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Can neurophysiology and nerve ultrasound differentiate acute-onset CIDP from GBS with treatment-related fluctuations?

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Abstract

Distinguishing acute-onset chronic inflammatory demyelinating polyneuropathy (A-CIDP) from Guillain-Barré syndrome (GBS) with treatment-related fluctuations (TRF) in the early phase of disease can be challenging. Although characteristic clinical features have been previously reported, there is limited data on neurophysiological features. We aim to identify the electrophysiological and ultrasonographic features that might help differentiate between these two conditions. Patients with GBS-TRF and A-CIDP were identified from an existing cohort of GBS patients presenting to University of Malaya Medical Centre, Kuala Lumpur, Malaysia from 2011 to 2020. The clinical, electrophysiological and nerve ultrasound data were recorded and analysed. Five GBS-TRF (mean age 42 ± 23 years) and five A-CIDP (mean age 66 ± 13 years) patients were included. The mean time to first neurological deterioration was longer in A-CIDP compared to GBS-TRF (11 ± 5 vs 5 ± 1 weeks, p=0.028). Based on two sets of nerve conduction studies (NCS), both GBS-TRF and A-CIDP patients fulfilled the electrodiagnostic criteria for demyelinating neuropathy. A-CIDP patients had more prolonged ulnar minimal F-wave latencies (40.8 ± 5.8 vs 28.6 ± 2.2 ms, p=0.020) and slower sural conduction velocities (26.3 ± 8.6 vs 41.4 ± 3.4 m/s, p=0.015) on NCS. Nerve ultrasound showed significantly larger cross-sectional area of ulnar nerve at the wrist (7 ± 2 vs 5 ± 1 mm^2, p=0.037) and forearm (8 ± 1 vs 5 ± 1 mm^2, p=0.025) in A-CIDP patients. The Ultrasound Pattern Sum Score-A was significantly higher in A-CIDP compared to GBS-TRF (10 ± 3 vs 5 ± 1, p=0.015). We found nerve electrophysiological and ultrasonographic features can be useful in differentiating between GBS-TRF and A-CIDP.

Keywords: Guillain-Barré syndrome, chronic inflammatory demyelinating polyneuropathy, treatment-related fluctuation, nerve ultrasound, acute-onset CIDP, nerve conduction study

INTRODUCTION

Classical Guillain-Barré syndrome (GBS) patients have a monophasic illness with nadir typically reached within 4 weeks[1], whereas in typical chronic inflammatory demyelinating polyneuropathy (CIDP), there is a subacute presentation with initial disease progression lasting > 8 weeks.[2] However, in up to 10% of GBS patients receiving plasma exchange (PLEX) or intravenous immunoglobulin (IVIG), there can be neurological deterioration after the initial improvement or stabilization. This group of patients is referred to as GBS with treatment-related fluctuations (GBS-TRF).[3,4] In 16% of CIDP patients, the clinical presentation can be acute with nadir reached less than 8 weeks from disease onset mimicking GBS, and this is referred to as acute-onset-CIDP (A-CIDP).[5] Distinguishing A-CIDP from GBS-TRF can be challenging, especially in the early phase of the disease. Clinical features supportive of A-CIDP include a longer time to first deterioration and a higher number of deteriorations.[3] A-CIDP patients are also less severely affected, less likely to be mechanically ventilated, and rarely had cranial nerve dysfunction.[3]

Reports of electrophysiological and nerve ultrasound features of A-CIDP and GBS, specifically GBS-TRFs are limited. Sural-sparing pattern and sensory ratio are two electrophysiological parameters that have been found to be useful markers of the acute inflammatory demyelinating polyneuropathy

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(AIDP) subtype of GBS.[6] However, neither parameters have been shown to differentiate AIDP from CIDP.[7,8] Instead, nerve ultrasound has been found to be helpful in distinguishing CIDP from GBS and other neuropathies[9], with larger nerves supporting a diagnosis of CIDP. Its utility in A-CIDP and GBS-TRF is less certain.

In the current study, we investigated the electrophysiological and nerve ultrasound features of patients with GBS-TRF and A-CIDP in order to identify markers that might help differentiate between the two diagnoses.

