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Clinical Features and Outcomes of Pediatric Guillain–Barré Syndrome in a northeastern Brazilian state: A 27-Year Observational Study

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Abstract

Guillain–Barré syndrome (GBS) remains the leading cause of acute flaccid paralysis in children following poliomyelitis eradication, with diagnostic challenges in pediatric populations. This is an observational cohort study that describes the epidemiological, clinical, and electrophysiological characteristics of pediatric GBS over a 27-year period (1995–2022) in Rio Grande do Norte, Brazil. A total of 132 patients under 18 years were included, with a median age of onset of 8 years; 22% were younger than 3 years. Antecedent events were identified in 71.2%, predominantly respiratory infections and diarrhea. Motor–sensory and pure motor forms were most common. Acute inflammatory demyelinating polyradiculoneuropathy was the predominant electrophysiological subtype, followed by axonal variants. Mechanical ventilation was required in 23.2% of cases, and mortality was 5.3%. Pain and meningeal signs were frequent and associated with elevated cerebrospinal fluid protein levels. Despite severe presentations, outcomes were favorable, with 93% regaining independent ambulation at six months. These findings highlight distinctive pediatric features of GBS and reinforce the importance of early diagnosis and treatment to improve prognosis and decrease morbidity.

1.0 Introduction

Guillain–Barré syndrome (GBS) is a rare, immune-mediated disorder of the peripheral nervous system, usually preceded by an infectious event or, less frequently, vaccination. It is characterized by acute inflammation and damage of peripheral nerves, nerve roots, and occasionally cranial nerves [1]. The worldwide annual incidence of GBS ranges from 0.38 to 2.53 cases per 100,000 person-years [2]. Although GBS can occur at any age, population-based studies from North America and Europe report a marked increase in incidence with advancing age, peaking between 50 and 70 years [3]. In contrast, in low- and middle-income countries, where populations are younger, GBS is more frequently reported in children and young adults [4, 5]. Despite this, pediatric GBS remains underrepresented in long-term cohort studies.

GBS is a clinically and pathophysiologically heterogeneous syndrome, with wide variation in clinical presentation, electrophysiological subtypes, and outcomes across different regions of the world [6]. The predominant phenotype is the classic sensorimotor form; however, several clinical variants have been described, including pure motor GBS, paraparetic GBS, pharyngo-cervical-brachial variant, bilateral facial palsy with paresthesias, acute sensory neuropathy, Miller Fisher syndrome, and Bickerstaff brainstem encephalitis [1]. Electrophysiologically, GBS is broadly classified into demyelinating and axonal subtypes [1]. Demyelinating and sensorimotor forms predominate in North America and Europe, whereas pure motor and axonal forms are more frequently observed in Asian and South American countries, a variation potentially influenced by regional differences in demographics, type of triggering infections, and environmental factors [6].

The diagnosis of GBS is based on a combination of clinical features supported by cerebrospinal fluid (CSF) analysis and electrodiagnostic studies. Early and accurate diagnosis is essential to guide

immunotherapy, prevent complications, and reduce long-term disability and mortality. In pediatric patients, however, diagnosis may be particularly challenging. Young children often present with non-specific symptoms, limited cooperation during neurological examination, and atypical manifestations, which may delay recognition of GBS [7]. Pain and signs of meningeal irritation, which are less observed in the adult population, may be prominent features in children and can further complicate the diagnostic process.

Despite the clinical importance of pediatric GBS, data from Brazil and other in low- and middle-income countries remain scarce, particularly studies with long-term follow-up and detailed clinical and electrophysiological characterization. Most available evidence derives from adult cohorts or short-term pediatric series, limiting the understanding of age-specific clinical features and outcomes. Therefore, the present study describes the epidemiological, clinical, and electrophysiological characteristics, as well as disease severity and outcomes, of a pediatric GBS group diagnosed over a 27-year period in northeastern Brazil, encompassing cases prior to Zika and thereafter.

