Guillain-Barré Syndrome in Egypt: Diagnostic Challenges and Subtype Evolution Over Time

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Abstract: *Background*: Guillain-Barré Syndrome (GBS) is the leading cause of acute flaccid paralysis worldwide, with diverse clinical and electrophysiological presentations. Regional variation in GBS subtypes exists, but limited data are available from Arab countries, particularly Egypt.

Objective: This study aimed to characterize clinical features, neurophysiological patterns, and disability outcomes among Egyptian GBS patients and evaluate changes in electrophysiological subtypes over time.

Methods: A prospective observational study was conducted on 49 adult patients diagnosed with GBS and admitted to Mansoura University Hospitals between May 2022 and May 2025. Clinical data, disability scores (Hughes, INCAT, mEGOS, MRC), and electrophysiological findings were collected within the first two weeks of symptom onset and repeated before discharge. Electrophysiological subtyping was performed using Rajabally's criteria at initial and repeat testing.

Results: The cohort had a median age of 50 years; 59.2% were male. Upper respiratory tract infections were the most common preceding illness (55.1%). Sensorimotor deficits were the predominant presentation (89.8%), with cranial nerve involvement observed in 28.6% and autonomic dysfunction in 20.4%. Most patients received plasma exchange, and 22.4% required additional immunotherapy. Initial electrophysiological studies were inconclusive in 37.4%, but follow-up improved diagnostic yield to 93.7%. Electrophysiological reclassification occurred in 54.1%, with cases shifting between axonal and demyelinating patterns. At follow-up, AIDP was slightly more prevalent (52%) than axonal forms (41.6%). Functional scores (Hughes, INCAT, MRC) improved significantly within one month, with >65% achieving favorable outcomes.

Conclusion: Serial neurophysiological assessment enhances diagnostic accuracy in early GBS subtypes classification, with substantial shifts occurring between initial and follow-up studies. A high proportion of Egyptian GBS patients presented with severe disability but showed marked improvement by the fourth week.

Keywords: Guillain-Barré Syndrome, GBS subtypes, Egypt, AIDP, AMAN, Electrophysiology, Autoimmune neuropathy, Disability outcome, Nerve conduction studies, Regional variation, Prognosis.

INTRODUCTION

Guillain-Barré syndrome (GBS) is a potentially devastating yet treatable disorder. It is classically characterized as a post-infectious, immune-mediated, monophasic polyradiculoneuropathy and represents the most common cause of acute neuromuscular paralysis worldwide. It can be a severe and life-threatening condition, and early treatment is essential for a better prognosis [1]. It accounts for an estimated 100,000 new cases annually worldwide, and it increases with age [2]. It is an autoimmune response triggered by prior infection or immune stimulation, causing the immune system to attack peripheral nerve myelin or axons via molecular mimicry [3].

Clinically, it is characterized by acute flaccid paralysis and/or sensory/autonomic nerve dysfunction that typically develops within 4 weeks after infection [1]. Electro physiologically, GBS is classified into demyelinating and axonal types based on nerve conduction studies. Pathologically, it may involve primary demyelination, axonal degeneration, or a combination of both, reflecting the diverse mechanisms underlying its presentation and progression [4]. In the absence of sufficiently sensitive and specific disease biomarkers, its diagnosis is largely based on clinical patterns with or without the support of laboratory findings and electrophysiology [1]. The electrophysiological criteria for its diagnosis have been continuously evolving, enhancing the precision of subtyping in the recognized illness. This is not the case of very early GBS, where initial nerve conduction studies (NCS) allow subtyping in just 20% of cases [5].

Although GBS is typically self-limiting, severe cases can lead to lasting disability or death [6]. Around one-third develop generalized neuropathy requiring mechanical ventilation, and mortality occurs in about 5% of cases [7]. Research indicates that the distribution and phenotypes of GBS subtypes vary

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geographically [3]. Within the Arab world, findings also differ between its countries, even neighboring ones [8, 9].

This prospective observational study aimed to characterize the clinical features and electrophysiological subtypes of GBS in Egyptian adults, assess short-term functional outcomes, and evaluate the accuracy of early electrophysiological classification within the first two weeks of onset

METHOD

We included 49 patients who fulfilled the clinical criteria made by the GBS classification group by Fokke *et al*., [10] and admitted to Mansoura University Hospitals in time between 2022 to 2025 were assessed clinically at onset by asking about clinical data as sex, age, with the preceding symptoms, presenting complaints, cranial nerves involvement, associated comorbidities, autonomic dysfunction, received treatment, and we assessed the median time to admission after symptoms onset, median time to received treatment. Neurological and functional assessments were performed using multiple scales: the Hughes Functional Grading Scale (0–6), to assess recovery based on initial and one month follow up assessment [11]; the Modified Erasmus GBS Outcome Score (mEGOS) for predicting disability at 1, 3, and 6 months [12]; the INCAT scale (0–10) assessing upper and lower limb function [13]; and the EGRIS, which predicts respiratory failure risk using early clinical variables [14].

Electrophysiological studies have been performed at the Clinical Neurophysiology Unit of Mansoura University hospitals, using (NIHON KOHDEN) © MODEL MEB-9400K, Serial number SNI-00833. Conduction studies were performed on the clinically most affected side, with skin temperature maintained at 34°C, and all NCS were performed by a single electrophysiologist to reduce variability. Standard motor and sensory NCS were conducted on the median, ulnar, tibial, and peroneal nerves, assessing MCV, DML, CMAP, F-wave latency, and conduction block (CB) using Clouston's criteria. F-waves were considered Impersistant if <80% were elicited and not applicable (NA) if CMAP <1 mV [15] Sensory studies (antidromic) were performed on the median, ulnar, and superficial peroneal nerves, analyzing SNAP amplitude and SCV. GBS subtypes were classified using Rajabally's criteria [16]. The second electrophysiological study was considered the most reliable for defining underlying pathology. We assessed clinical characteristics, outcome scores among Egyptian GBS cases with their electrophysiological subtyping.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics. Categorical variables were summarized as frequencies and percentages, while continuous variables were described using medians and interquartile ranges (Q1–Q3) due to non-normal distribution, which was assessed using the Shapiro-Wilk test. Comparisons between categorical variables (e.g., subtype distribution, gender differences) were made using the Chi-square test or Fisher's exact test when expected counts were small. For continuous variables, the Mann-Whitney U test was used for comparing two independent groups, and the Wilcoxon signed-rank test was applied for paired comparisons (e.g., disability scores at admission and discharge). A p-value < 0.05 was considered statistically significant.

