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Original Article

Factors Predicting Prolonged Mechanical Ventilation in Guillain–Barré Syndrome

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ABSTRACT: ***Background:*** Up to 30% of patients with Guillain–Barré syndrome require mechanical ventilation and 5% die due to acute complications of mechanical ventilation. There is a considerable group of patients that will need prolonged mechanical ventilation (considered as >14 days) and should be considered for early tracheostomy. The objective of this study is to identify risk factors for prolonged mechanical ventilation. ***Methods:*** We prospectively analyzed patients with Guillain–Barré diagnosis with versus without prolonged mechanical ventilation. We considered clinical and electrophysiological characteristics and analyzed factors associated with prolonged mechanical ventilation. ***Results:*** Three hundred and three patients were included; 29% required mechanical ventilation. When comparing the groups, patients with prolonged invasive mechanical ventilation (IMV) have a lower score on the Medical Research Council score (19.5 ± 16.2 vs 27.4 ± 17.5, $p = 0.03$) and a higher frequency of dysautonomia (42.3% vs 19.4%, $p = 0.037$), as well as lower amplitudes of the distal compound muscle action potential (CMAP) of the median nerve [0.37 (RIQ 0.07–2.25) vs. 3.9 (RIQ1.2–6.4), $p = <0.001$] and ulnar nerve [0.37 (RIQ0.0–3.72) vs 1.5 (RIQ0.3–6.6), $p = <0.001$], and higher frequency of severe axonal damage in these nerves (distal CMAP ≤ 1.0 mV). Through binary logistic regression, severe axonal degeneration of the median nerve is an independent risk factor for prolonged IMV OR 4.9 (95% CI 1.1–21.5) $p = 0.03$, AUC of 0.774, (95%CI 0.66–0.88), $p = < 0.001$. ***Conclusions:*** Severe median nerve damage is an independent risk factor for prolonged mechanical ventilation.

RÉSUMÉ : Les facteurs prédictifs du recours à la ventilation mécanique prolongée dans le cas du syndrome de Guillain-Barré. ***Contexte :*** Jusqu'à 30 % des patients atteints du syndrome de Guillain-Barré (SGB) ont besoin de ventilation mécanique tandis que 5 % d'entre eux finissent par décéder en raison de complications aiguës liées à ce dispositif. Il existe aussi un groupe considérable de patients qui auront besoin de ventilation mécanique prolongée (VMP) (> 14 jours) et qui devraient être considérés pour une trachéotomie précoce. L'objectif de cette étude est donc d'identifier les facteurs de risque du recours à la VMP. ***Méthodes :*** Nous avons analysé prospectivement des patients ayant reçu un diagnostic de SGB avec ou sans recours à la VMP. Nous avons pris en compte leurs caractéristiques cliniques et électro-physiologiques et ainsi analysé les facteurs associés à ce recours. ***Résultats :*** Au total, ce sont 303 patients qui ont été inclus dans notre étude ; de ce nombre, 29 % d'entre eux ont eu besoin de la ventilation mécanique. En comparant entre eux nos groupes de patients, nous avons trouvé que ceux ayant recouru à la ventilation obligatoire intermittente (VOI) prolongée donnaient à voir des scores moins élevés à l'échelle du *Modified Medical Research Council* (19,5 ± 16,2 contre 27,4 ± 17,5 ; $p = 0,03$), une fréquence plus élevée de dysautonomie (42,3 % contre 19,4 % ; $p = 0,037$), des amplitudes plus basses du potentiel d'action musculaire composé (PAMC ou *compound muscle action potential)* de la partie distale du nerf médian [0,37 (EI 0,07 – 2,25) contre 3,9 (EI 1,2 - 6,4), $p = < 0,001$] et du nerf ulnaire [0,37 (EI 0,0 – 3,72) contre 1,5 (EI 0,3 – 6,6), $p = < 0,001$] et finalement une fréquence plus élevée de dommages axonaux sévères dans ces nerfs (PAMC de la partie distale ≤ 1,0 mV). Grâce à la régression logistique binaire, on a pu noter que la dégénérescence axonale sévère du nerf médian constitue un facteur de risque indépendant du recours à la VOI prolongée [RC : 4,9 (IC 95 % 1,1-21,5), $p = 0,03$; surface sous la courbe : 0,774 (IC 95 % 0,66 - 0,88), $p = < 0,001$]. ***Conclusions :*** En somme, une atteinte grave du nerf médian représente un facteur de risque indépendant du recours à la VMP.

