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Central Nervous System Infections as Triggers of Guillain–Barré Syndrome in Children Under 13 Years: A Retrospective Cohort from Iraq

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Abstract

Background: Guillain-Barre syndrome (GBS) is one of the most frequent causes of acute flaccid paralysis in children, which is usually preceded by infections. There is recent evidence of a connection between GBS and central nervous system (CNS) infections (meningitis and encephalitis) in an immune-mediated manner. This study examines post-CNS infection GBS in Iraqi children investigating clinical, laboratory, and electrophysiological presentations to enhance the early identification and treatment in resource- limited settings.

Methods: This is a retrospective observational study (January 2021-December 2023) at the Central Teaching Hospital of children, Baghdad, where patients under the age of 13 years were diagnosed with CNS infection and developed GBS during hospitalization or within 4 weeks of discharge. Recorded data were on demographics, clinical, lab, and electrodiagnostic data. Based on the criteria of CSF, CNS infections were categorized as either viral or bacterial and GBS was diagnosed according to the Brighton Level 1 standards. The treatment, outcomes, and disability scores were compared and statistically analyzed with SPSS v26.

Results: In 26 children with CNS infection-related GBS (mean age 7.3 years), viral agents were the most common (65.4%). The average duration of time to develop GBS was 5.8 days. All were weak at the lower-limbs; 23% had acquired bulbar or respiratory problems necessitating ventilation. CSF demonstrated that 53.8 percent had an initial pleocytosis and progressive protein increase with albuminocytological dissociation. The most common subtype was AIDP (73.1%). IVIG was administered to 88.5%. Median hospitalization was 28.7 +- 12.4 days and no severe disability on discharge.

Conclusions: This study revealed a strong association between CNS infections and Guillain-Barre Syndrome in children below the age of 13. Autoimmune neuropathy can be induced by both the viral and bacterial infections through the creation of autoantibodies. Timely electrodiagnostic confirmation, early neurological assessment, serial CSF analysis, as well as early IVIG therapy enhance the prognosis which reinforces the role of clinical alertness and regular monitoring.

Keywords: Guillain–Barré syndrome; CNS infections; children; autoimmune neuropathy

Introduction

Guillain-Barre syndrome (GBS) is the most common cause of acute flaccid paralysis among children in the world with an annual incidence of 0.4-1.3 per 100,000 in children [1, 2]. It is a polyradiculoneuropathy that is usually triggered by antecedent infections, whereby Campylobacter jejuni, cytomegalovirus, or Epstein-Barr virus is the most common [3]. Even though peripheral infections are confirmed triggers, there is an increasing amount of evidence that central nervous system (CNS) infections, such as viral/bacterial meningitis and encephalitis, can also initiate GBS due to mechanisms that include molecular mimicry and systemic immune activation [4, 5]. This association has not been well-studied in children especially in resource-squeezed environments where CNS infections are common.

The immune system of children is more reactive and plastic, which may predispose them to post-infectious auto-immunological reactions such as GBS [6]. Children in high-burden areas of infectious diseases such as Iraq have a tendency to present with severe CNS infections that may unveil or provoke secondary neurological complications [7]. Although the typical features of adult GBS include progressive symmetric weakness and areflexia, children may have rather typical manifestations, with cranial nerves prominent involvement and faster progression [8].

Diagnosis of GBS after infection of the CNS is complicated by the overlapping clinical and cerebrospinal fluid (CSF) results. Classical albuminocytological dissociation (elevated protein and normal cell count) can be lost in the presence of persistent CSF pleocytosis due to the initial CNS infection [9]. Electrodiagnostic tests are also important to confirm but nerve conduction studies (NCS) and electromyography (EMG) are not readily available in low-resource hospitals [10].

Recent reports in Asia and the Middle East point to an increase in the rates of axonal GBS subtypes (AMAN/AMSAN), which may be related to the exposures of the regions to pathogens and genetic factors [11, 12]. In Iraq, CNS infections are still a major neurological threat with the gap in vaccination coverage of Haemophilus influenzae type b and Streptococcus pneumoniae despite improvement [13]. The commonality of CNS infection and GBS in such a setting is worth a thorough characterization because it will enhance the outcomes and early detection.

