Others have postulated that surgical stress induced the activation of neuroendocrine stress axis and cell-mediated immunosuppression, which in turn might promote infections resulting in the production of cross-reactive antibodies.[9,10] We aimed to understand whether patients who presented with GBS within 8 weeks of undergoing a surgery or procedure are different from other patients developing GBS.

METHODS

We retrospectively reviewed medical records of patients diagnosed with GBS between January 1995 and June 2014 at Mayo Clinic. We used the search terms acute demyelinating polyneuropathy, Guillain-Barré syndrome, and Miller Fisher syndrome in the reason for admission, discharge diagnosis, body of the clinical documents, and problem list to identify patients. All charts were reviewed and only patients aged 18–90 years with GBS diagnosed on the basis of diagnostic criteria proposed in 1990 (figure 1) were included.[11] We excluded patients who

Figure 1 **Diagnostic criteria for Guillain-Barré syndrome**

1. Progressive motor weakness of more than one limb with potential involvement of the trunk muscles, bulbar and facial muscles, and external ophthalmoplegia
2. Distal areflexia
3. Reaching the nadir of symptoms within 4 weeks
4. At least one of the following laboratory and electrodiagnostic criteria:
   - A. Albuminocytologic dissociation in cerebrospinal fluid (elevated protein, ≥50 mononuclear leucocytes/mm3)
   - B. Electrophysiological abnormalities that represent a demyelinating affection of the peripheral nerves (prolonged distal latencies, slowed nerve conduction velocities, temporal dispersion of motor action potentials, slowed, or absent F-wave responses)
   - C. Elevated antiganglioside antibodies in serum or cerebrospinal fluid (Asialo-GM1, GM1/2, GD1A/B, GQ1B)

Patients with postsurgical GBS were more likely to have comorbid autoimmune conditions compared to those who developed GBS without a prior surgery.

were diagnosed with GBS outside Mayo Clinic or were experiencing a recurrent episode of GBS.

Variables collected included basic demographics, history of autoimmune disorders and malignancy, other established GBS triggers, surgery/procedure type, type of anesthesia, number of days from the surgery/procedure to onset of GBS, details of treatment for the GBS episode, duration of hospital stay, and recurrence of GBS during follow-up. Active malignancy was defined as a diagnosis of cancer within 6 months of enrollment, any treatment for cancer within the previous 6 months, or documented recurrent or metastatic cancer.[12] The study group was defined as those patients who developed postsurgical GBS, defined as GBS symptom onset within 8 weeks of a surgery or procedure.[3] Patients with postsurgical GBS were compared with those with GBS and no prior surgery/procedure to determine the differences between groups.

Standard protocol approvals, registrations, and patient consents

This study was reviewed by and received approval from our institutional review board. Patients who had not given consent to use their medical records for research were excluded.