Keywords: Guillain-Barré syndrome; Median nerve; Predictor; Axonal damage; Prolonged mechanical ventilation; Tracheostomy

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Introduction

Guillain–Barre Syndrome (GBS) is the most common cause of acute inflammatory polyneuropathy and represents an important cause of functional disability. It is preceded by an infection (upper respiratory tract or gastrointestinal) in 70% of cases, causing an aberrant immune response directed against the peripheral nerve and its roots. Intravenous immunoglobulin (IVIg) and plasma exchange (PE) have been demonstrated to be effective therapies in the treatment of GBS. Despite early identification and/or proper treatment, 20–30% of patients require invasive mechanical ventilation (IMV) upon admission or during the first week, increasing the risk of complications, prolonged hospital stay, and even death.[1,2]

The most frequent electrophysiological variant in Asia and Latin American countries, including Mexico, is acute motor axonal neuropathy (AMAN), which is commonly associated with preceding diarrhea caused by *Campylobacter jejuni*. Clinically, this variant is associated with a more severe clinical picture compared to the demyelinating variant.[1,3]

Several factors are related to poor short- and long-term functional outcomes and mortality in GBS, including prolonged mechanical ventilation (>14 days).[4] The rapid progression and severity of muscle strength impairment as well as the involvement of lower cranial nerves are risk factors for mechanical ventilation.[5] Diaphragmatic weakness due to phrenic nerve damage, as well as lower cranial nerve involvement, are anatomically involved in respiratory failure in GBS.[6]

Prolonged mechanical ventilation has been studied in countries where acute inflammatory demyelinating polyneuropathy (AIDP) is the most common variant. However, there is little information on countries where AMAN is the most frequent variant. The duration of mechanical ventilation may vary from few days to months. Optimal timing for tracheostomy is uncertain and early tracheostomy is associated with less delirium, analgesic use, and faster oral nutrition. However, it may be associated with bleeding, pneumothorax, and local injuries, and some patients may recover ventilatory function before needing tracheostomy. On the other hand, delayed tracheostomy is associated with esophageal, vocal cord, and tracheal damage due to the prolonged presence of an endotracheal tube.[7] Optimal clinical and paraclinical decision-making is required to identify the subset of patients who benefit from early tracheostomy. The objective of the present study is to identify clinical and/or electrophysiological factors associated with prolonged IMV (>14 days) in patients with GBS to select which patients would benefit from early tracheostomy.

Methods

A prospective observational study was conducted in patients with GBS diagnosis. We included patients fulfilling Asbury criteria[8] from a single healthcare neurological center in Mexico from January 2018 to December 2021. We included patients with versus without prolonged mechanical ventilation. Prolonged mechanical ventilation was defined as >14 days.[6] Data collected for all patients were age, gender, time from symptom onset to admission, preceding infections, cranial nerve involvement, muscle strength by Medical Research Council (MRC) sumscore at admission, GBS disability scale (GDS) at admission, EGRIS scale score at admission, autonomic dysfunction (variability in heart rate or blood pressure not explained by any medical condition, at the decision of the treating physician), and treatment were considered. Cerebrospinal fluid (CSF) analysis was also obtained in admission, and albumin-cytological dissociation was defined as CSF protein >45 mg/dl with CSF cell count ≤50 cells/microliter.

We obtained nerve conduction studies within 1 week of admission and included the following data: distal latency, conduction velocity and distal compound muscle action potential (CMAP) of the ulnar, median, tibial, and peroneal nerves, as well as recordings of sensory nerve action potentials of median and sural sensory nerves. We applied the *Uncini* criteria to classify GBS into axonal, demyelinating, equivocal, or nonexcitable using valued adjusted to our population. We defined severe distal motor axonal damage as CMAP ≤ 1.0 mV.[9]

Ethical Approval/Informed Consent

This project was approved by the institutional review board and complies with ethical guidelines.

Statistical Analysis

For descriptive analysis, continuous variables were described as means or medians according to their distribution. Categorical variables were described in frequencies and percentages. To search for differences between groups, we used x2 and Fisher's exact test for categorical variables, Student's *t*-test to compare means, and Mann–Whitney *U* test to compare medians. A value of $p < 0.05$ was considered statistically significant. Survival analysis was performed using Kaplan–Meyer curves to observe independent gait recovery between groups, and a Log-Rank value <0.05 was considered significant.