2.0 Results

2.1 Demographic characteristics

A total of 132 children and adolescents diagnosed with Guillain–Barré syndrome (GBS) were included in the study. The median age of GBS onset was 8 years (range, 1–17 years), and 72 patients (54.5%) were males. When stratified by age at diagnosis, 29 patients (22.0%) were aged 0–3 years, 29 (22.0%) were 4–7 years, 35 (26.5%) were 8–11 years, 27 (20.4%) were 12–15 years, and 12 (9.1%) were 16–17 years.

The annual distribution of pediatric GBS cases over the study period, compared with 445 adult GBS cases followed at the same institution, is shown in Fig. 1. Pediatric cases occurred throughout the year, with the highest number observed in May, followed by January and July. This is usually the rainy season and more children upper respiratory diseases are diagnosed. Analysis of temporal distribution by electrophysiological subtype showed a higher frequency of axonal forms between January and July.

2.2. Recent Past medical history

Antecedent events within four weeks prior to symptom onset were identified in 94 patients (71.2%). Respiratory tract infections were the most frequent antecedent event, reported in 74 patients (56.0%), followed by diarrhea in 20 patients (15.2%). Antecedent diarrhea was more frequently observed in patients with axonal electrophysiological subtypes.

2.3 Guillain Barré Clinical classification

Based on clinical presentation, 75 patients (56.8%) presented with the classic sensorimotor variant of GBS. Pure motor GBS was observed in 35 patients (26.5%). Miller Fisher syndrome was diagnosed in 2 patients (1.5%), and overlap Miller Fisher syndrome–GBS in 10 patients (7.6%). Other variants included

pure sensory GBS in 4 patients (3.0%), paraparetic GBS in 2 patients (1.5%), pharyngo-cervical-brachial variant in 1 patient (0.8%), and Bickerstaff brainstem encephalitis in 3 patients (2.3%). According to the Brighton Collaboration criteria, 61 patients (46.2%) fulfilled level 1 diagnostic certainty, 56 (42.4%) level 2, and 3 patients (2.3%) level 3.

Nerve conduction studies were performed in 123 patients (93.2%). The most frequent electrophysiological subtype was acute inflammatory demyelinating polyradiculoneuropathy (AIDP), identified in 79 patients (64.2%). Axonal forms were observed in 37 patients (30.1%), including both acute motor axonal neuropathy (AMAN) and acute motor–sensory axonal neuropathy (AMSAN). The remaining cases were classified as equivocal. These findings are summarized in Table 1.

Table 1
Clinical characteristics of the pediatric population

Variables	N values
Antecedent events	
Respiratory infection	70/125 (56.0%)
Diarrhea	19/125 (15.2%)
Vaccination	11/123 (8.9%)
Electrophysiological subtype	
Demyelinating	79/123 (64.2%)
Axonal	37/123 (30.1%)
Mixed	7/123 (5.7%)
Clinical variants	
Classic	75/132 (56.8%)
Miller Fisher syndrome (MFS)	7/132 (5.3%)
Overlap GBS + MFS	10/132 (7.6%)
Sensory ataxic	4/132 (3.0%)
Paraparesis	4/132 (3.0%)
Bickerstaff brainstem encephalitis (BBE)	3/132 (2.3%)
Pharyngo–cervico–brachial	11/132 (8.4%)
Signs and symptoms	
Pain	86/132 (65.1%)
Meningeal irritation	61/125 (48.8%)
Sensory deficit	80/121 (66.1%)
Proximal weakness	60/128 (46.9%)
Distal weakness	40/128 (31.3%)
Global weakness	26/128 (20.3%)
Cranial nerve VII	63/122 (52.1%)
Cranial nerves IX and X	61/123 (49.6%)
Clinical outcomes	
Invasive ventilation	29/125 (23.2%)