RESULTS

Of the 51 cases examined, two were excluded due to a diagnosis of Miller Fisher Syndrome (MFS), leaving 49 patients for analysis. Electrophysiological Classification was performed in 48 patients after the second nerve conduction study, as one case died. Demyelinating forms were most frequent (n=25, 52%), followed by axonal forms (n=20, 41.6%), with only two patients (4.2%) remaining inexcitable and one (2.1%) classified as equivocal. Within the axonal subgroup, 16 patients exhibited no reversible conduction failure (nRCF) and four demonstrated reversible conduction failure (RCF). In the demyelinating subgroup, 11 patients had nRCF and 14 had RCF (Figure **1**).

This study included 49 GBS patients with a median age of 50 years (IQR 35–62.5), with a male predominance (59.2%). URTI was the most frequent antecedent illness (55.1%), followed by gastrointestinal symptoms (26.5%), while 18.4% reported no preceding illness. The median onset-to-admission interval was 7 days (IQR 4.5–8), and onset-to-treatment interval was 9 days (IQR 7–11.5). Median hospital stay was 22 days (IQR 19–33). These findings reflect relatively delayed hospital presentation and treatment initiation in the cohort (Table **1**).

At baseline, disability was substantial:

- **Hughes Disability Score (HDS):** 4.0 (IQR 3.5–4.0)

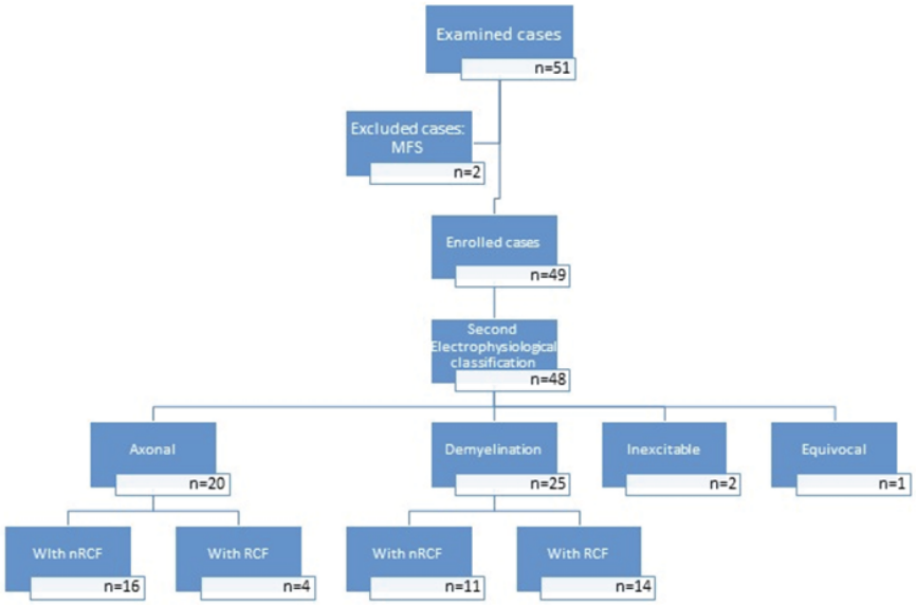

Figure 1: Flow diagram for patient recruitment (CONSORT chart).

Table 1: Characteristics of the Studied Cases (n=49)

Characteristic	N	%
Sex		
Male	29	59.2%
Female	20	40.8%
Preceding symptoms		
GIT symptoms	13	26.5%
URT symptoms	27	55.1%
No preceding symptoms	9	18.4%
Presenting symptoms		
Motor	5	10.2%
Sensorimotor	44	89.8%
Sensory only	0	0%
Cranial nerve involvement	14	28.6
Unilateral facial nerve palsy	3	6.1%
Bilateral facial palsy	3	6.1%
Bulbar palsy	3	6.1%
Facial and bulbar palsy	4	8.2%
Facial and vestibular n. palsy	1	2%
Dysautonomia	10	20.4%
BP variability	3	6.1%
HR variability	3	6.1%
More than one autonomic symptom	4	8.2%
Complication		
Thromboembolic complication	2	4.1%
Mechanical ventilation needs	5	10.2%
Need for another immunotherapy	11	22.4%
	Median	Q1-3
Age (years)	50	35-62.5
Onset to admission interval (days)	7	4.5-8
Onset to treatment interval (days)	9	7-11.5
Length of hospital stay (days)	22	19-33

GIT: gastrointestinal, URT: upper respiratory tract, BP: Blood pressure, HR: Heart rate.

Table 2: Shows the Median Scores at Admission and Discharge

Characteristic	Median	Q1-3
Initial INCAT	8	6-9
EGRIS	3	2-3.5
Initial HUGHES	4	3-4
Initial mEGOS	6	3-8
Initial MRC	36.5	33-43
4th HUGHES	2	1-2.75
4th INCAT	4	2-5.75
4^{th} MRC	48	42-52

INCAT: Inflammatory Neuropathy Cause and Treatment, EGRIS: Erasmus Guillain-Barré syndrome Respiratory Insufficiency Score, mEGOS: Modified Erasmus GBS Outcome Score, MRC:Medical Research Council.

- **INCAT:** 8.0 (IQR 6.0–9.0)
- **mEGOS:** 6.0 (IQR 3.0–8.0)
- **MRC Sum Score:** 36.5 (IQR 33.0–43.0)

By the fourth week, marked improvements were seen:

- **HDS**: decreased to 2.0 (IQR 1.0–2.75), median difference –2.0 points (95% CI: –2.5 to –1.5; $p < 0.001$)
- **INCAT**: improved to 4.0 (IQR 2.0–5.75), median difference –4.0 points (95% CI: –4.5 to –3.0; $p < 0.001$)
- **MRC sum score**: increased to 48.0 (IQR 42.0–52.0), median difference +11.5 points (95% CI: +9.0 to +14.0; $p < 0.001$)

These changes indicate significant functional recovery over the first month (Table **2**).

At admission, 71% of patients had severe disability (HDS ≥4), including 8.2% who were ventilator-dependent (HDS 5). By discharge, the proportion with severe disability fell to 20%, an absolute reduction of –51% (95% CI: –64% to –38%; $p < 0.001$). The proportion achieving minimal or no disability (HDS 0–1) rose from 2.0% to 32.6% (absolute increase +30.6%, 95% CI: +18.0% to +43.2%; $p < 0.001$) (Table **3**).