An analysis was performed to identify risk factors for prolonged mechanical ventilation, following the TRIPOD consensus to develop a predictive model.[10] We developed a univariate and multivariate logistic regression model. We included the following covariates for the univariate model: MRC sumscore of 0 on admission, severe axonal damage of the median nerve, severe axonal damage of the ulnar nerve, and presence of nerve conduction block in any explored nerve. We included the following variables for the multivariable model: MRC sumscore of 0 at admission, severe axonal damage of the median nerve, and the ulnar nerve. We assessed the goodness-of-fit by the Hosmer–Lemshow test and the model performance by the area under the curve analysis. Results are reported as odds ratios (OR) with 95% confidence intervals (95%CI). All statistical analyzes were performed using SPSS 22.0

Results

In the cohort of 303 patients with GBS, 29% of patients required mechanical ventilation at admission or within the first week. Regarding ventilated patients, 64.8% were male, with a mean age of 46.7 ± 18.9, median MRC score scale of 22.7 ± 17.5 points, and median EGRIS score of 4 (IQR 4–6) points. All patients received either IVIg (68.2%) or PE (31.8%). Fifty-nine percent of ventilated patients required prolonged mechanical ventilation.

In the comparative analysis of baseline characteristics, we observed that patients with prolonged mechanical ventilation had lower scores on the MRC sumscore (19.5 ± 16.2 vs 27.4 ± 17.5, $p = 0.03$); a score of 0 on this scale (25 % vs 5.5%, $p = 0.02$), and a lower percentage of recovery of independent walking at 6 months of follow-up (22% vs 92%, $p = <0.001$) (Table 1) (Figure 1). Patients who did not require prolonged mechanical ventilation were more likely to have unilateral facial paralysis and/or the Miller-Fisher/Overlap GBS clinical variant.

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Table 1: Baseline characteristics of ventilated patients

	Mechanical ventilation >14 days $N = 52$	Mechanical ventilation <14 days $N = 36$	*P* value
Age (years), mean (SD)	49.3 ± 17.9	45.3 ± 18.1	0.30
Age ≥60 years, *n* (%)	14 (27)	8 (22.2)	0.80
Male, *n* (%)	32 (61.5)	25 (69.4)	0.50
Time from symptom onset to admission (days), median (IQR)	5 (2–7.5)	3 (2–7.5)	0.70
Symptom onset to admission ≤3 days, *n* (%)	22 (42.3)	20 (55.5)	0.27
Preceding respiratory infection, *n* (%)	12 (23)	11 (30.5)	0.46
Preceding diarrhea, *n* (%)	21 (40.3)	12 (33.3)	0.65
MRC sumscore at diagnosis mean (SD)	19.5 ± 16.2	27.4 ± 17.5	**0.03**
MRC sumscore ≤30, *n* (%)	42 (80.7)	23 (63.8)	0.08
MRC sumscore ≤20, *n* (%)	28 (53.8)	14 (38.8)	0.19
MRC sumscore ≤10, *n* (%)	17 (32.7)	7 (19.4)	0.22
MRC sumscore 0, *n* (%)	13 (25%)	2 (5.5%)	**0.02**
Deltoid strength ≤2 at admission, *n* (%)	39 (75)	21 (58.3)	0.11
EGRIS (points), median (IQR)	5 (4–6)	5 (3–5.25)	0.08
Cranial nerve involvement, *n* (%)	42 (80.7)	29 (80.5)	0.99
Unilateral facial, *n* (%)	3 (5.7)	10 (27.7)	0.006
Bilateral facial, *n* (%)	30 (57.7)	18 (50)	0.51
Bulbar, *n* (%)	34 (65.4)	24 (66.6)	0.99
Autonomic dysfunction, *n* (%)	24 (46%)	21 (58.3)	0.28
Clinical variants:			
Sensorimotor, *n* (%)	29 (55.7)	18 (50)	0.66
Pure motor, *n* (%)	18 (34.6)	10 (27.7)	0.64
Miller Fisher/Overlap, *n* (%)	1 (1.9)	5 (13.8)	0.04
Pharyngocervicobrachial, *n* (%)	3 (5.7)	1 (2.7)	0.63
Albuminocytological dissociation, *n* (%)	15 (28.8)	10 (27.7)	0.78
Protein levels (mg/dl), Median (IQR)	38.5 (28–80)	33.5 (24–60)	0.53
Treatment:			
IVIg, *n* (%)	38 (73)	22 (61.1)	0.25
PE, *n* (%)	14 (27)	14 (38.9)	
Hospital stay (days), median (IQR)	52 (33–80)	15 (19–28)	<0.001
Independent gait recovery at 6 months follow-up. *n* (%)	9/41 (22)	22/24 (92)	<0.001

SD: standard deviation, IQR: interquartile range, IVIg: intravenous immunoglobulin, PE: plasma exchange, MRC: medical research council.