Statistical analysis

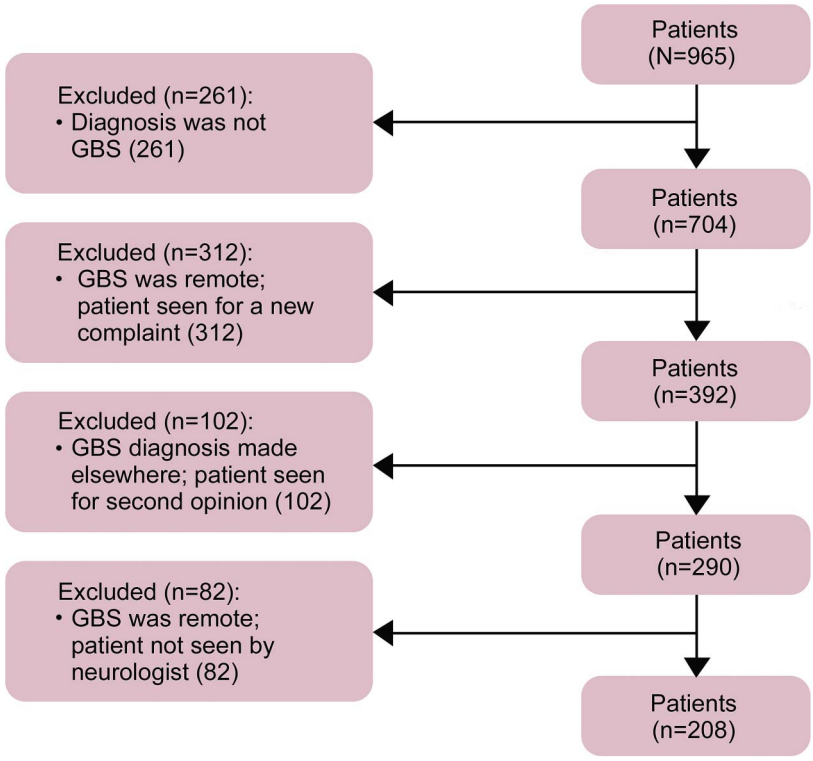
Descriptive summaries were reported using frequencies and percentages for categorical variables and median and interquartile range (IQR) for continuous variables. Statistical significance was set at the conventional 2-tailed α level of 0.05. Associations between the categorical variables were assessed using the χ^2 test or Fisher exact test as appropriate and Kruskal-Wallis test was used to compare the continuous variables as they were not normally distributed. Further analysis was performed using the multivariate logistic regression. Variables with p values of <0.05 were considered as candidate variables for multivariate regression. Since only 3 variables could be analyzed simultaneously in the model (due to small size of the population and to avoid overfitting), multiple models with all possible combinations of important variables were analyzed, before arriving at the final set of included variables. Odds ratio and 95% confidence intervals were used to quantify the strength of associations. All the statistical analyses were performed using JMP 10.0.0 (SAS Institute Inc., Cary, NC).

RESULTS

We identified 969 patients using our search terms, 208 of whom met inclusion criteria (figure 2). Median age was 55 years (IQR 41–68), and 120 (58%) were men. The demographic details are presented in table 1. Thirty-one (15%) patients developed postsurgical GBS. The median age of patients with postsurgical GBS was 63 years (IQR 57–71) and 20 (65%) were men. Details of the surgeries and procedures are included in table 2. The most commonly used type of anesthesia was general in 18 (52%) patients, followed by conscious sedation in 13 (41.9%) patients. No patient developed GBS after a procedure in which they were administered only spinal or local anesthesia. The median duration from the surgery or procedure to the onset of GBS symptoms was 19 days (IQR 11–38).

Nineteen (61%) patients had an associated malignancy and 10 patients (52.6%) had active malignancies. Gastrointestinal (5; 26%) and hematologic malignancies (5; 26%) were the most common type, followed by prostate (3; 16%) and gynecologic cancer (3; 16%). Comorbid

Figure 2 Patient flow diagram



GBS = Guillain-Barré syndrome.

autoimmune conditions were present in 9 (29%) patients with postsurgical GBS. Apart from surgical procedures, other triggers were identified in 11 (36%) patients, including chemotherapy in 5 (45%), blood transfusion in 4 (35%), and trauma and vaccination in 2 (7%) patients each. Twenty (65%) patients were treated with IV immunoglobulin (IVIg), 8 (26%) with plasmapheresis, and 2 (7%) received both IVIg and plasmapheresis. The median duration of hospital stay was 12 days (IQR 6–19). Five (16%) patients died during the hospital stay. The cause of death is unknown in 2 patients, related to underling malignancy in 2 patients, and related to respiratory failure due to pneumonia in 1 patient. The median duration of follow-up after the diagnosis was 42 months (IQR 2–123). Sixteen patients underwent surgical procedures after the diagnosis of GBS and the median number of surgical procedures was 1 (IQR 0–2). There were no recurrences of GBS in these patients.

On univariate analysis, the factors that were associated with GBS after a surgery or procedure were age ($p = 0.003$), malignancy ($p < 0.0001$), active malignancy ($p = 0.05$), or preexisting autoimmune disorder ($p = 0.001$) (table 3). On multivariate logistic regression analysis, age ($p = 0.05$), malignancy ($p < 0.0001$), and preexisting autoimmune disorder ($p = 0.004$) remained significant (table 4).