Variables	N values
Tracheostomy	10/128 (7.8%)
Days on mechanical ventilation (median)	9.5 (7–19)
Dysautonomia	42/127 (34.7%)
Days to reach nadir (median)	7 (5–10)
Days to improve by 1 point on the Hughes Scale (median)	15 (8–25)
Days to recover ambulation (median)	16 (8.5–31.25)
Good prognosis	107/115 (93.0%)
Death	5/95 (5.3%)

2.4 Neurological findings:

The main clinical features of the 132 children diagnosed with Guillain–Barré syndrome included in the study are summarized in Table 1. Regarding disease severity, 106 patients (80.3%) presented with severe weakness which impaired ambulation, and 23.2% required mechanical ventilation. Only one patient experienced disease recurrence (0.75%). Pain was a frequent symptom, reported in 70.5% of cases, and signs of meningeal irritation were observed in 48.8% of patients. In addition, most patients were classified as having the classic clinical variant and a demyelinating electrophysiological subtype. Lumbar puncture was performed a median of 10.1 days after symptom onset (range 1–60 days). The median cerebrospinal fluid protein concentration was 76 mg/dL. Patients presenting with pain and meningeal irritation showed significantly higher cerebrospinal fluid protein levels compared with those without these features ($p = 0.02$ and $p = 0.01$, respectively).

2.5 Comparison between AMAN and AIDP:

Age and sex distributions did not differ significantly between children with acute inflammatory demyelinating polyneuropathy and those with acute motor axonal neuropathy. However, children with acute motor axonal neuropathy were more likely to present with prior history of diarrhea, predominantly distal weakness, and a more rapid disease course compared with those with acute inflammatory demyelinating polyneuropathy. Detailed comparisons between the two subtypes are presented in Table 2.

Table 2
Correlation between electrophysiological subtypes and clinical-demographic characteristics of patients (N = 132)

Variables	Demyelinating	Axonal	P-values
Demographic Characteristics			
Age (years, median)	8 (2–17)	8 (1–17)	0.52
Male	38/79 (41.8%)	26/37 (70.2%)	-
Female	41/79 (51.8%)	11/37 (29.7%)	-
Antecedents			
Respiratory infection	48/78 (61.5%)	16/53 (49.6%)	0.06
Diarrhea	8/77 (10.3%)	10/34 (29.4%)	0.02
Vaccination	7/76 (9.2%)	3/29 (10.3%)	0.77
Distribution of muscle weakness			
Proximal	56/78 (71.7%)	29/55 (52.7%)	< 0.001
Distal	11/78 (14.1%)	2/55 (7.1%)	0.11
Global	12/78 (15.3%)	24/55 (43.6%)	< 0.001
Clinical outcomes			
Days on mechanical ventilation (median)	8 (7-122)	14 (4–39)	0.13
Tracheostomy	6/78 (7.6%)	1/34 (2.9%)	0.46
Days to improve by 1 point on the Hughes Scale (median)	15 (7–29)	21 (7–75)	0.22
Days to clinical nadir (median)	8.0 (3–20)	5.5 (0–10)	< 0.001
Albuminocytologic dissociation	40/51 (78.4%)	7/14 (50.0%)	0.06
CSF protein (mg/dL,median)	106 (35–384)	75.5 (4-640)	0.40
Sensory déficit	53/78 (67.9%)	3/34 (8.8%)	< 0.001
Pain	46/78 (58.9%)	25/34 (73.5%)	0.13

Variables	Demyelinating	Axonal	P-values
Meningeal irritation	18/78 (23.0%)	16/34 (47.0%)	0.001
Facial palsy	53/77 (68.8%)	6/32 (18.7%)	< 0.001
Bulbar palsy	40/77 (51.9%)	20/34 (58.8%)	0.47
Dysautonomia	28/75 (37.3%)	10/30 (33.3%)	0.72
Need for mechanical ventilation	19/78 (24.3%)	12/34 (35.3%)	0.12
Days to recover ambulation (median)	23 (15–45)	30 (7-102)	0.02
Good prognosis	66/71 (92.9%)	31/33 (93.9%)	0.85
Death	2/65 (0.03%)	2/25 (0.08%)	0.31