Table 3: Shows the Distribution of Cases Initially and at Discharge according to HDS

	Hughes Score	Count	Percentage
Initial HDS (N:49)	0	1	2.0%
	2	5	10.2%
	3	8	16.3%
	4	30	61.2%
	5	4	8.2%
	6	1	2.0%
	Hughes Score	**Count**	**Percentage**
At discharge mean 1 month HDS (N:49)	0	6	12.24%
	1	10	20.4%
	2	16	32.6 %
	3	7	14.28 %
	4	5	10.2 %
	5	4	8.3%
	6	1	2.04%

HDS: Hughes Disability Score.

Table 4: Shows the Median of Initial Electrophysiological Parameters among the Studied Cases

Characteristics	Total Cases
1st motor CV	
median	45 (35-53)
Ulnar	45 (33-51.5)
Tibial	33 (0-44.5)
Peroneal	37 (0-49)
1st CMAP	
median	3.5 (1.515-5.45)
Ulnar	3.5 (1.28-5.785)
Tibial	.51 (0-3.9)
Peroneal	.45 (0-1.35)
1st motor Latency	
median	5.7 (3.6-9)
Ulnar	4 (3-5.5)
Tibial	5 (0-7.95)
Peroneal	4 (0-5.7)
1st sensory CV	
median	30 (0-52)
Ulnar	47 (0-52)
Sup peroneal	0 (0-36.5)
1st SNAP	
median	5 (0-14.5)
Ulnar	5 (0-18)
Superficial peroneal	0 (0-3.95)
1st Peak sens Lat.	
median	2.2 (0-3.35)
Ulnar	2.6 (0-3.25)
Sup peroneal	0 (0-4)

1st: first, CV: conduction velocity, CMAP: compound motor action potential, Sup.: superficial, sens.: Sensory, Lat.: latency.

Motor conduction velocities were most reduced in the tibial (median 33 m/s) and peroneal (median 37 m/s) nerves. CMAP amplitudes were lowest in the tibial (0.51 mV) and peroneal (0.45 mV) nerves. Sensory conduction was most impaired in the superficial peroneal nerve, with also absent response reported from the upper limb sensory nerves. These findings are consistent with severe mixed sensorimotor involvement mainly in the lower limbs at baseline (Table **4**).

Initially, inexcitable nerves were present in 4%-26.5% of motor nerves, with the highest percentage in lower limbs, and 16.3–32.5% of sensory nerves. On repeat studies, rates increased to 32.6% (peroneal motor) and 44.8% (superficial peroneal sensory), representing absolute increases of +12.2% (95% CI: +2.4% to +22.0%; p = 0.02) and +12.3% (95% CI: +3.1% to +21.5%; p = 0.01), respectively. Conduction block was most frequent in peroneal and upper limb motor studies Reported in 8,6, and 6 cases, respectively. F-wave abnormalities were reported in 65%-75.5% of cases initially (Table **5**).

At baseline, 37.4% of patients had inconclusive subtyping. Following repeat NCS, classification was possible in 93.7% of cases (median yield difference +56.3%, 95% CI: +41.2% to +71.4%; p < 0.001). Demyelinating (AIDP) forms increased from 29.1% to 52.0% (absolute change +22.9%, 95% CI: +8.4% to +37.4%; p = 0.003), while axonal (AMAN/AMSAN) forms accounted for 41.6% at follow-up (Table **6**).

Subtype shifts between baseline and follow-up were frequent. Four patients with inexcitable nerves initially remained inexcitable (n=2) or converted to demyelinating (n=2). Three initially axonal cases were reclassified as demyelinating with reversible conduction failure (RCF). Of 14 initially equivocal cases, 13 (92.8%) were definitively classified, most commonly as axonal without reversible conduction failure (nRCF) or

Table 5: Motor and Sensory Nerves Electrophysiological Characteristics

Nerve	Motor				Sensory		
	Median	Ulnar	Tibial	peroneal	Median	Ulnar	Sup peroneal
Inexcitable nerves							
Initial study	2 (4%)	-	10 (20.4%)	13 (26.5%)	11(22.4%)	8 (16.3%)	16 (32.5%)
Second study	2 (4%)		14(28.5%)	16(32.6%)	16(32.5%)	13(26.5%)	22(44.8%)
CB	6	6	1	8	-	-	-
F wave initial response							
Normal	15	17	12				
Absent	11	10	5				
Impersistant	2	4	3				
Prolonged	15	13	6				
NA	2	1	19				

CB: Conduction Block; NA: Not Applicable.

Table 6: Shows the Neurophysiological Subtyping among the Studied Cases Initially and at Discharge

Electrophysiological classification (n=48)	N (%)
Initial Electrophysiological Classification	
Normal	2 (4.1%)
Demyelination	6 (12.5%)
Demyelination +CB	8 (16.6%)
Axonal	11(22.9%)
Axonal +CB	3 (6.25%)
Inexcitable	4 (8.3%)
Equivocal	14(29.1%)
The second Electrophysiological Classification	
Demyelination nRCF	14 (29.16%)
Demyelination RCF	11 (22.9%)
Axonal nRCF	16 (33.3%)
Axonal RCF	4 (8.3 %)
Inexcitable	2 (4.1%)
Equivocal	1 (2 %)

CB: conduction block, RCF: reversible conduction failure, nRCF: non-reversible conduction failure.

demyelinating with RCF. These changes highlight the importance of repeat NCS after the first two weeks (Table **7**).

Serial NCS showed selective but significant changes over time. Motor conduction velocity (CV) in the tibial nerve increased from a median of 33.0 m/s (IQR 0–44.5) to 39.5 m/s (IQR 28.5–46.7), a median difference of +6.5 m/s (95% CI: +2.1 to +10.9; p = 0.005). CMAP also showed trends towards increased, although not significant specially in lower limb nerves, indicating transient CB with reversibility. Distal motor latency (DML) in the peroneal nerve increased from 4.0 ms (IQR 0–5.7) to 5.0 ms (IQR 3–8), median difference +1.0 ms (95% CI: +0.3 to +1.7; p = 0.004), indicating more demyelinating features in the second study, that were not apparent in the initial one. Regards sensory studies, more prominent demyelinating features appeared in CV, supporting the delay in sensory findings compared with motor nerves (Table **8**).