DISCUSSION

In this large consecutive series of patients with GBS, we found that malignancy and comorbid autoimmune disease were strongly and independently associated with the development of postsurgical GBS, while older age was less strongly associated.

A positive history of autoimmune conditions was present in nearly 10% of the patients with postsurgical GBS in our series. Patients with postsurgical GBS were more likely to have comorbid autoimmune conditions compared to those who developed GBS without a prior surgery. Existing literature with regard to adult populations reported autoimmune conditions to be

Table 1 Demographic and clinical features of adults with Guillain-Barré syndrome

Demographics	Values (total n = 208)
Age, y, median (IQR)	55 (41.3-68)
Male, n (%)	120 (57.7)
Duration of hospital stay, d, median (IQR)	7 (4-16)
Mortality	5 (2.4)
Underlying malignancy, n (%)	46 (22.1)
GI carcinomas	10 (4.8)
Blood cancers	10 (4.8)
Skin cancer	7 (3.3)
Prostate cancer	5 (2.4)
Gynecologic cancer	5 (2.4)
Active cancer	22 (10.6)
Autoimmune conditions, n (%)	20 (9.6)
Ulcerative colitis	5 (2.4)
Type 1 DM	2 (1)
Sjögren syndrome	3 (1.5)
Giant cell arteritis	1 (0.5)
Addison disease (immune)	1 (0.5)
Sarcoidosis	1 (0.5)
Endometriosis	1 (0.5)
Autoimmune hepatitis	1 (0.5)
Immune-mediated thrombocytopenia	1 (0.5)
Antiphospholipid antibody syndrome	1 (0.5)
Graves disease	1 (0.5)
Rheumatoid arthritis	2 (1)
Treatment, n (%)	
IVIg	125 (60.1)
Plasmapheresis	34 (16.3)
IVIg + plasmapheresis	15 (7.2)
Steroids	6 (2.9)
IVIg + Steroids	2 (0.9)
No treatment	26 (12.5)
Triggers, n (%)	32 (15.4)
Chemotherapy	10 (31.3)
Vaccination	8 (25)
Blood transfusion	5 (15.6)
Trauma	3 (9.4)
Recent surgery or procedure	31 (14.9)

Abbreviations: DM = diabetes mellitus; GI = gastrointestinal; IQR = interquartile range; IVIg = IV immunoglobulin.

Table 2 Details of surgeries and procedures

Surgery or procedure	No. (%) (total n = 31)
Gastrointestinal surgery	10 (32.1)
Colonoscopy with biopsy	4 (12.9)
Colectomy	3 (9.6)
Abdominal aneurysm repair	1 (3.2)
Cholecystectomy	1 (3.2)
Post biliary stenting	1 (3.2)
Cardiac surgery	5 (16.1)
Angioplasty with stenting	3 (9.6)
Coronary bypass surgery	1 (3.2)
AICD placement	1 (3.2)
Orthopedic surgery	4 (12.9)
Total knee replacement	2 (6.4)
Hip replacement	1 (3.2)
Bimalleolar fracture open reduction	1 (3.2)
Thoracic surgery	3 (9.6)
Lung lobectomy	1 (3.2)
Lung biopsy	2 (6.4)
Uro-gynecologic surgery	3 (9.6)
Urethral stent placement	2 (6.4)
Hysterectomy and salpingo-oophorectomy	1 (3.2)
Bone marrow biopsy	3 (9.6)
Transsphenoidal resection of pituitary adenoma	1 (3.2)
Oral maxillofacial surgery	1 (3.2)
Septoplasty	1 (3.2)

Abbreviation: AICD = automatic implantable cardioverter defibrillator.

more prevalent in patients with GBS, accounting for 10%–14%. This finding was consistent with the results of the present study and higher than the incidence of autoimmune disorders in the population at large.[13,14] This might be attributed to the transient immunosuppression, which in turn caused an inflammatory response to the neurogenic protein in the peripheral

Table 3 Factors associated with patients developing Guillain-Barré syndrome (GBS) after surgery on univariate analysis

	GBS without recent surgery (n = 177)	GBS after surgery (n = 31)	*p* Value
Age, y, median (IQR)	52 (40.5-66)	63 (57-71)	0.003
Male, n (%)	100 (56.5)	20 (64.5)	0.4
Malignancy, n (%)	27 (15.3)	19 (61.3)	<0.0001
Active malignancy, n (%)	10 (37.1)	12 (66.7)	0.05
Autoimmune, n (%)	11 (6.2)	9 (29)	0.001

Abbreviation: IQR = interquartile range.