METHODS

GBS patients admitted to University of Malaya Medical Centre (UMMC), Kuala Lumpur between 2011 and 2020 were retrospectively reviewed. The diagnosis of GBS was made based on established criteria.[10] Patients with an initial diagnosis of GBS who developed deterioration after immunotherapy were further classified as GBS-TRF or A-CIDP based on the following definitions. In GBS-TRF, a TRF was defined as deterioration of the Medical Research Council (MRC) sum score or GBS disability score within 8 weeks after an initial improvement or stabilization with IVIG or PLEX.[3] A-CIDP was diagnosed when patients had a relapse after 8 weeks or had ≥ 3 relapsing and remitting events.[3]

Patient demographic data and clinical characteristics, including history of antecedent illness, sensory dysfunction, cranial nerve involvement, autonomic dysfunction and mode of treatment were obtained. Muscle strength was determined based on MRC sum score (ranging from 0 "paralysis" to 60 "normal strength") whereas functional ability was based on the GBS disability score (ranging from 0 "no symptoms or signs" to 6 "dead").[11,12] Both scores were determined at onset and nadir.

As part of their investigations, all patients also had cerebrospinal fluid (CSF) analysis and nerve conduction study (NCS) at presentation. A second NCS was repeated within 3-8 weeks of disease onset. NCS was performed using the Natus Synergy® EMG machine as previously described.[13] Briefly, 3 motor nerves (median, ulnar and tibial) and 3 sensory nerves (median, ulnar and sural) were tested. Sural-sparing pattern was defined as the presence of an abnormal ulnar SNAP with a normal sural SNAP amplitude.[14] The sensory ratio was calculated as follows: (sural + radial) / (ulnar + median) SNAP amplitudes.[6] A sensory ratio of > 1 was used as a cut off to compare between the two groups. Reference values were derived from normal ranges that were previously established at our laboratory.[15] The electrodiagnosis of GBS was determined based on the two-study criteria by Uncini *et al*.[16]

Nerve ultrasound was performed using a broad-band linear transducer (Mindray®, M7) with the frequency set at 14-MHz. Measurements of nerve cross-sectional area (CSA) of the median, ulnar, superficial radial, tibial, fibular and sural nerves were obtained as previously described.[17] Nerve ultrasound was performed after patients developed their first deterioration. The sub-score Ultrasound Pattern Sum Score A (UPSA) was calculated based on the unilateral findings of median nerve at upper arm, elbow and mid-forearm; ulnar nerve at upper arm and mid-forearm; tibial nerve at popliteal fossa and ankle, and fibular nerve at lateral popliteal fossa. Each nerve enlargement < 50% of the upper limit of normal was scored 1 and each enlargement > 50% was scored 2. A maximum score which takes into account eight measurement points was 16 points.[9] Reference nerve ultrasound data were derived from established values from our laboratory.[18] The study was approved by the UMMC research ethics committee and all patients provided informed consent.

Statistical analysis

Categorical data were presented as frequencies and percentages, and continuous variables were displayed as mean ± standard deviation (SD). Continuous data were compared with Mann-Whitney *U*-test and categorical data were compared with Fisher exact test. Corrections for multiple testing were not performed because the aim of the study was to investigate any differences in electrophysiology and nerve ultrasound between patients with GBS-TRF and A-CIDP in the context of an exploratory study. Significance level was set at p values < 0.05. Statistical analyses were performed with SPSS v24 (IBM Corp., USA) for Windows.

RESULTS

Clinical characteristics and disease course

From 130 patients presenting with an initial diagnosis of GBS, five patients each fulfilled the criteria for GBS-TRF and A-CIDP respectively. Mean age of patients with GBS-TRF and A-CIDP were 42 ± 23 years and 66 ± 13 years respectively (Table 1). Both groups had equal gender distribution (3 males: 2 females). Sensory deficits

were present in four (80%) patients in each group. Cranial nerve dysfunction was more common in GBS-TRF (60%) with facial involvement in two patients and bulbar weakness in one patient; as compared to facial involvement in only one patient with A-CIDP group. Antecedent infection was documented in three (60%) GBS-TRF patients and four (80%) A-CIDP patients. Autonomic dysfunction was reported in one GBS-TRF and two A-CIDP patients. These three patients experienced hemodynamic instability along the course of illness. Both groups demonstrated CSF albuminocytological dissociation. All patients from both groups received IVIG as the first line treatment in the acute phase of the disease. During subsequent episodes of clinical deterioration, all but one patient in each group received further courses of IVIG with or without steroids. The one patient from each group who did not have IVIG was treated with PLEX instead.