Compared to demyelinating cases, axonal cases had significantly longer hospital stays (median difference +12.5 days, 95% CI: +4.3 to +20.7; p = 0.006) and were more likely to present with pure motor symptoms (+25%, 95% CI: +8% to +42%; p = 0.013). Recovery was slower in axonal cases, with a 44% higher proportion experiencing slow recovery (95% CI: +18% to +70%; p = 0.004). At 4 weeks, axonal cases had higher INCAT scores (median difference +0.5, 95% CI: +0.1 to +0.9; p = 0.019) and higher Hughes scores (median difference +1.0, 95% CI: +0.4 to +1.6; p = 0.003). No significant differences were found in

Table 7: Shifting between Classifications in the 1st and 2nd Electrophysiological Examination

		Initial electrophysiological classification							TOTAL
		Normal	Demyelination	Demyelination with CB/CF	Axonal	Axonal with CB/CF	In-excitable	Equivocal	
The 2nd Electro-physiological Classification	Demyelination with nRCF	0	6	3	0	0	0	2	11
	Demyelination with RCF	0	0	5	3	0	2	4	14
	Axonal with nRCF	2	0	0	8	0	0	6	16
	Axonal with RCF	0	0	0	0	3	0	1	4
	Inexcitable	0	0	0	0	0	2	0	2
	Equivocal	0	0	0	0	0	0	1	1
Total		2	6	8	11	3	4	14	48

CB: conduction block, CF: conduction failure, RCF: reversible conduction failure.

Table 8: Comparison between the Initial and Second Electrophysiological Parameters

Characteristics	Initial	Second	Sig
motor CV			
median	45 (35-53)	44 (33-56)	.869
Ulnar	45 (33-51.5)	45.5 (32.2-50)	.980
Tibial	33 (0-44.5)	39.5 (28.5-46.7)	.005
Peroneal	37 (0-49)	35.5 (29.2-45)	.270
CMAP			
median	3.5 (1.515-5.45)	3.9 (1.6-4.8)	.250
Ulnar	3.5 (1.28-5.785)	3.4 (2.17-4.9)	.650
Tibial	.51 (0-3.9)	1.15 (.2-2.175)	.681
Peroneal	.45 (0-1.35)	.95 (.127-1.1)	.895
motor Latency			
median	5.7 (3.6-9)	6 (4-9)	.657
Ulnar	4 (3-5.5)	4.2 (3.2-6)	.049
Tibial	5 (0-7.95)	5.8 (3.4-9.6)	.106
Peroneal	4 (0-5.7)	5 (3-8)	.004
sensory CV			
median	30 (0-52)	22(0-49.25)	.493
Ulnar	47 (0-52)	34 (0-50)	.414
SNAP			
median	5 (0-14.5)	4.5 (0-14.75)	.526
Ulnar	5 (0-18)	5.55 (0-16)	.903
Superficial peroneal	0 (0-3.95)	0 (0-4)	
Peak sens Lat.			
median	2.2 (0-3.35)	2 (0-3.4)	.647
Ulnar	2.6 (0-3.25)	2.9 (0-4)	.429

1st: first, CV: conduction velocity, CMAP: compound motor action potential, Sup.: superficial, sens.: Sensory, Lat.: latency, EDC: extensor digitorum communis, MCD: mean consecutive difference, T.A: tibialis anterior.

age, sex, comorbidities, cranial nerve involvement, or need for repeat immunotherapy (Table **9**).

A 4th-week Hughes score ≤2 predicted rapid recovery with 100% sensitivity and 75% specificity (AUC 0.895, 95% CI: 0.772–0.965; $p < 0.001$). A hospital stay ≤26 days predicted rapid recovery with 90.6% sensitivity and 81.25% specificity (AUC 0.841, 95% CI: 0.707–0.930; $p = 0.001$). Initial Tibial

Table 9: Comparisons of Axonal vs. Demyelinating Cases

Characteristic	Subtyping		Sig.
	Axonal (20)	Demyelination (25)	
Sex			.142
Male /Female	9 /11	17/8	
Age (years)	48 (30.5-64.5)	49 (35-56.5)	.607
Onset to admission interval (days)	6 (4-7)	7 (5.5-10)	.097
Onset to treatment interval (days)	9 (6.25-9.75)	9 (7-12)	.114
Length of hospital stay (days)	33 (20.5-35)	20 (19-23.5)	.006
Preceding symptoms			.696
No	2 (10%)	4 (16%)	
GIT	7 (35%)	6 (24 %)	
URTI	11 (55%)	15 (60%)	
Presenting symptoms			.013
Motor	5 (25%)	0	
Sensorimotor	15 (75%)	25 (100%)	
Dysautonomia	4 (20%)	5 (17.9%)	.648
Cranial nerve involvement	4 (20%)	9 (36%)	.887
Comorbidity	3 (15%)	4 (14.3%)	1
Thromboembolic complications	1 (5%)	1 (4%)	.697
Probability of respiratory insufficiency within the first week of admission by EGRIS (%)	15 (5-32)	5 (5-15)	.128
MV requirement	3 (15%)	1 (4%)	.309
Need for another immunotherapy	7 (35%)	3 (12%)	.083
Recovery			.004
Rapid	8 (40%)	21 (84%)	
Slow	12 (60%)	4 (16%)	
Changed NCS classification	9 (45%)	13 (46.4%)	.9
Initial MRC category	36 (32.25-42.5)	39 (33.5-44)	.657
Mild (48-60)	0	2 (7.1%)	
Moderate (36-47)	10 (50%)	17 (60.7%)	
Severe (0-35)	10 (50%)	9 (32.1%)	
Initial mEGOS	6.5 (3.2-8)	5 (3-7.5)	.394
Initial INCAT	9 (6.5-9)	7 (6-9)	.113
Initial HUGHES	4	4 (3-4)	.06
EGRIS	3 (2-4)	2 (2-3)	.132
4th wk INCAT	4.5 (2.25-6.75)	4 (1-4)	.019
4th w HUGHES	2 (2-3)	1 (1-2)	.003
Predicted probability of being unable to walk unaided after 4 weeks (%)	67.5% (28%-82%)	51% (25%-77%)	.394
Predicted probability of being unable to walk unaided after 3 months (%)	35% (21%-60%)	21% (15%-55%)	.231
Probability of respiratory insufficiency within the first week of admission (%)	15% (5%-32%)	5% (5%-15.5%)	.178

GIT: gastrointestinal, URT: upper respiratory tract, INCAT: Inflammatory Neuropathy Cause and Treatment, EGRIS: Erasmus Guillain-Barré syndrome Respiratory Insufficiency Score, mEGOS: Modified Erasmus GBS Outcome Score, MRC:Medical Research Council, MV: mechanical ventilation, NCS: nerve conduction study.