Table 4 Factors associated with patients developing Guillain-Barré syndrome after surgery on multivariate analysis

	Odds ratio	95% CI	*p* Value
Age	1.03	1.001–1.06	0.045
Autoimmune conditions	5.19	1.70–15.81	0.004
Malignancy	7.19	3.04–17.69	<0.0001

Abbreviation: CI = confidence interval.

nervous system (PNS), thereby triggering GBS.[15] Multiple previously published case reports have documented the association between GBS and immunodeficiency and malignancy.[16–21] A population-based study in patients with GBS concluded that cancer patients were more prone to develop GBS, with a reported prevalence of 2.4 times higher when compared to the general population.[19] Interestingly, comorbid malignancy was an independent risk factor for the development of postsurgical GBS in the present study when compared to the control group. Still, there is no consensus about the association between postsurgical GBS and malignancy or preexisting autoimmune disease. We presumed that the surgical procedure caused exposure of nerve roots, followed by oncoantigen-mediated misdirection of autoimmune response to epitopes present in the PNS.[19] The other possibility considered was immune dysregulation secondary to the pharmacologic agents (e.g., halothane, enflurane, propofol, ketamine) used during the last few decades. Most of these drugs were lipid soluble and could interact with PNS myelin and initiate the cascade of immunologic process. Such lipid-soluble drugs and trauma to the nerve root could cause demyelinating disorders in the setting of transient immunosuppression.[13] A third possibility is immunosuppression triggering GBS, secondary to surgical procedure, underlying malignancy, or subclinical infections.

Other factors known to be associated with GBS include transplantation, Hodgkin disease, leukemia, infection, vaccination, and blood transfusion.[16–21] Our study population was comparable to previously published series with respect to the distribution of other triggering factors.

During the study period, more than 50,000 surgeries were performed under general anesthesia and innumerable procedures were performed under conscious sedation at the institution where the study was undertaken. The occurrence of GBS after a surgery or procedure was therefore extremely rare and this was demonstrated earlier in large population studies.[3,4,22]

A surgery or procedure preceded the onset of GBS symptoms in 15% of patients in the present study, conducted over 18.5 years. In the Massachusetts General Hospital, a series of 169 patients with GBS was observed from 1962 to 1978. About 10 patients with postsurgical GBS were reported with postoperative wound infections or blood transfusions, and one patient with cytomegalovirus infection. The findings of the present study were higher than what was previously reported with smaller study populations.[3–5,22]

One study reported an incidence of postsurgical GBS of 9.5%. Within a relatively small cohort of 63 patients, 6 patients developed GBS within 6 weeks of a surgery. The affected group included patients with exposure to general anesthesia during the surgical procedure only.[4] We included a large series of 208 patients who underwent both general and conscious sedation. Traditionally, most of the procedures were done under general anesthesia. However, over the last 2 decades, due to advances in surgical techniques and anesthetic medications, higher prevalence of side effects associated with general anesthesia, and endotracheal intubation, most procedures such as colonoscopy, endotracheal biopsy, and laparoscopic surgeries were performed under conscious sedation.[23–26] We presumed that the risk of nerve damage associated with procedures and transient immunosuppression remained the same, regardless of the duration of procedure and type of anesthetic medications used.

In addition, we included patients with symptom onset within 8 weeks from the onset of a surgical procedure. These 2 factors probably accounted for the higher incidence of postsurgical GBS in the current study. In another study, including pediatric and adult patients, 1,034 patients developed GBS during the study period, among whom 52 (5%) experienced symptom onset within 8 weeks of a surgical procedure.[3] In the abovementioned study, patients with other potential triggering factors for GBS occurring within 8 weeks of symptom onset were not excluded. This aspect was similar to the present study.