2.6 Age-group comparison and pediatric versus adult GBS

When demographic and clinical characteristics were compared between children younger than 9 years (< 9 years; *n* = 72; 54.5%) and those aged 9 years or older (≥ 9 years; *n* = 60; 45.5%), the only significant difference observed was the presence of meningeal signs. Meningeal irritation was more frequent among children younger than 9 years (41/65; 63.1%) compared with those aged 9 to 17 years (20/55; 36.4%) ($p = 0.0058$). No significant differences were identified between age groups with respect to sex, time to nadir, type of antecedent events, cerebrospinal fluid protein levels, electrophysiological subtype, facial palsy, bulbar palsy, ophthalmoparesis, autonomic dysfunction, requirement for mechanical ventilation, duration of mechanical ventilation, or time to recovery of independent ambulation.

3.0 Discussion

In this 27-year observational cohort of children with Guillain–Barré syndrome (GBS) from northeastern Brazil, we provide a comprehensive description of epidemiological, clinical, electrophysiological, and outcome characteristics of pediatric GBS in a low- and middle-income setting. The predominant phenotype was the classic sensorimotor variant, with both demyelinating and axonal electrophysiological subtypes represented. Despite the high frequency of severe presentations during the acute phase, functional recovery at six months was favorable in most patients, but there was a 5% mortality.

A major strength of this study is the high diagnostic certainty, as nearly 90% of patients fulfilled Brighton Collaboration levels 1 or 2. The Brighton criteria were originally developed for surveillance and vaccine safety studies, aiming to improve diagnostic accuracy and case definition [16]. Previous studies have

demonstrated good sensitivity of these criteria in pediatric GBS, particularly among hospitalized patients with more severe disease [18, 19]. In our cohort, 80.3% of patients had Hughes scores of 4 or 5 at nadir, reflecting the severity of cases evaluated and supporting the robustness of the diagnostic classification.

Consistent with previous reports, the classic sensorimotor variant was the most frequent clinical presentation, followed by pure motor GBS and Miller Fisher syndrome or overlap forms [20]. Notably, pure motor GBS was proportionally more frequent in children than in adults, as previously described [20]. Although the overall clinical presentation of pediatric GBS resembles that of adults, important age-related differences were observed. In our cohort, the median time to nadir was shorter than typically reported in adults, and pain was a particularly prominent symptom, occurring in more than two-thirds of patients.

Regarding electrophysiological subtypes, demyelinating GBS (AIDP) predominated; however, the frequency of axonal forms (approximately 30%) was relatively high. This finding is consistent with reports from Central and South America, as well as from Asia, including Japan, Israel, Bangladesh, and China, where axonal subtypes account for 30–67% of cases [21–24]. The true prevalence of axonal GBS may be underestimated in our cohort. Current electrophysiological criteria used to distinguish demyelinating from axonal subtypes have inherent limitations, particularly in early disease stages [21, 25]. We applied the criteria proposed by Hadden and Ho, which favor sensitivity for demyelinating features due to higher cut-off values for defining demyelination [10, 11]. These criteria do not fully account for reversible conduction failure, a phenomenon increasingly recognized in axonal GBS [25, 26].

Reversible conduction failure reflects immune-mediated dysfunction at the nodes of Ranvier or motor nerve terminals, often associated with anti-ganglioside antibodies, and may mimic demyelinating features in early electrophysiological studies [26]. In such cases, serial nerve conduction studies are required to accurately classify the subtype. In our cohort, proximal weakness predominated in demyelinating GBS, whereas distal weakness was more common in axonal forms, a pattern consistent with previous observations. These findings highlight the need for standardized recommendations regarding the timing and indication of serial electrophysiological studies in pediatric GBS.