Table 10: Diagnostic Performance of the Studied Clinical and Neurophysiological Parameters for Rapid Recovery Prediction

Predictor	Cutoff	AUC	95% CI of AUC	p-value	SE	Sensitivity	Specificity
4th w HUGHES	≤2	0.895	0.772 - 0.965	<.001	0.0557	100%	75%
4th INCAT	≤4	0.667	0.516 - 0.796	.061	0.0890	78.12 %	62.5%
Initial peroneal nerve CMAP (mV) reflecting CB	≤0.22	0.682	0.531 - 0.809	0.0252	0.0811	50%	81.25%
Length of hospital-stay (days)	≤26	0.841	0.707 - 0.930	.001	0.0697	90.62%	81.25%
Initial ulnar nerve motor CMAP (mV) reflecting CB	≤3.5	0.701	0.552 - 0.825	.008	0.0759	62.5%	75%
Initial Tibial nerve CV (m/s)	≤22	0.723	0.575 - 0.842	.003	0.0760	50%	93.75%

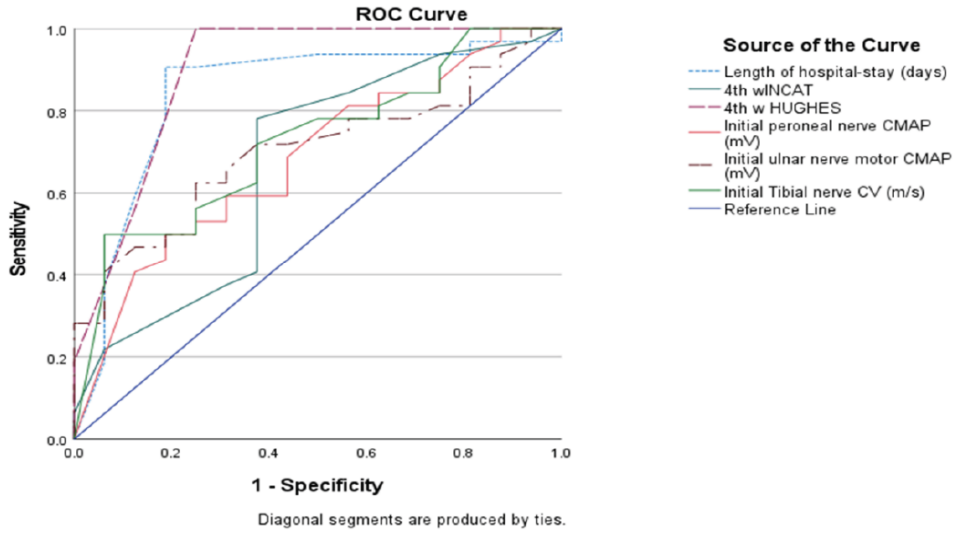

Figure 2: ROC curves for Clinical and Electrophysiological cutoff values in predicting rapid recovery in GBS.

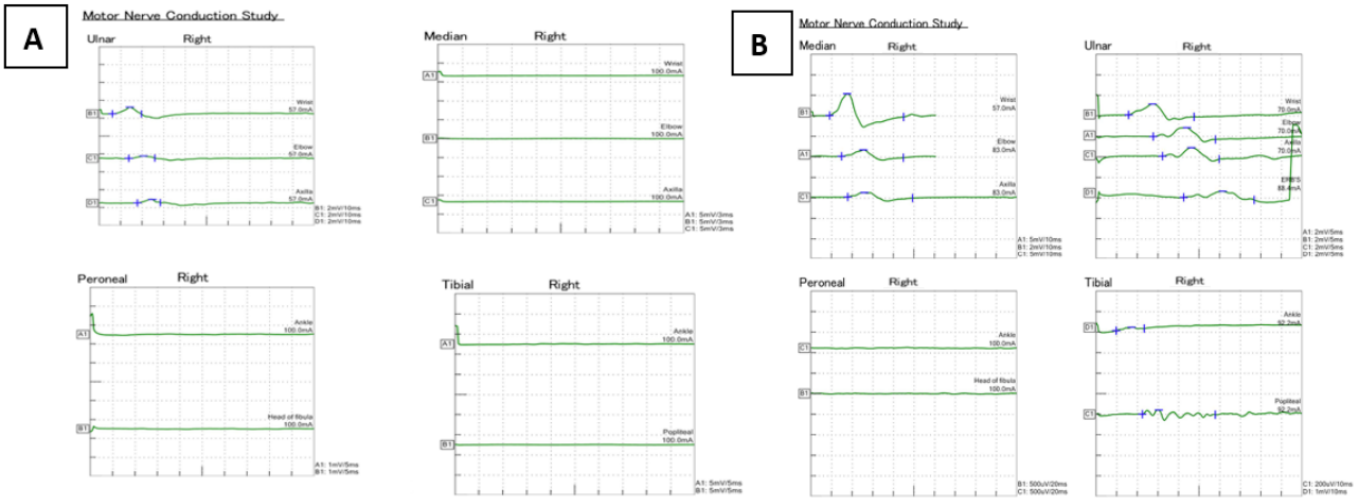

Figure 3: A, an initial electrophysiological study that shows the absence of all nerve responses, with only low ulnar CMAP, and difficult electrophysiological subtyping. **B**, the second study for the same case, which shows RCF in median and tibial nerves with signs of demyelination as temporal dispersion at tibial nerve, CB at median and ulnar nerves, prolonged distal latency of median and Tibial nerves.

Table 11: Univariate Logistic Regression with all Previous Significant Parameters for Rapid Recovery

Recovery prediction parameter	SE	Sig.	COR	95% C.I. for odds ratio	
				Lower	Upper
Non Axonal cases (2nd classification)	.707	.002	9	2.25	35.98
Presenting symptoms sensori motor	1.169	.046	10.33	1.046	102.08
No Need for other immunotherapy	.789	.011	7.519	1.603	35.26
Length of hospital stay ≤ 26	.882	<.001	41.88	7.435	236.010

Coefficients						
Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	SE	Beta		
1	(Constant)	3.969	0.812		4.886	0.000
	Age (years)	0.002	0.008	0.024	0.202	0.841
	Onset to admission interval (days)	0.127	0.116	0.339	1.089	0.282
	Onset to treatment interval (days)	-0.095	0.120	-0.249	-0.786	0.436
	Length of hospital-stay (days)	-0.081	0.016	-0.624	-5.070	0.000
a. Dependent Variable: HUGHES.diff						

CV ≤22 m/s had 93.75% specificity but only 50% sensitivity (AUC 0.723, 95% CI: 0.575 - 0.842; p = 0.003). These findings highlight the prognostic value of combining early functional scores with electrophysiological markers). Notably, low initial CMAP in peroneal, and ulnar nerves were associated with rapid recovery, supporting transient CB that associated with initial low CMAP, and later on good recovery state (Table **10**), Figure **2**.