The mean time to first neurological deterioration was 10.7 ± 5.2 weeks in A-CIDP and 4.5 ± 1.2 weeks in GBS-TRF (p=0.028) (Table 1). The mean number of deteriorations were equal in

Table 1: Clinical characteristics, laboratory findings, course, number, and severity of deteriorations in GBS-TRF and A-CIDP

Characteristics	GBS-TRF (n=5), n (%)	A-CIDP (n=5), n (%)	P value
Demographic			
Sex (M:F)	3:2	3:2	ns
Age of onset, years, mean (SD)	42 (23)	66 (13)	ns
Clinical features			
Sensory deficits	4 (80)	4 (80)	ns
Cranial nerve dysfunction			
Oculomotor	0 (0)	0 (0)	ns
Facial	2 (40)	1 (20)	ns
Bulbar	1 (20)	0 (0)	ns
Antecedent infection			
Upper respiratory tract	3 (60)	1 (20)	ns
Gastroenteritis	0 (0)	3 (60)	ns
Autonomic dysfunction	1 (20)	2 (40)	ns
Cerebrospinal fluid			
White cells, x10^6/L, mean (SD)	7 (9)	3 (5)	ns
Protein, g/L, mean (SD)	2.3 (1.6)	1.5 (0.6)	ns
Disease course			
Days to reach nadir, mean (SD)	16 (9)	12 (4)	ns
Weeks to reach first deterioration, mean (SD)	4.5 (1.2)	10.7 (5.2)	0.028
Number of deterioration, mean (SD)	2 (0)	2 (1)	ns
Disease severity			
MRC sum score at nadir, mean (SD)	36 (3)	39 (10)	ns
GDS at nadir, mean (SD)	4 (0)	4 (1)	ns
MV along disease course	0 (0)	2 (40)	ns
Treatment			
Onset (n)	5 IVIG	4 IVIG 1 IVIG then PLEX	-
Deteriorations (n)	2 IVIG x2 2 IVIG x2 + CS 1 PLEX	2 IVIG x2 1 IVIG x1 1 IVIG x3 + CS 1 PLEX	-

M: male, F: female, GDS: GBS disability score, MRC: Medical Research Council, MV: mechanical ventilation, IVIG: intravenous immunoglobulin, PLEX: plasma exchange, CS: corticosteroid, ns, not significant

both groups (two episodes each). The severity of the disease at nadir as assessed by MRC sum score and GBS disability score were similar in both conditions. Two A-CIDP patients required mechanical ventilation during the course of their illness whereas none were ventilated in the GBS-TRF group.

Electrophysiology and ultrasound findings

There were no differences in the timing of the two sets of NCS between the two groups (Table 2). Patients from both GBS-TRF and A-CIDP groups fulfilled the electrodiagnostic criteria for AIDP. A-CIDP patients demonstrated more marked demyelinating features than GBS-TRF on first and second study, as evidenced by longer distal motor latencies, slower motor conduction velocities and more prolonged/absent minimal F-waves latencies. In the first study, the ulnar F-wave (40.8 ± 5.8 vs 28.6 ± 2.2 ms, p=0.020) were more affected in A-CIDP. The tibial motor conduction velocities (CV) showed a slower trend in A-CIDP (32.1 ± 3.4 vs 38.0 ± 4.8 m/s, p=0.050). For the sensory studies, sural amplitudes were relatively preserved in the GBS-TRF group compared to A-CIDP on first and second study (first set: 23.9 vs 6.0 μV; second set: 10.4 vs 4.8 μV). The sural CV were slower in second study in A-CIDP group (26.3 ± 8.6 vs 41.4 ± 3.4 m/s,