This study examines clinical, laboratory, and electrophysiological aspects of GBS that follows known CNS infection in children below the age of 13 years in a tertiary pediatric unit in Baghdad, Iraq. We would like to inform clinical practices to recognize and manage this potentially life-threatening complication in the early stages to support clinical guidelines through outlining progression patterns, diagnostic issues, and outcomes in a similar setting [14].

Methods

Study Design and Setting

Our study was a retrospective observational study conducted in Baghdad, Iraq, the Central Teaching Hospital of Children, which is a large referral hospital with a population serving central Iraq since January 2021 and until December 2023. The hospital offers advanced medical services in pediatric neurology, infectious diseases as well as respiratory critical care.

Participants

Children <13 years of age having a confirmed CNS infection (meningitis or encephalitis) and who later developed clinical characteristics congruent with GBS during hospitalization or in the 4 weeks following hospitalization were included. They were determined through electronic and paper medical records of pediatric neurology, infectious disease, respiratory care units.

Inclusion Criteria: The following criteria were met by eligible patients: (1) the age was less than 13 years; (2) CNS infection was confirmed by CSF and PCR/imaging tests; (3) the weakness of the affected lower limbs was acute and progressive, symmetric, bilateral; (4) the affected lower limbs had no reflex; (5) electrodiagnostic acute polyradiculoneuropathy was confirmed; and (6) the clinical course and investigations had been fully documented.

Exclusion Criteria: Children with pre-existing neuromuscular disorders, previous GBS episodes, neurotoxin exposure, lesions of the spinal cord on MRI, metabolic/genetic neuropathies, or other alternative diagnoses (e.g., transverse myelitis, myasthenia gravis) were excluded.

Data Collection

Demographics (age, sex, origin), clinical (fever, seizures, weakness onset), neurological (MRC grades, cranial nerve function) and outcomes (ventilation, disability) were obtained. Data on laboratory such as CSF (cell count, protein, glucose, PCR, HSV, VZV, EBV, and enterovirus), blood tests, and EMG/NCS were analyzed using age-adjusted norms [15].

CNS Infection Classification

Lymphocytic pleocytosis, normal/mildly elevated protein (less than 100mg/dl), normal glucose, and positive viral PCR when available were the criteria of viral CNS infection. Neutrophilic pleocytosis, protein <100mg/dL, glucose <40mg/dL or <50%serum, and/or positive culture was required as evidence of bacterial infection [16].

GBS Diagnosis

The diagnosis was performed based on Brighton Collaboration Level 1: progressive bilateral limb weakness, areflexia, and electrophysiological evidence of acute polyradiculoneuropathy [17]. According to Hadden *et al.* [18], electrodiagnostic patterns were divided into AIDP, AMAN, and AMSAN.

Treatment and Outcomes

Data on IVIG (2 g/kg over 2-5 days), antimicrobials (acyclovir with viral encephalitis; ceftriaxone/vancomycin with bacterial meningitis), and supportive care (mechanical ventilation; feeding) were taken. The GBS disability scale of functional status at discharge (0-6) was used to score [19].

Statistical Analysis

Data was summarized using descriptive statistics. Mean ± SD or median (IQR) were used to report continuous variables, and frequencies (percentages) were used to report categorical variables. The t-tests, Mann-Whitney U, chi-square or Fisher exact tests ($p < 0.05$) were used as the group comparisons. Analyses used SPSS v26.0.

Ethics

The hospital ethics committee gave consent to the study (Ref: CTHC-IRB-2021-08). The anonymity of patients was ensured; informed consent was not considered in case of the retrospective chart review in accordance with the Declaration of Helsinki.