Mortality rate was 16% among patients with postsurgical GBS. Deaths were predominantly noticed during the acute and early plateau phase. Mortality was shown to be higher in cancer patients who developed GBS when compared to the general population.[19] In an earlier study involving a large series of patients with GBS, the mortality rate was 2.8%–3.9% at 1 year follow-up. It was also observed that death occurred predominantly during the recovery phase.[27] We attributed this to the preexisting comorbid conditions and complications of the surgical procedures in these patients, since the mortality rate for the entire GBS series, including patients without an antecedent surgical procedure, was 2.4%.

The duration of hospital stay in patients with GBS after the surgical procedure was found to be longer when compared to that in patients who did not undergo surgery. This might be due to the time required for postoperative recovery. However, future studies should examine if the course of GBS would be more severe and prolonged in patients with antecedent surgery than without.

We were not able to document the incidence and attributable risk in the study population without an exact number of surgical procedures performed during the study period. Nonetheless, patients developing GBS after a surgery or procedure represent a rare, distinct cohort with more frequent history of malignancy and autoimmunity.

Surgical procedures antedated GBS in 15% of patients, which was unexpectedly high. In most instances, surgery does not predispose patients to new onset of quadriparesis within 8 weeks of the procedure. In the event of such an occurrence, a search for malignancy or autoimmune disease may be warranted.

REFERENCES

1. Arnason BG, Asbury AK. Idiopathic polyneuritis after surgery. Arch Neurol 1968;18:500–507.
2. Wiederholt WC Mulder DW Lambert EH. The Landry-Guillain-Barré Strohl syndrome or polyradiculoneuropathy: historical review, report on 97 patients, and present concepts. Mayo Clin Proc 1964;30:427.
3. Hurwitz ES, Holman RC, Nelson DB, Schonberger LB. National surveillance for Guillain-Barré syndrome: January 1978-March 1979. Neurology 1983;33:150–157.
4. Gensicke H, Datta AN, Dill P, Schindler C, Fischer D. Increased incidence of Guillain-Barré syndrome after surgery. Eur J Neurol 2012;19:1239–1244.
5. Ropper AH, Wijdicks EFM, Truax BT. Antecedent and associated illness, Guillain-Barré syndrome. In: Contemporary Neurology Series, vol 34. Philadelphia: FA Davis; 1991:71.
6. Algahtani H, Moulin DE, Bolton CF, Abulaban AA. Guillain-Barre syndrome following cardiac surgery: difficult diagnosis in the intensive care unit. Neurosciences (Riyadh) 2009;14:374–378.
7. Chang CG, Helling TS, Black WE, Rymer MM. Weakness after gastric bypass. Obes Surg 2002;12:592–597.
8. Papantoni E, Sakorafas GH, Zouros E, Peros G. Guillain-Barré syndrome following total gastrectomy/esophagectomy: a very rare and dramatic post-operative complication with a favourable outcome. ANZ J Surg 2010;80:858.
9. Hogan BV, Peter MB, Shenoy HG, Horgan K, Hughes TA. Surgery induced immunosuppression. Surgeon 2011;9:38–43.
10. Vucic S, Kiernan MC, Cornblath DR. Guillain-Barré syndrome: an update. J Clin Neurosci 2009;16:733–741.
11. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. Ann Neurol 1990;27(suppl):S21–S24.
12. Lee AY, Levine MN, Baker RI, et al. Low-molecular-weight heparin versus a coumadin for the prevention of recurrent venous thromboembolism in patients with cancer. N Engl J Med 2003;349:146–153.