Non-axonal classification at follow-up was associated with a 9-fold higher odds of rapid recovery (95% CI: 2.25–35.98; *p* = 0.002). Sensorimotor presentation predicted rapid recovery with an OR of 10.33 (95% CI: 1.046–102.08; *p* = 0.046). Patients who did not require additional immunotherapy were 7.52 times more likely to recover rapidly (95% CI: 1.603–35.26; *p* = 0.011). Hospital stay ≤26 days had the strongest association with rapid recovery (OR 41.88, 95% CI: 7.44–236.01; *p* < 0.001) (Table **11**).

DISCUSSION

GBS epidemiology varies across regions and countries. Two incidence peaks were reported globally, at ages 5–9 and 60–64 [3]. In our Egyptian cohort, most patients were male (59.2%), with a median age of 50 years (IQR 35–62.5), consistent with global data showing a higher GBS incidence in males and with advancing age [17]. The slight rise in age-standardized prevalence may reflect improved survival due to better care and earlier diagnosis. Conversely, studies from low- and middle-income countries where Campylobacter is endemic, such as Egypt, report a decline in GBS incidence with age, as infections are more common in children and decrease over time [18]. Similarly, some reports from Arab populations suggest GBS is more frequent in individuals in their twenties and thirties, with no significant sex difference in incidence [8, 19]. These age-related patterns highlight the complex interaction between infectious exposures, immune response, and neurological vulnerability across different populations.

In terms of antecedent events, our cohort showed that 81% of patients reported a preceding illness—most commonly RTI, 55.1%, followed by GIT symptoms 26.5%, while 18.4% had no identifiable trigger. This aligns with findings from the Middle East and North Africa, where RTIs are more prevalent (e.g., 24% vs. 8% in Egypt, and 51% vs. 32% in Morocco) [20]. This predominance of RTIs may reflect regional epidemiological patterns of circulating pathogens, seasonal factors, or healthcare-seeking behaviors. Interestingly, C. jejuni–related GIT symptoms were less frequent in our cohort (26.5%), which is in contrast to studies from South Asia, such as Bangladesh, where GIT illness is the leading antecedent [22]. Such differences emphasize the influence of regional dietary habits, sanitation standards, and pathogen prevalence on GBS triggers.

Moreover, age appears to be an additional determinant: previous studies reported higher RTI rates in older adults and more frequent C. jejuni–related GIT symptoms in younger patients [21]. This may suggest age-related variations in exposure and immune response, potentially mediated by diet and comorbidities. In our study, 18.4% of patients had no identifiable antecedent, which may reflect subclinical infections, noninfectious immune triggers, or limitations in recall.

In our cohort, combined sensorimotor symptoms were predominant (89.8%), while pure motor symptoms occurred in 10.2%; no pure sensory cases were observed. Cranial nerve involvement was seen in 28.6%, most frequently combined facial and bulbar palsy (8.2%). Autonomic dysfunction occurred in 20.4% of ICU-admitted patients, often presenting with multiple symptoms. Thromboembolic events were reported in 4.1%, 10% required mechanical ventilation, and one death (1%) occurred due to aspiration pneumonia.

These findings align with a Lebanese study reporting 77% sensorimotor involvement and 25% cranial neuropathies [23], and a systematic review on Arab populations where facial weakness was the most common cranial nerve finding, and autonomic dysfunction occurred in 4–17% of cases. Conversely, mortality rates in LMICs were higher—up to 16% [21, 24]. Our lower mortality rate indicates a relatively favorable outcome. While overall GBS mortality is 3–7%, it may reach 20% in ventilated patients due to ARDS, sepsis, PE, or cardiac arrest [25]. A Jordanian study reported high ICU admission (80%) and intubation (75%) rates, though its small sample size (12 patients) limits generalizability [26]. Despite prolonged hospital stays and transient complications, GBS outcomes across Arab countries, including Saudi Arabia, remain generally favorable [27].

While most patients received a single course of treatment, which was mainly plasma exchange, being the most available, affordable, a total of 11 patients (22.4%) required escalation to a second immuno-therapy or extended sessions of plasma exchange more than five. This goes with what is reported: 25% may require multiple treatments, particularly those with severe GBS [28]. Rescue treatment with a second course of IVIG or PLEX in poorly responsive patients has shown short-term benefits, although long-term outcomes remain unclear [29].

In our cohort, the median interval from symptom onset to hospital admission was 7 days, reflecting some delay in seeking medical care, with treatment typically initiated by day 9. The median duration of hospital stay was 22 days. Approximately 65.3% of patients achieved a favorable prognosis, while 32.6% experienced poor outcomes. Notably, around 71% of patients presented with a Hughes Disability Score (HDS) >3 at admission, which decreased to 20% after one month of follow-up.

Several studies have highlighted the variability in GBS prognosis depending on population characteristics and follow-up duration. In our cohort, 69% of patients presented with severe disability, ventilation dependence, or required ICU admission, while mortality was low (2%). At one month after onset, 32.6% had a poor prognosis, while 65.3% showed favorable outcomes; approximately 20% had a Hughes Disability Score (HDS) >3. These findings align with regional data from Egypt and the Middle East, where 76% of patients had an HDS >2 at admission [21, 24]. Similarly, a pediatric cohort study reported 25% with poor prognosis at one month, while 75% showed early improvement [30]. In Beghi *et al*. [31], clinical improvement began within the first week in 36% and within four weeks in 85% of cases, with mean times to nadir, initial improvement, and full recovery of 12, 28, and 200 days, respectively. A Middle Eastern study by Dahbour *et al*. [26] involving 12 adult patients reported a median time to independent ambulation of 62 days, with overall favorable outcomes despite some complications. Long-term outcomes vary; some studies report 92% functional independence within 3 months, while others document persistent disability in up to 39% at 11 months and 7% with severe limitations at one year [20]. Mortality rates ranged from 0–8%, reflecting differences in disease severity and healthcare access. In another study from Egypt, over half of the patients had unfavorable outcomes at discharge, emphasizing the need for early intervention and optimized management to improve recovery [23].