GBS is an important cause of acute flaccid paralysis in childhood in Latin America, including Brazil, where an incidence of approximately 0.40 cases per 100,000 children under 15 years of age has been reported [27]. In our hospital-based cohort, nearly one-third of all GBS cases occurred in individuals younger than 18 years, with a substantial proportion of cases in children younger than 9 years and even under 3 years of age. Although GBS is often described as a disease with increasing incidence with age, studies from developing countries such as Brazil and Bangladesh have demonstrated a significant burden in the pediatric population [17, 22]. Recent European data have also suggested an increase in GBS incidence among children younger than 3 years, possibly reflecting heightened susceptibility to triggering infections in early childhood [7].

Antecedent infectious events were identified in more than 70% of patients, with respiratory tract infections and diarrhea being the most common. Diarrhea was particularly frequent among patients with

axonal subtypes, corroborating previous findings from northeastern Brazil [4]. While respiratory infections are more commonly reported as antecedent events in North America and Europe, diarrheal illness predominates in countries such as Bangladesh [22]. Although specific infectious agents were not identified in our study, the temporal distribution of cases and subtype-specific associations support the role of different infectious triggers in pediatric GBS.

Campylobacter jejuni is a well-established risk factor for GBS, particularly for axonal subtypes, and has been associated with disease clusters in countries such as China, Bangladesh, Mexico, and Peru [1, 22]. In Brazil, despite limited data, *C. jejuni* is believed to be a major infectious trigger of GBS [28, 29]. Cytomegalovirus infection, in contrast, has been associated with demyelinating GBS, particularly in younger patients [17]. Arboviral infections, including Zika virus, dengue, and chikungunya, have also been linked to GBS during outbreaks, although pediatric data remain limited [1]. During the SARS-CoV-2 pandemic, GBS cases were reported infrequently, and we did not observe an increase in pediatric or adults GBS incidence during this period [1].

Vaccination preceded symptom onset in a small number of patients, all of whom had favorable outcomes. Since the influenza A (H1N1) vaccination campaign in the 1970s, GBS has been recognized as an adverse event of special interest following immunization [32]. Subsequent studies have shown that the risk of GBS following influenza vaccination is extremely low and substantially outweighed by the benefits of vaccination [33]. With the global implementation of COVID-19 vaccination, renewed surveillance identified a small excess risk of GBS following the first dose of the Oxford–AstraZeneca vaccine, without a specific clinical phenotype [36]. Our study was not designed to establish causal relationships between vaccination and GBS, and the observed cases may represent coincidental associations.

Pain was a prominent feature in our pediatric cohort, affecting more than 70% of patients. Previous prospective studies have reported neuropathic pain in up to 79% of children with GBS, with a substantial proportion experiencing severe pain requiring aggressive management [37]. In children, pain is often an early or presenting symptom of GBS and may precede overt weakness [38]. Additionally, nearly half of our patients exhibited signs of meningeal irritation, particularly among younger children, a finding consistent with previous reports [4, 39, 40]. Age-related differences in pain perception may contribute to this observation, as younger individuals tend to have lower pain thresholds [41]. The presence of signs such as a positive Lasègue test may provide a useful and objective marker for monitoring disease evolution and response to treatment in pediatric GBS [42].

The pathophysiology of pain in GBS is likely multifactorial, involving inflammation and traction of nerve roots, as well as irritation of the nervi nervorum, resulting in referred pain to the limbs and back. During the acute phase, clinical attention often focuses on progressive weakness and life-threatening complications, such as respiratory failure and autonomic dysfunction, potentially leading to underrecognition of pain and delayed diagnosis leading to prolonged hospitalization [43].

In our cohort, higher cerebrospinal fluid protein levels were associated with the presence of pain and meningeal signs, consistent with previous studies [44]. Although demyelinating subtypes tended to have higher protein concentrations than axonal forms, this difference was not statistically significant. Elevated CSF protein levels have been associated with disease severity, mechanical ventilation requirement, and dysautonomia in other studies [45]. CSF protein elevation in GBS likely reflects inflammatory processes affecting the blood–nerve and blood–CSF barriers and may be influenced by age, sex, clinical variant, and timing of lumbar puncture [45, 46].