Regarding the comparative analysis of electrophysiological characteristics, it is important to highlight that there were no differences in the time of performing the electrophysiological study between both groups. There were no significant differences between the frequency of electrophysiological variants (Table 2). Interestingly, we observed that the group of patients with prolonged mechanical ventilation presented lower amplitudes of the distal CMAP of the median [0.37 mV (0.07–2.25) vs 3.9 mV (1.2–6.4), $p = <0.001$] and ulnar nerves [0.37 mV (0.0–3.72) vs 1.5 mV (0.3–6.6), $p = <0.001$], as well as a higher frequency of severe axonal damage (≤1.0 mV) in both nerves.

In the multivariate analyses using binary logistic regression, it was observed that severe axonal degeneration of the median nerve (≤1.0 mV) is an independent risk factor for prolonged IMV with OR of 4.8 (95% CI 1.1–21.3) $p = 0.03$ (Table 3). The performance of this multivariate model through AUC was 0.74, 95% CI (0.62–0.86), $p = 0.001$.

Discussion

Mechanical ventilation increases the risk of mortality up to threefold when compared to nonmechanical ventilation subjects. In addition, it increases the risk of nosocomial pneumonia and length of hospital stay.[11,12] Twenty-nine percent of patients in our cohort required mechanical ventilation, like other cohorts.[7] Treatment in GBS is aimed at curbing the immune response that causes damage

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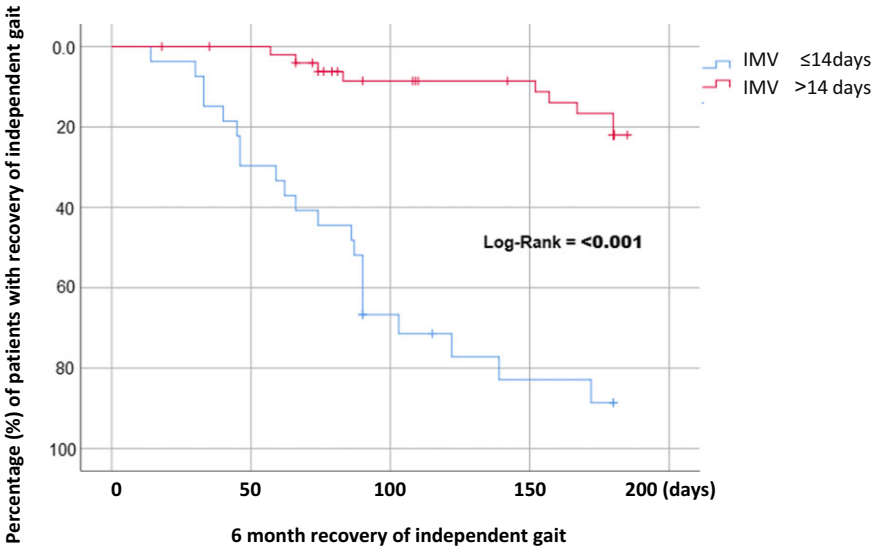

Figure 1: Percentage (%) of patients with recovery of independent gait at 6 months follow-up.

Table 2: Electrophysiological characteristics from ventilated patients

	Mechanical ventilation >14 days $N = 41$	Mechanical ventilation ≤14 days $N = 29$	*P* value
Time from symptom onset to nerve conduction study realization (days), median (IQR).	6 (3–12)	6 (3–15)	0.66
Electrophysiological findings			
AIDP, *n* (%).	13 (31.7)	12 (41.3)	0.45
AMAN, *n* (%).	16 (39)	10 (34.4)	0.80
AMSAN, *n* (%).	5 (12.1)	3 (10.3)	0.99
Inexcitable, *n* (%).	6 (14.6)	1 (3.4)	0.22
Equivocal, *n* (%).	2 (4.8)	3 (10.3)	0.64
Distal CMAP (mV):			R
Median, median (IQR).	0.3 (0.07–2.25)	3.9 (1.2–6.4)	**<0.001**
Ulnar, median (IQR).	0.37 (0.0–3.72)	3.4 (1.1–5.9)	**<0.001**
Tibial, median (IQR).	0.75 (0.1–2.55)	1.5 (0.3–6.6)	0.054
Peroneal, median (IQR).	0.5 (0.0–2.57)	0.7 (0.1–3.2)	0.06
SNAP (μV):			
Median, median (IQR).	0.1 (0–1.0)	0.26 (0.0–1.0)	0.30
Sural, median (IQR).	11.6 (3.45–20.4)	10.3 (0.0–20)	0.63
Severe axonal damage (distal CMAP ≤1.0 mV):			
Median, *n* (%).	28 (68.3)	7 (24.1)	**0.001**
Ulnar, *n* (%).	28 (68.3)	9 (31)	**0.003**
Tibial, *n* (%).	23 (56)	10 (34.4)	0.31
Peroneal, *n* (%).	25 (61)	16 (55.1)	0.80
Complete conduction block, *n* (%).	11 (26.8)	16 (55.1)	**0.014**