Table 2: Electrophysiological findings in GBS-TRF and A-CIDP

Parameters		First study, mean (SD)		P value	Second study, mean (SD)		P value
		GBS-TRF (n=5)	A-CIDP (n=5)		GBS-TRF (n=5)	A-CIDP (n=5)	
Days from onset		10.4 (7.3)	10.6 (7.4)	ns	42.0 (6.5)	44.6 (21.5)	ns
Motor studies							
Median	DML, ms	6.8 (3.5)	11.3 (7.7)	ns	9.6 (6.2)	9.8 (5.0)	ns
	dCMAP, mV	7.9 (4.1)	4.2 (3.7)	ns	6.7 (4.5)	2.9 (2.6)	ns
	CV, m/s	44.9 (10.9)	35.0 (8.5)	ns	40.9 (6.5)	37.7 (8.1)	ns
Ulnar	DML, ms	4.3 (1.3)	6.0 (3.1)	ns	5.5 (2.5)	6.0 (3.2)	ns
	dCMAP, mV	4.7 (2.4)	3.9 (1.4)	ns	4.6 (1.8)	3.0 (0.9)	ns
	CV, m/s	46.1 (12.6)	48.8 (9.7)	ns	42.4 (13.7)	40.6 (8.9)	ns
Tibial	DML, ms	6.8 (2.2)	9.7 (4.2)	ns	11.2 (6.1)	10.7 (4.4)	ns
	dCMAP, mV	7.3 (3.6)	4.1 (2.5)	ns	3.4 (2.3)	1.5 (1.0)	ns
	CV, m/s	38.0 (4.8)	32.1 (3.4)	ns	31.4 (4.7)	30.3 (4.3)	ns
F-wave							
Median, ms		36.0 (11.7)	49.7 (13.3)	ns	45.8 (17.4)	40.0 (11.1)	ns
Ulnar, ms		28.6 (2.2)	40.8 (5.8)	0.020	46.8 (14.2)	37.6 (12.9)	ns
Tibial, ms		45.8 (21.4)	67.6 (8.3)	ns	70.0 (5.0)	38.1 (-)	ns
Sensory studies							
Median	SNAP, μV	14.2 (5.0)	14.6 (9.0)	ns	5.2 (6.6)	6.5 (2.1)	ns
	CV, m/s	41.0 (8.3)	35.0 (1.8)	ns	38.9 (8.5)	36.3 (1.5)	ns
Ulnar	SNAP, μV	10.6 (6.2)	11.1 (11.3)	ns	2.6 (1.5)	9.9 (7.2)	ns
	CV, m/s	39.5 (10.7)	34.1 (2.4)	ns	38.6 (7.6)	37.4 (0.4)	ns
Sural	SNAP, μV	23.9 (16.0)	6.0 (1.0)	ns	10.4 (8.0)	4.8 (0.7)	ns
	CV, m/s	37.9 (4.9)	37.0 (8.8)	ns	41.4 (3.4)	26.3 (8.6)	0.015
Sural-sparing pattern, n (%)		0/5 (0%)	2/4 (50%)	ns	3/5 (60%)	0/4 (0%)	ns
Sensory ratio > 1, n (%)		4/4 (100%)	4/4 (100%)	ns	2/4 (50%)	5/5 (100%)	ns
Uncini *et al*. criteria		All fulfil AIDP			All fulfil AIDP		

DML: distal motor latency, dCMAP: distal compound muscle action potential, CV: conduction velocity, SNAP: sensory nerve action potential, AIDP: acute inflammatory demyelinating polyneuropathy, ns: not significant

p=0.015). At first study, sural-sparing pattern was not seen in the GBS-TRF but present in 2/4 A-CIDP patients. On second study, 3/5 GBS-TRF demonstrated sural-sparing pattern compared to none of the A-CIDP patients.

Nerve ultrasound was performed following the first clinical deterioration. Data was available for four patients in each ultrasound group (Table 3). In the A-CIDP patients, the mean time of ultrasound was longer compared to GBS-TRF patients (209 ± 50 vs 53 ± 23 days from onset of disease). In comparison to GBS-TRF, A-CIDP patients had larger CSA of ulnar nerve at the wrist (7 ± 2 vs 5 ± 1 mm^2, p=0.037) and forearm (8 ± 1 vs 5 ± 1 mm^2, p=0.025). The overall UPSA score was significantly higher in A-CIDP compared to GBS-TRF (9.7 ± 2.5 vs 4.8 ± 2.5, p=0.015). All A-CIDP patients had UPSA ≥ 7 in comparison to none in the GBS-TRF group (p=0.029).

DISCUSSION

In the current study, we describe the clinical, electrophysiological and nerve ultrasound features of patients with GBS-TRF and A-CIDP. We found patients with A-CIDP were more likely to relapse later in the course of their illness, have greater delays in F-waves latencies on NCS and larger nerves on nerve ultrasound.