Despite the severity of acute presentations, long-term outcomes in our pediatric cohort were favorable. More than 90% of patients regained independent ambulation at six months, and recovery occurred relatively rapidly. Although axonal subtypes showed a slightly longer time to functional improvement, outcomes did not differ significantly between electrophysiological subtypes, consistent with previous reports [4]. Mechanical ventilation was required in approximately one-quarter of patients, a rate lower than that reported in adults but comparable to pediatric series [23, 47].

This study has limitations, including its observational design and the lack of systematic identification of infectious agents or anti-ganglioside antibodies. Nonetheless, the long duration of follow-up, consistent diagnostic assessment, and detailed clinical and electrophysiological characterization strengthen the validity of our findings.

In conclusion, axonal subtypes of GBS are frequent in children and are often associated with antecedent diarrhea, distal weakness, and rapid disease progression. Pain and meningeal signs are common and may represent prominent features of pediatric GBS, independent of electrophysiological subtype. Although the disease can be severe in the acute phase, with a substantial proportion of patients requiring mechanical ventilation, the overall prognosis at six months is favorable. Early recognition of pediatric-specific clinical features and timely initiation of appropriate treatment remain essential. Future studies incorporating serial electrophysiological assessments, microbiological testing, and anti-ganglioside antibody profiling are needed to refine diagnostic criteria and improve prognostic stratification in pediatric GBS.

In summary, Pediatric Guillain–Barré syndrome presents with distinct clinical features compared with adult disease, particularly a high frequency of pain and meningeal signs, which may complicate early diagnosis. In this 27-year observational cohort, both demyelinating and axonal electrophysiological subtypes were common, with axonal forms frequently associated with antecedent diarrhea and distal weakness. Although the acute phase was often severe, with a substantial proportion of children requiring mechanical ventilation, functional recovery at six months was favorable in most patients, regardless of electrophysiological subtype. These findings underscore the importance of recognizing pediatric-specific manifestations of GBS to ensure timely diagnosis and appropriate management. Future studies incorporating serial electrophysiological evaluations, microbiological investigations, and immunological markers are warranted to refine diagnostic criteria and improve prognostic stratification in pediatric populations.

4.0 Methods

4.1. Study design and setting

This was an observational study based on clinical and electrophysiological data from children and adolescents (< 18 years) diagnosed with Guillain–Barré syndrome in the state of Rio Grande do Norte, northeastern Brazil, between January 1995 and December 2022. Patients were identified among those hospitalized in the main public and private hospitals in the city of Natal, the state capital. The cohort was followed at the neurology center of the Integrated Clinical Medicine Department of the Onofre Lopes University Hospital, Federal University of Rio Grande do Norte, Natal, Brazil. All patients were evaluated and followed by the same neurologist (M.E.T. Dourado Júnior), who also performed or supervised the neurophysiological examinations, ensuring diagnostic consistency over the study period. Parts of this cohort have been described previously [4, 8, 17].

4.2. Diagnostic criteria and clinical classification

The diagnosis of GBS was established according to the criteria proposed by Asbury and Cornblath [9]. Diagnostic certainty was further classified using the Brighton Collaboration criteria, which define four levels of diagnostic certainty based on clinical features and ancillary investigations [16]. Clinical variants were classified as classic sensorimotor GBS, pure motor GBS, paraparetic GBS, pharyngo-cervical-brachial variant, bilateral facial palsy with paresthesias, pure sensory GBS, Miller Fisher syndrome (MFS), and Bickerstaff brainstem encephalitis [13]. Patients with MFS who developed limb weakness were classified as overlap MFS–GBS. Patients with acute ataxic neuropathy were classified either as pure sensory GBS or as an incomplete form of MFS, according to clinical and electrophysiological findings [14].