mV: milivolts; μV: microvolts; SNAP: sensory nerve action potential; CMAP: compound muscular action potential.

to the myelin or axon of the peripheral nerves. However, even if patients are diagnosed and treated early, 20–30% have a poor long-term functional outcome and 5% die.[13-15] Both intravenous human immunoglobulin and PEs are equally effective in the treatment of GBS.[1] In our center, we have both therapies; however, we decided to treat with intravenous human immunoglobulin patients with GBS who enter the institution requiring mechanical ventilation for two reasons: 1) because a large percentage of these patients have hemodynamic compromise due to cardiovascular dysautonomia, PEs would increase the risk of worsening the

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Table 3: Risk factors for prolonged mechanical ventilation

	Univariate analysis				Multivariate analysis	
	Mechanical ventilation >14 days	Mechanical ventilation ≤14 days	OR (95%)	*P* value	OR (IC95%	*P* value
MRC sumscore 0	13 (25%)	2 (5.5%)	1.6 (1.2–2.1)	0.02	2.1 (0.37–12.6)	0.39
Median severe axonal damage, *n* (%)	28 (68.3)	7 (24.1)	7.5 (2.3–23.6)	0.001	**4.8 (1.1–21.3)**	**0.03**
Ulnar severe axonal damage, *n* (%)	28 (68.3)	9 (31)	4.7 (1.7–13.3)	0.003	1.6 (0.4–6.35)	0.49
Complete conduction block, *n* (%)	11 (26.8)	16 (55.1)	0.27 (0.1–0.7)	0.013		

Model: Chi-squared 15.45, GL 3, $p = 0.001$.
Hosmer–Lemoshow: Chi-squared 0.661, GL 3, $p = 0.88$.
Model accuracy: AUC: 0.74, IC 95% (0.62-0.86), $p = 0.001$.

hemodynamic state and 2) intravenous human immunoglobulin is easier to make available for immediate initiation of treatment in our institution.

Mechanical ventilation is a risk factor for poor functional outcomes (short and long term) in patients with GBS, but in patients with prolonged ventilation, the prognosis works, it is even more discouraging. In our population only 22% of patients recovered independent gait at 6 months of follow-up.[5,16] Risk factors include rapid and severe progression of muscle weakness and involvement of lower cranial nerves (VII, IX, and X). These clinical variables are incorporated into the EGRIS scale, which is the most widely used to assess the risk of mechanical ventilation on admission.[6] In our cohort, patients without mechanical ventilation were admitted to the hospital in a greater number of days from the onset of symptoms versus patients requiring ventilation [6 (IQR 2–9) vs 3 (IQR 2–6) days, $p = < 0.001$]. Interestingly, there was no significant difference between patients with prolonged ventilation versus non-prolonged ventilation (Table 1).

The presence of albumin-cytological dissociation in CSF represents nerve root involvement.[17] Although patients clinically present with severe proximal muscle weakness, less than 30% of patients with mechanical ventilation present protein elevation. This may be because patients were admitted early (median 5 days), as it is well-known that protein elevation is present in 90% of patients at 3 weeks since symptom onset.[3]