Previous studies have described clinical characteristics that distinguish GBS-TRF and A-CIDP[3,7,8,19,20] and these, along with the current study are summarised in Table 4. In keeping with other studies, our GBS-TRF patients had an earlier clinical deterioration (mean 5 weeks) when compared to A-CIDP patients (mean 11 weeks).[3,21] This was not unexpected as the definition of the dichotomous classification was based on the time to first deterioration. However, in contrast to previous reports, our A-CIDP patients were more severely affected with 2/5 patients requiring mechanical ventilation compared to none of the GBS-TRF patients.[3]

All GBS-TRF and A-CIDP patients in the current study fulfilled the electrodiagnostic criteria of AIDP based on two sets of NCS[16], in keeping with previous reports.[3] We also found prolonged ulnar F-wave latencies in A-CIDP at the first NCS when compared to GBS-TRF. This suggests that despite the acute presentation, A-CIDP patients may have subclinical demyelination that was

Table 3: Nerve ultrasound findings in GBS-TRF and A-CIDP

Ultrasound cross-sectional area, mm^2		**GBS-TRF (n=4), mean (SD)**	**A-CIDP (n=4), mean (SD)**	**P value**
Days from onset		53 (23)	209 (50)	0.001
Median	Wrist	11 (3)	14 (3)	ns
	Forearm	10 (3)	10 (3)	ns
	Elbow	11 (2)	16 (7)	ns
	Midarm	12 (5)	14 (5)	ns
Ulnar	Wrist	5 (1)	7 (2)	0.037
	Forearm	5 (1)	8 (1)	0.025
	Elbow	8 (1)	9 (3)	ns
	Midarm	7 (1)	8 (1)	ns
Superficial radial		3 (1)	2 (1)	ns
Tibial	Ankle	23 (8)	13 (4)	ns
	Popliteal	20 (6)	33 (14)	ns
Fibular	Fibular head	9 (3)	12 (4)	ns
	Popliteal	8 (2)	17 (12)	ns
Sural		3 (1)	3 (1)	ns
Ultrasound Pattern Sum Score-A (UPSA)		4.8 (1.0)	9.7 (2.5)	0.015
UPSA ≥ 7, n (%)		0 (0)	4 (100)	0.029

ns: not significant

Table 4: Differences in clinical, neurophysiological and ultrasound characteristics reported in existing literature

Study	Ruts *et al.*[3]	Dionne *et al.*[7]	Alessandro *et al.*[19]	Grimm *et al.*[20]	Kerasnoudis *et al.*[8]	Our study
Comparison (study sample size)	GBS-TRF (16) vs A-CIDP (8)	AIDP (30) vs A-CIDP (15)	AIDP (77) vs A-CIDP (14)	GBS (33) vs CIDP (34)	AIDP (15) vs A-CIDP (20)	GBS-TRF (5) vs A-CIDP (5)
Clinical features						
Antecedent illness	ns	More in AIDP	ns	ND	ns	ns
Sensory symptoms	ns	ns	ns	ND	More in A-CIDP	ns
Proprioception/ vibration disturbances	ND	More in A-CIDP	More in A-CIDP	ND	ND	ND
Sensory ataxia	ND	More in A-CIDP	More in A-CIDP	ND	ND	ND
Autonomic dysfunction	ND	More in AIDP	ns	ND	ns	ns
CN involvement	More in GBS-TRF	More in AIDP	ns	ND	More in AIDP	ns
Mechanical ventilation	More in GBS-TRF	More in AIDP	ns	ND	More in AIDP	ns
Timing to deterioration	Longer in A-CIDP	ND	ND	ND	ND	Longer in A-CIDP
Number of deteriorations	More deterioration in A-CIDP	ND	ND	ND	ND	ns
Nerve conduction study						
SNAP	ns	ND	ND	ND	ND	ns
SCV	ND	ND	ND	ND	ND	Slower in A-CIDP
Increased F-wave latency	ns	ND	ND	ND	ND	Longer in A-CIDP
Decreased MCV	More in A-CIDP	ND	ND	ND	ND	Slower in A-CIDP
Sural-sparing pattern	ND	ns	ND	More in GBS	ns	ns
Sensory ratio > 1	ND	ns	ND	ND	ns	ns
A-waves	ND	ns	ND	More in GBS	ns	ND
Nerve ultrasound						
Cross-sectional area	ND	ND	ND	ND	ND	Larger in A-CIDP
Ultrasound pattern sum score	ND	ND	ND	(UPSS, UPSA*, UPSC†) Larger in A-CIDP	ND	(UPSA) larger in A-CIDP
Bochum ultrasound score‡ ≥ 2	ND	ND	ND	ND	More in A-CIDP	ND