4.3 Electrophysiological studies

Nerve conduction studies were performed in patients when clinically feasible, and some patients underwent more than one examination during the disease course. Electrophysiological classification into acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), or acute motor–sensory axonal neuropathy (AMSAN) was based on established criteria [10, 11]. AMSAN was defined by the absence of demyelinating features and by a reduction of sensory nerve action potential amplitudes to less than 50% of the lower limit of normal in at least two sensory nerves, in accordance with Ho's criteria [12]. Cases that did not fulfill criteria for a specific subtype were classified as equivocal. Demyelinating cases with predominantly sensory involvement were classified as pure sensory GBS, whereas equivocal cases presenting with ataxia were classified as MFS [14].

4.4 Clinical severity and outcomes

Disease severity was assessed at the nadir using the Guillain–Barré Syndrome Disability Scale (Hughes scale), which ranges from 0 (normal) to 6 (death) [15]. Patients were classified as having mild disease (Hughes score ≤ 3) or severe disease (Hughes score ≥ 4). Mechanical ventilation requirement and

duration were recorded. Primary outcome measures included functional status at 6 months, assessed using the Hughes scale, and time to recovery of independent ambulation. A poor outcome was defined as a Hughes score ≥ 3 at 6 months, corresponding to the inability to walk independently for 5 meters.

4.5 Treatment

Patients with severe disease (Hughes score ≥ 4) and normal serum immunoglobulin A levels were treated with intravenous immunoglobulin (IVIG) at a total dose of 2 g/kg body weight, administered according to standard clinical practice, under the sponsorship of the Brazilian Minister of Health.

4.6 Data collection

Demographic and clinical data collected included age, sex, date of symptom onset, time to nadir, antecedent events (including respiratory infections, diarrhea, and vaccination), distribution and severity of muscle weakness, sensory symptoms, cranial nerve involvement, autonomic dysfunction, and need for mechanical ventilation. Cerebrospinal fluid (CSF) analysis included protein concentration and cell count, and the timing of lumbar puncture relative to symptom onset was recorded. For comparative purposes, demographic and clinical data from adult patients with GBS followed at the same institution and previously published were used [4, 8, 17].

4.7 Statistical analysis

Continuous variables with normal distribution were expressed as mean ± standard deviation, and categorical variables as absolute numbers and percentages. Non-normally distributed continuous variables were expressed as median and interquartile range (Q1–Q3). Normality was assessed using the Kolmogorov–Smirnov test.

Comparisons between groups were performed using the independent samples Student's *t* test or the Mann–Whitney *U* test, as appropriate. Associations between categorical variables were assessed using the chi-square test or Fisher's exact test. Univariate logistic regression analysis was performed to identify predictors of favorable or unfavorable outcomes, using the Mann–Whitney *U* test for continuous variables and Fisher's exact test for categorical variables. A p value < 0.05 was considered statistically significant.

4.8 Ethical considerations

The study protocol was reviewed and approved by the Ethics Review Board of the Federal University of Rio Grande do Norte (CEP-UFRN) under approval number 6.281.721. All procedures were conducted in accordance with the ethical standards for research involving human participants, in compliance with the Declaration of Helsinki and Brazilian ethical regulations governing human research. Written informed consent was obtained from all participants or their legal guardians prior to participation in the study.

Declarations

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Author contributions

METD recruited all study subjects and performed the electrophysiological studies. He wrote the first draft of the manuscript.

DFFL recruited subjects and reviewed the first draft of the manuscript.

IAFF recruited subjects and reviewed the first draft of the manuscript.

TFL recruited subjects and reviewed the first draft of the manuscript.

HPB performed the statistical analysis and reviewed the final version of the manuscript.

SMBJ recruited the subjects, managed the samples and reviewed the final version of the manuscript.

Availability of Data and Materials

All data are provided within the manuscript in the form of tables and figure.

Conflict of Interest

None of the authors have any conflict of Interest.