Prolonged mechanical ventilation increases mortality and complications (pressure ulcers, infections, neuropathy, and critically ill myopathy, etc.).[18] The definition varies in the literature. Some authors consider from 5 days to 21 days (19). In GBS, some authors consider prolonged mechanical ventilation as >2 months. However, most authors consider it in >14 days, as we did.[5,20] A study reported that older age is a risk factor for prolonged mechanical ventilation >21 days. (21) In our study, we observed that age > 60 years is not associated with prolonged mechanical ventilation.[5] Severe impairment of muscle strength, measured by the MRC sumscore is a risk factor for the mechanical ventilation.[6] In our population, we observed that severely affected patients with zero points on the MRC score at admission are at risk for prolonged mechanical ventilation. Fourrier et al analyzed 40 patients with GBS and observed that patients who could not dorsiflex the foot at the end of immunotherapy were at risk for prolonged mechanical ventilation >15 days.[22] Walgaard et al reported that severe decrease in strength (≤2 points) in some muscles (deltoid, biceps, extensor carpi, iliopsoas, quadriceps, and tibialis anterior) measured 1 week after admission were also risk factors for prolonged mechanical ventilation ≥14 days.[5] From our consideration, the neuromuscular physical examination in ventilated patients with several days of hospitalization may be influenced by conditions typical of critically ill patients (delirium, hydro-metabolic alterations, sedation effects) that may alter an appropriate assessment.[23]

Nerve conduction studies are very important as they establish the primary mechanism of peripheral nerve damage (demyelinating or axonal).[2] In some patients who undergo the very early studies (≤3 days of evolution within symptom onset), a second study is suggested because 20% of patients with AIDP fulfill criteria for axonal variants afterwards.[24,25] For patients with mechanical ventilation, we consider that the initial study is the most important as subsequent neurophysiological studies may be altered due to medication (use of sedatives or muscle relaxants), hydroelectrolytic disturbances, and overlap with critically ill neuropathy and myopathy.

AMAN has a worse functional outcome compared to AIDP, and a previous study in our country reported a higher frequency of mechanical ventilation in axonal variants.[1,26] In our study, we did not observe differences between the electrophysiological variants requiring prolonged mechanical ventilation. Severe axonal damage (distal CMAP ≤1.0 mV) is associated with a worse outcome.[16,27]

However, the presence of distal CMAP ≤1.0 mV in motor nerve recordings may be due to several factors. In the AMAN variant, it may be due to length-dependent conduction failure because of severe damage in proximal portions of the nerve (undergoing Wallerian-like degeneration), and in the case of the demyelinating variant, it may be due to distal conduction blocks.[28]

Prior to this study, no electrophysiological risk factors were published associated with prolonged mechanical ventilation. Interestingly, we observed that patients with prolonged mechanical ventilation present severe axonal damage in the motor nerves of the upper extremities (median and ulnar), but not in the motor nerves of the lower extremities (tibial and peroneal), resulting in the multivariate analysis that the severe axonal damage of the median nerve is an independent risk factor for prolonged IMV.

In GBS, both the peripheral nerve and nerve roots are damage by the immune response, including cervical (some form the brachial plexus), thoracic, and lumbar roots. In the case of patients with GBS with prolongated ventilation, an important factor is diaphragmatic weakness due to damage to the phrenic nerves that exit the cervical roots C3-C4-C5; however, nerve conduction studies of the phrenic nerve are not technically easy to obtain and, in most hospitals, do not have adequate technical equipment.[29-31] Based on the above, researchers have reported valuable clinical observations, *Walgaard et al.,* observed that the decrease in the strength of the deltoid muscle was considered a risk factor for prolonged ventilation, as it represents an indirect data on the affection of the C5

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root which is also part of the phrenic nerve.[5] Following the concept of cervical root involvement and association with prolonged IMV, we observed that severe damage of the median nerve (C8-T1 roots) is an independent risk factor for prolonged mechanical ventilation in patients with GBS. We theorize severe damage to the median nerve may be a "window" to assess the severity of damage to the cervical roots. Other nerve conduction studies are used to assess nerve roots such as F-wave measurement, but this is not valuable when distal CMAP are severely diminished.[9]

Limitations

Our study has some limitations. As a single-center study, we report the experience and protocols used in our center, which may not be the same for other healthcare centers. As other studies, the group of ventilated patients was small to obtain a prognostic model. We hope that this study sets a background for further studies to identify potential risk factors for prolonged mechanical ventilation. Another limitation is that we did not have serological or fecal studies to document infection by *Campilobaceter jejuni* and antiganglioside studies.

Conclusion

Our study proves association between prolonged mechanical ventilation (established as more than 14 days) with longer hospital stay and poor functional outcomes in GBS. Moreover, an independent risk factor for prolonged mechanical ventilation was severe median nerve axonal damage (CMAP ≤1.0 mV) in nerve conduction studies.

Conflicts of Interest. None of the authors report any conflicts of interest.

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