Results

We identified 26 pediatric patients (mean age 7.3 ± 3.1 years; range 2–12) with CNS infection–associated GBS. Males comprised 57.7% (n = 15). The mean interval from CNS symptom onset to GBS development was 5.8 ± 2.4 days (range 2–12). (Table 1)

Table 1: Demographic and Clinical Characteristics of Study Population (N=26)

Characteristic	Value
Age (years), mean ± SD	7.3 ± 3.1
Age range (years)	2-12
Gender, n (%)	
Male	15 (57.7)
Female	11 (42.3)
Weight (kg), mean ± SD	24.6 ± 8.7
Type of CNS infection, n (%)	
Viral	17 (65.4)
Bacterial	9 (34.6)
Interval between CNS infection onset and GBS symptoms (days), mean ± SD	5.8 ± 2.4
Interval range (days)	2-12

CNS Infection Characteristics

CSF analysis confirmed viral infection in 17 cases (65.4%) and bacterial in 9 (34.6%). Among viral cases, PCR identified enterovirus (n = 4), HSV-1 (n = 2), VZV (n = 1), and EBV (n = 1). Bacterial pathogens included *S. pneumoniae* (n = 3), *N. meningitidis* (n = 2), and *H. influenzae* type b (n = 1). Initial CNS symptoms included fever (100%), vomiting (76.9%), altered mental status (73.1%), headache (61.5% in children >5 years), neck stiffness (53.8%), and seizures (38.5%). (Table 2)

Table 2: Initial CSF Characteristics at CNS Infection Diagnosis

Parameter	Viral Infection (n=17) Mean ± SD	Bacterial Infection (n=9) Mean ± SD
White blood cell count (cells/µL)	124.3 ± 87.6	842.7 ± 456.3
Neutrophil percentage (%)	18.4 ± 12.3	78.6 ± 14.2
Lymphocyte percentage (%)	81.6 ± 12.3	21.4 ± 14.2
Protein (mg/dL)	68.4 ± 32.7	187.3 ± 76.5
Glucose (mg/dL)	62.3 ± 14.7	28.4 ± 16.2
CSF/serum glucose ratio	0.61 ± 0.12	0.28 ± 0.14

Neurological Presentation and Progression

All patients exhibited bilateral lower-limb weakness and areflexia. Three progression patterns emerged:

- Pattern 1 (46.2%, n = 12): weakness confined to lower limbs (MRC 3–4); no respiratory involvement.
- Pattern 2 (30.8%, n = 8): ascending weakness to upper limbs within 4.2 ± 1.8 days (MRC 2–4).
- Pattern 3 (23.1%, n = 6): bulbar/facial/respiratory muscle involvement; required mechanical ventilation after 6.7 ± 2.1 days (mean duration: 12.3 ± 4.6 days).

Cranial nerve involvement occurred in 38.5% (n = 10)—most commonly facial palsy (n = 8). Sensory symptoms (paresthesias) affected 26.9% (n = 7); autonomic dysfunction (urinary retention, arrhythmias) occurred in 19.2% (n = 5). (Table 3)

Table 3: Neurological Presentation and Progression Patterns (N=26)

Clinical Feature	n (%)
Pattern of weakness distribution	
Limited lower limb involvement (Pattern 1)	12 (46.2)
Ascending to upper limbs (Pattern 2)	8 (30.8)
Extensive with bulbar/respiratory involvement (Pattern 3)	6 (23.1)
Associated neurological features	
Cranial nerve involvement	10 (38.5)
Facial nerve palsy	8 (30.8)
Oculomotor dysfunction	3 (11.5)
Dysphagia	4 (15.4)
Sensory symptoms	7 (26.9)
Autonomic dysfunction	5 (19.2)
Urinary retention	3 (11.5)
Cardiac arrhythmias	2 (7.7)
Blood pressure instability	2 (7.7)
Severity indicators	
Respiratory care unit admission	6 (23.1)
Mechanical ventilation required	6 (23.1)
Nasogastric tube feeding	9 (34.6)
Time to upper limb involvement (days), mean ± SD	4.2 ± 1.8
Time to respiratory failure (days), mean ± SD	6.7 ± 2.1

CSF Findings

Initial CSF protein was 68.4 ± 32.7 mg/dL (viral) vs. 187.3 ± 76.5 mg/dL (bacterial). Repeat LP after 7 days showed protein elevation in 18 patients (69.2%), rising to 142.6 ± 54.3 mg/dL (viral) and 245.8 ± 89.2 mg/dL (bacterial). Albuminocytological dissociation emerged in 14 (53.8%);