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Figures

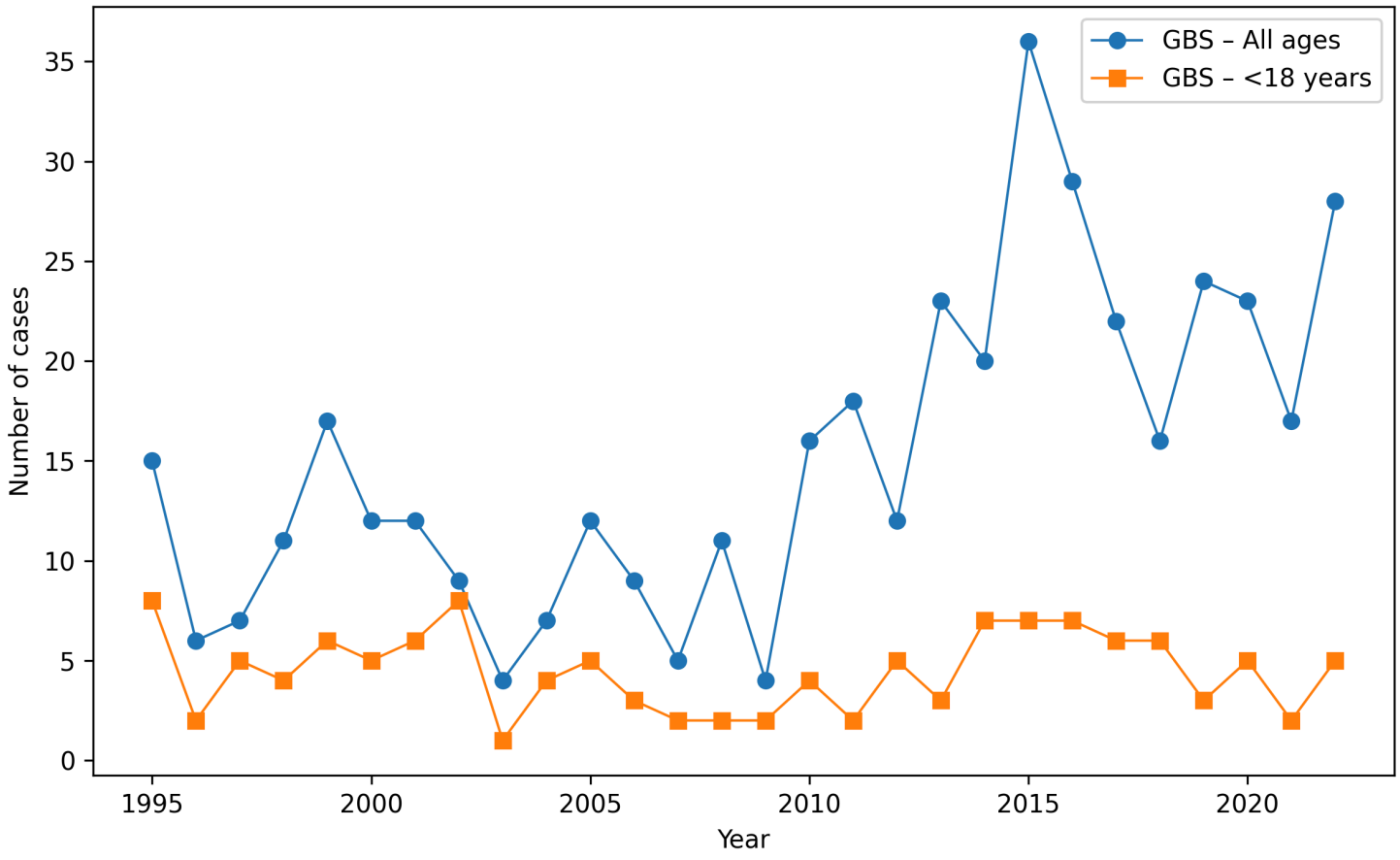


Figure 1

Guillain-Barré cases in children and adults (1995-2022) in the state of Rio Grande do Norte, Brazil.