In our cohort, the demyelinating subtype (AIDP) was slightly more common, accounting for 52% of cases compared to 41.6% for the axonal subtype (AMAN). Although the difference was not large, this distribution may be influenced by the fact that Egypt is considered an endemic region for C. jejuni, a pathogen more frequently associated with axonal forms of GBS. This goes with [21, 24], who focused on the Middle East population; AIDP vs AMAN is more prevalent (76% vs 8% in Egypt and 81% vs 19% in Morocco). In harmony with studies that explored the GBS characteristics in a Lebanese tertiary care center over 12 years, and North

America and Europe, finding AIDP to be the most prevalent variant [23, 32].

In contrast, the axonal subtype (AMAN) has been reported as more prevalent in other regions, accounting for 30–65% of GBS cases in parts of Asia, Central America, and South America [33]. Interestingly, a recent study from Saudi Arabia, a neighboring country, found AMAN to be the most common subtype, highlighting the heterogeneity in subtype distribution even within the same broader region [9].

The predominance of the demyelinating subtype (AIDP) in our cohort is noteworthy, particularly given Egypt's endemicity for *Campylobacter jejuni*—a pathogen strongly associated with axonal variants. This suggests that factors beyond the distribution of antecedent infections may play a pivotal role in determining GBS subtype expression. Host genetic background has been increasingly recognized as a key determinant: population-specific variation in HLA alleles and immune response genes can modulate susceptibility to particular subtypes. For instance, HLA-DQB1 and HLA-DRB1 alleles have been linked to an increased risk of axonal forms in East Asian cohorts, whereas European and North African populations tend to exhibit genetic patterns more often associated with demyelination [3].

In Arab populations, although data are limited, emerging evidence points toward distinctive immune response profiles shaped by shared ancestry, environmental exposures, and endemic infectious triggers. This may help explain why our Egyptian cohort showed a distribution pattern more comparable to Western and some North African studies, where AIDP predominates, rather than to Asian regions where AMAN accounts for up to 65% of cases. Such findings highlight the need for deeper exploration of host–pathogen–genetic interactions within the Egyptian and broader Arab context. Multicenter studies integrating genomic data with clinical and electrophysiological phenotyping would be instrumental in clarifying the interplay between genetic predisposition, environmental factors, and infectious triggers in shaping GBS subtype expression [6].

Interestingly, Sensory nerves were more often unexcitable, especially in studies conducted after two weeks. this goes with what reported by Yadegari *et al*., [34]. This may be attributed to the slower onset of Wallerian degeneration in sensory fibers, causing SNAP amplitudes to reach their lowest point later than CMAP amplitudes [35]. There were notable shifts in electrophysiological classification between the first and second examinations, highlighting the diagnostic value of follow-up studies. Three patients initially classified as "axonal" GBS transitioned to "demyelination with RCF "patterns. 4 cases initially had inexcitable nerves; two of them still had inexcitable nerves, the rest transitioned into demyelination cases, reflecting the conduction failure, not exclusively axonal pathology. Notably, 14 patients were reclassified as having demyelination with reversible conduction failure (RCF), indicating transient conduction abnormalities. Additionally, out of the 14 initially equivocal cases, 13 (92.8%) were reclassified into specific electrophysiological categories upon follow-up testing, reflecting the value of serial electrophysiological assessments in clarifying uncertain early findings. The largest proportion (6/14; 42.8%) shifted toward axonal with no reversible conduction failure (nRCF), followed by demyelination with RCF (4/14) and demyelination with nRCF (2/14). Only one case (7.1%) remained equivocal.

This shift illustrates that initial equivocal classifications often reflect limitations in early electrophysiological changes, which may become clearer over time as pathological processes evolve. It also reinforces the importance of repeating NCS after the first two weeks, particularly in cases with unclear diagnoses.

Our findings align with previous literature emphasizing the value of serial NCS in accurately classifying GBS subtypes. Uncini *et al*. [36] demonstrated that initial electrophysiological evaluations may misclassify axonal variants as AIDP due to RCF, a phenomenon that resolves over time, leading to a diagnostic shift upon follow-up. Uncini *et al*., Subsequent studies confirmed this trend, showing a notable reclassification from demyelinating to axonal forms, with reported diagnostic yield improvements ranging from 9.6% to 24% [37, 38]. The resolution of reversible conduction failure and misclassification of subtypes were the major reasons for diagnostic shifts [38].

Conversely, some researchers advocate for the use of modified electrodiagnostic criteria, which may enhance diagnostic accuracy even in single-time-point studies [17, 32]. However, challenged the necessity of serial testing altogether, suggesting that in most patients, subtype classification can be achieved reliably with a single early study, regardless of the criteria applied. These contrasting perspectives highlight the ongoing debate regarding the optimal timing, presence of RCF, and criteria for electrophysiological diagnosis

in GBS and underscore the importance of integrating serial studies, especially in ambiguous or evolving cases. However, their findings also showed that classification varies with different criteria, and it was highly dependent on the definitions and thresholds used within each classification system. Thus, EDX-based classification may not fully capture the underlying pathophysiology, especially in borderline or mixed-pattern cases, reinforcing the value of serial assessments and multimodal diagnostic approaches.

Regarding GBS subtypes, Significant clinical differences were observed between axonal and demyelinating cases. Patients with axonal GBS had a longer hospital stay ($p = 0.006$), delayed recovery ($p = 0.004$), and poorer short-term outcomes, as indicated by higher INCAT and Hughes scores at four weeks ($p = 0.019$ and $p = 0.003$, respectively). Pure motor presentations were more frequent in axonal cases ($p = 0.013$), while sensorimotor involvement was universal in the demyelinating group.

Although some studies reported similar clinical features and intensive care needs between subtypes, including hospital stay, ventilatory support, antecedent infections, and ability to walk unaided at discharge [39, 40]. Others have shown more severe outcomes in axonal GBS. These include increased rates of dysautonomia [41], respiratory involvement, more severe clinical course, greater short-term morbidity, and slower recovery compared to demyelinating GBS [42]. Additionally, differences in age of onset and types of preceding infections have been noted between subtypes [42]. Moreover, it had poorer outcomes compared to AIDP [3, 43].