13. Korn-Lubetzki I, Abramsky O. Acute and chronic demyelinating inflammatory polyradiculoneuropathy: association with autoimmune diseases and lymphocyte response to human neuritogenic protein. Arch Neurol 1986;43:604–608.
14. Abramsky O, Korn-Lubetzky I, Teitelbaum D. Association with autoimmune diseases and cellular immune response to the neuritogenic protein in Guillain-Barré syndrome. Trans Am Neurol Assoc 1980;105:350–354.
15. Steiner I, Argov Z, Cahan C, Abramsky O. Guillain-Barré syndrome after epidural anesthesia: direct nerve root damage may trigger disease. Neurology 1985;35:1473–1475.
16. Ozkan A, Taskapilioglu O, Bican A, et al. Hairy cell leukemia presenting with Guillain-Barre syndrome. Leuk Lymphoma 2007;48:1048–1049.
17. Qureshi AI, Cook AA, Mishu HP, Krendel DA. Guillain-Barré syndrome in immunocompromised patients: a report of three patients and review of the literature. Muscle Nerve 1997;20:1002–1007.
18. Re D, Schwenk A, Hegener P, Bamborschke S, Diehl V, Tesch H. Guillain-Barré syndrome in a patient with non-Hodgkin's lymphoma. Ann Oncol 2000;11:217–220.
19. Vigliani MC, Magistrello M, Polo P, Mutani R, Chiò A; Piemonte and Valled'Aosta Register for Guillain-Barré Syndrome. Risk of cancer in patients with Guillain-Barré syndrome (GBS): a population-based study. J Neurol 2004;251:321–326.
20. Brannagan TH III, Zhou Y. HIV-associated Guillain-Barré syndrome. J Neurol Sci 2003;208:39–42.
21. El-Sabrout RA, Radovancevic B, Ankoma-Sey V, Van Buren CT. Guillain-Barré syndrome after solid organ transplantation. Transplantation 2001;71:1311–1316.
22. Sipilä JO, Soilu-Hänninen M. The incidence and triggers of adult-onset Guillain-Barré syndrome in southwestern Finland 2004-2013. Eur J Neurol 2015;22:292–298.
23. Lobo R, Kiernan T. The use of conscious sedation in elective external direct current cardioversion: a single centre experience. BMJ Qual Improv Rep 2015;4. eCollection 2015.
24. Rosati M, Bramante S, Conti F, Rizzi M, Frattari A, Spina T. Laparoscopic salpingo-oophorectomy in conscious sedation. JSLS 2015;19.
25. Grau-Talens EJ, Cattáneo JH, Giraldo R, Mangione-Castro PG, Giner M. Transcylindrical cholecystectomy under local anesthesia plus sedation: a pilot study. Endoscopy 2010;42:395–399.
26. Sivalingam S, Tamm-Daniels I, Nakada SY. Office-based ureteral stent placement under local anesthesia for obstructing stones is safe and efficacious. Urology 2013;81:498–502.
27. Van den Berg B, Bunschoten C, van Doorn PA, Jacobs BC. Mortality in Guillain-Barre syndrome. Neurology 2013;80:1650–1654.

Received June 3, 2016. Accepted in final form August 11, 2016.

AUTHOR CONTRIBUTIONS

E. Nagarajan was responsible for study design, study concept, data collection, data analysis, and drafting the manuscript. M. Rubin was responsible for data collection and revision of the manuscript. E.F.M. Wijdicks was responsible for study concept and manuscript revision. S.E. Hocker was responsible for study design, study concept, drafting the manuscript, and manuscript revision.

ACKNOWLEDGMENT

The authors thank Dr. Tarun Singh for assistance with creating the tables and statistical review.

STUDY FUNDING

No targeted funding reported.

DISCLOSURES

E. Nagarajan and M. Rubin report no disclosures. E.F.M. Wijdicks receives publishing royalties from books published by Oxford University Press. S.E. Hocker serves on a scientific advisory board for SAGE Therapeutics; has received speaker honoraria from the AAN and honoraria from *Continuum*; and serves as an Associate Editor for *Frontiers in Neurology Education*, on the editorial board of *Journal of Stroke and Cerebrovascular Diseases*, and as Review Editor for *Frontiers in Stroke* and *Frontiers in Neurocritical and Neurohospitalist Care*. Full disclosure form information provided by the authors is available with the **full text of this article at Neurology.org/cp**.

Guillain-Barré syndrome after surgical procedures: Predisposing factors and outcome

Elanagan Nagarajan, Mark Rubin, Eelco F.M. Wijdicks, et al.

Neurol Clin Pract published online November 23, 2016
DOI 10.1212/CPJ.0000000000000329