ND: no data, ns: not significant, CN: cranial nerve, SNAP: sensory nerve action potential, SCV: sensory conduction velocity, MCV: motor conduction velocity, UPSS: ultrasound pattern sum score, UPSA: ultrasound pattern sum score A, UPSC: ultrasound pattern sum score C
* scoring for median nerve upper arm, elbow and mid-forearm; ulnar nerve upper arm and mid-forearm; tibial nerve popliteal and in the ankle, and peroneal nerve popliteal
† scoring for sural, superficial radial, and superficial peroneal nerve
‡ scoring for ulnar nerve in the Guyon canal, ulnar nerve in the upper arm, radial nerve in the spiral groove, sural nerve between the lateral and medial head of the gastrocnemius muscle

present before clinical threshold was reached. Interestingly, the differences were no longer apparent at the second NCS. The sural nerve was also more affected in A-CIDP when compared to GBS-TRF, with smaller amplitudes and slower CV.

We did not find the sural-sparing pattern or sensory ratio useful in distinguishing GBS-TRF from A-CIDP. The sural-sparing pattern has been reported to be indicative of AIDP and has been demonstrated in up to two thirds of GBS patients in the first week of illness.[14,22] Whether the sural-sparing pattern is more apparent at the earlier vs later stages of GBS is debatable.[23-25] Our findings were in keeping with previous studies which also found the sural-sparing pattern and sensory ratio not useful in differentiating AIDP from A-CIDP.[7,8]

Nerve ultrasound has been increasingly utilised in peripheral nerve disorders including inflammatory neuropathies.[8,9,20] In the current study, we found that the A-CIDP patients had larger ulnar nerve compared to GBS-TRF. These findings reflect the ulnar NCS findings that suggest more prolonged minimal F-wave latencies in A-CIDP patients. Our findings are in keeping with previous reports of ultrasound parameters of typical CIDP patients which demonstrated more diffuse, larger average nerve size index compared to typical GBS patients.[26,27] Nerve enlargement in CIDP patients tend to be more pronounced at proximal and non-entrapment sites[27,28], and the enlargement is homogenous in chronic CIDP patients.[30] In the current study, we also investigated the value of the ultrasound score, UPSA, a sub-score from Ultrasound Pattern Sum Score (UPSS).[9] We found a higher UPSA scores in our A-CIDP patients (all scoring ≥ 7), whereas our GBS-TRF patients had scores < 7. This was consistent with the report suggesting a UPSA score of ≥ 7 was able to differentiate CIDP from other neuropathies (including GBS) with good sensitivity, specificity and positive predictive value.[9] One reason for the increased nerve size in A-CIDP and GBS-TRF could be attributed to the underlying pathogenesis of both conditions. In the latter, the disease is typically monophasic with improvement over time whereas in CIDP, patients tend to have a relapsing and remitting course suggesting an ongoing immune-mediated process. It should be noted that the timing of the ultrasound was different. Ultrasound was performed at the first clinical deterioration which was later in A-CIDP patients compared to the GBS-TRF cohort. However, we have previously reported on nerve enlargement in the progressive phase of GBS which subsequently shows a reduction in size after 3 weeks.[17] In contrast, nerve enlargement in CIDP typically persists for up to 6 months.[20]

There were several limitations in the current study. Due to the rarity of both conditions[3-5], the number of cases in each group was small. As such, the statistical significance in our study has to be interpreted with caution. However, in this exploratory study, the demonstration of significant differences in certain nerve electrophysiological and ultrasound parameters merit further prospective study in a larger cohort. The study was also limited by the retrospective nature which resulted in differences in the timing of the nerve ultrasound. Our A-CIDP cohort was older and had an unusually severe clinical pattern with two patients requiring mechanical ventilation. Both characteristics may have had an impact on the electrophysiological and ultrasound results. Previous studies have shown that older age is associated with increased nerve size on ultrasound and slower CV on NCS.[30,31]

In conclusion, differentiating between A-CIDP and GBS-TRF is important but can be challenging. The early recognition of both conditions not only allows determination of disease prognosis but also the approach to treatment. In the current study, we found that a slower nerve parameters on NCS and larger nerve size on ultrasound can be helpful in distinguishing A-CIDP from GBS-TRF.

DISCLOSURE

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Conflict of interest: None

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