The variability in reported prognosis across studies may stem from differences in classification criteria, timing of electrophysiological assessment, patient populations, and healthcare infrastructure. Furthermore, the presence of reversible conduction failure within the axonal spectrum can lead to favorable outcomes, which may explain discrepancies between studies reporting poor versus similar recovery trajectories compared to AIDP.

CLINICAL IMPLICATIONS

Our findings carry several practical implications for the clinical management of GBS in Egypt and similar settings. First, given the high rate of diagnostic reclassification between initial and repeat electrophysiological studies, serial nerve conduction testing—optimally performed after the first two weeks—should be considered standard practice, particularly in cases with equivocal early findings. This approach can prevent misclassification of reversible conduction failure as axonal degeneration, ensuring more accurate prognostication and tailored therapy.

Second, early disability scores such as the Hughes Disability Score, INCAT, and mEGOS may serve as useful predictors of short-term outcomes, enabling clinicians to identify patients at higher risk of prolonged disability. Those with high baseline HDS (≥4) or mEGOS scores may benefit from closer monitoring, early rehabilitation planning, and consideration for rescue immunotherapy if early improvement is absent.

Finally, awareness of regional subtype distributions, possible genetic influences, and prevalent antecedent infections can help clinicians anticipate clinical course and counsel patients more effectively. Integration of these practical measures into routine care could improve both diagnostic accuracy and functional recovery rates in GBS.

LIMITATIONS

This study has several limitations. First, the sample size was relatively small ($n = 49$), which may limit statistical power for detecting differences between subgroups, particularly in less common electrophysiological patterns. This raises the potential for Type II errors, whereby clinically relevant differences may have gone undetected. Second, although data completeness was high for most variables, occasional missing data points—particularly in follow-up electrophysiological parameters—could have influenced classification accuracy and outcome assessment.

Third, this study was conducted in a single tertiary referral center, which may introduce referral bias. Such centers are more likely to admit severe or diagnostically complex GBS cases, potentially overestimating the proportion of patients with severe disability, ICU requirements, or atypical electrophysiological patterns. This limits the generalizability of our findings to the broader Egyptian GBS population, particularly those managed in primary or secondary healthcare settings. Future studies should aim for multicenter collaboration across diverse healthcare levels to capture the full clinical spectrum, reduce selection bias, and provide more representative epidemiological and prognostic data.

Fourth, the follow-up duration in this study was short, restricted to hospitalization or one-month outcomes. Given that GBS recovery often extends over several months—with some patients showing meaningful functional gains between 3–12 months—our design may have underestimated the proportion achieving complete recovery and overestimated residual disability rates. Future studies should incorporate follow-up at 3, 6, and 12 months, as recommended in prior prognostic studies, to better capture long-term disability trajectories, late relapses, and the persistence of pain or fatigue. Extended follow-up would also allow correlation of early electrophysiological patterns with sustained functional outcomes, enabling refinement of prognostic models such as mEGOS for the Egyptian population.

Finally, the absence of serological or microbiological confirmation of antecedent infections, such as *Campylobacter jejuni*, limits our ability to directly link infectious triggers to specific electrophysiological subtypes.

CONCLUSION

This prospective study highlights the clinical spectrum, electrophysiological subtypes, and short-term outcomes of Guillain-Barré Syndrome in an Egyptian cohort. The findings affirm regional consistency with a predominance of the demyelinating subtype (AIDP), male preponderance, and increasing incidence with age. Upper respiratory tract infections were the most common antecedent events, and most patients presented with sensorimotor symptoms. Despite challenges in early electrophysiological classification, repeat studies enhanced diagnostic accuracy and confirmed subtype evolution. While most patients experienced moderate to severe disability at onset, significant clinical improvement was observed within the first month, with favorable outcomes in over 65% of patients.

This study was conducted in a tertiary referral center in Egypt and subjects who are referred from different areas could be a fair representation of the whole country. Further multicenter studies are recommended to explore genetic and environmental factors influencing GBS subtype distribution and outcomes in Arab populations.

LIST OF ABBREVIATIONS

GBS = Guillain-Barré Syndrome

AIDP = Acute Inflammatory Demyelinating Polyneuropathy

AMAN = Acute Motor Axonal Neuropathy

AMSAN = Acute Motor and Sensory Axonal Neuropathy

URTI = Upper Respiratory Tract Infection

GIT = Gastrointestinal Tract

HDS = Hughes Disability Score

INCAT = Inflammatory Neuropathy Cause and Treatment

EGRIS = Erasmus GBS Respiratory Insufficiency Score

mEGOS = Modified Erasmus GBS Outcome Score

MRC = Medical Research Council

MRCSS = Medical Research Council Sum Score

PE = Plasma Exchange

IVIg = Intravenous Immunoglobulin

NCS = Nerve Conduction Study

CMAP = Compound Muscle Action Potential

SNAP = Sensory Nerve Action Potential

CV = Conduction Velocity

EDC = Extensor Digitorum Communis

TA = Tibialis Anterior

MCD = Mean Consecutive Difference

SFEMG = Single Fiber Electromyography

CB = Conduction Block

RCF = Reversible Conduction Failure

nRCF = Non-Reversible Conduction Failure

ICU = Intensive Care Unit

MV = Mechanical Ventilation

ARDS = Acute Respiratory Distress Syndrome

PE (complication) = Pulmonary Embolism

ETHICAL CONSIDERATIONS

This study was conducted in accordance with the Declaration of Helsinki and adhered to the ethical guidelines for human research outlined by the World Medical Association. Ethical approval was obtained from the Ethical Committee of the Faculty of Medicine, Mansoura University (IRB approval number: MD.22.06.658). Written informed consent was obtained from all participants or their legally authorized representatives prior to enrollment. For participants requiring surrogate consent due to illness severity, consent was documented in the presence of an independent witness. The confidentiality and privacy of all participants were strictly maintained throughout the study, and all data were anonymized before analysis. No invasive procedures were performed outside of standard diagnostic or therapeutic protocols.

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AUTHOR CONTRIBUTIONS

Dena Elghzzawy: Conceptualization, study design, clinical data acquisition, manuscript drafting.

Ayatallah Farouk: Neurophysiological data analysis and interpretation, methodology design.

Ahmed Abdelkhalek: Radiological data acquisition, statistical analysis (including dataset preparation, non-parametric testing, and regression modeling).

Ibrahim Elmenshawi: Supervision, critical review and editing of the manuscript, final approval.

All authors have read and approved the final version of the manuscript.

CONFLICTS OF INTEREST

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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