12 retained mild pleocytosis but showed declining WBC counts. Notably, 8 patients (30.8%) had no protein elevation despite electrodiagnostic GBS confirmation. (Table 4)

Table 4: Serial CSF Analysis Findings

Parameter	Initial CSF Mean ± SD	Repeat CSF (after 1 week) Mean ± SD	p-value
Viral infection group (n=17)			
White blood cell count (cells/μL)	124.3 ± 87.6	32.7 ± 28.4	<0.001
Protein (mg/dL)	68.4 ± 32.7	142.6 ± 54.3	<0.001
Glucose (mg/dL)	62.3 ± 14.7	68.4 ± 12.3	0.18
Bacterial infection group (n=9)			
White blood cell count (cells/μL)	842.7 ± 456.3	156.3 ± 124.7	<0.001
Protein (mg/dL)	187.3 ± 76.5	245.8 ± 89.2	0.04
Glucose (mg/dL)	28.4 ± 16.2	54.7 ± 18.3	<0.001
All patients (n=26)			
Albuminocytological dissociation, n (%)	3 (11.5)	14 (53.8)	<0.001
Protein elevation (>45 mg/dL), n (%)	21 (80.8)	26 (100)	0.02

Electrodiagnostic Findings

EMG/NCS (performed at 8.4 ± 3.2 days after weakness onset) confirmed GBS in all cases:

- **AIDP**: 19 (73.1%)—prolonged distal latencies, slowed conduction, absent F-waves.
- **AMAN**: 5 (19.2%)—reduced CMAP amplitudes, preserved conduction velocities.
- **AMSAN**: 2 (7.7%)—reduced CMAP and SNAP amplitudes.

Key abnormalities: prolonged/absent F-waves (92.3%), reduced CMAP (69.2%), conduction blocks (46.2%), sensory involvement (34.6%).

All received pathogen-directed antimicrobials. IVIG was given to 23 (88.5%); 3 did not receive it due to contraindications (n = 1) or rapid improvement (n = 2). Plasmapheresis was unavailable. Six (23.1%) required mechanical ventilation; 9 (34.6%) needed NG feeding.

Mean hospital stay was 28.7 ± 12.4 days—significantly longer for ventilated patients (42.3 ± 14.7 vs. 23.1 ± 8.9 days; $p < 0.01$). At discharge:

- GBS disability scale 0–1 (full/near-full recovery): 11 (42.3%)
- Scale 2–3 (moderate disability): 14 (53.8%)
- Scale 4 (bed-bound): 1 (3.8%)
- No deaths occurred.

Treatment and Outcomes

Table 5: Treatment Interventions and Clinical Outcomes (N=26)

Intervention/Outcome	n (%) or Mean ± SD
Treatment interventions	
Antimicrobial therapy	26 (100)
Acyclovir (for viral infections)	17 (65.4)
Antibiotics (for bacterial infections)	9 (34.6)
Intravenous immunoglobulin (IVIG)	23 (88.5)
Corticosteroids	4 (15.4)
Mechanical ventilation	6 (23.1)
Duration of mechanical ventilation (days), mean ± SD	12.3 ± 4.6
Clinical outcomes	
Hospital stay duration (days), mean ± SD	28.7 ± 12.4
RCU patients hospital stay (days), mean ± SD	42.3 ± 14.7
General ward patients hospital stay (days), mean ± SD	23.1 ± 8.9
Functional outcome at discharge (GBS disability scale)	
Grade 0-1 (complete recovery/minimal weakness)	11 (42.3)
Grade 2-3 (moderate disability)	14 (53.8)
Grade 4 (severe disability, bed-bound)	1 (3.8)
Grade 5 (mechanical ventilation required)	0 (0)
Grade 6 (death)	0 (0)
Mortality	0 (0)

No significant differences were found between viral vs. bacterial groups in GBS severity, subtype, or recovery—though bacterial cases had longer pre-GBS hospitalization (9.2 vs. 6.1 days; $p = 0.04$).

Discussion

This study shows that GBS can caused by both viral and bacterial CNS infections in children below age 13 years of age, and they have varied clinical courses and high morbidity. The findings of our study in Iraq (where there is a high infectious burden and an increasing measure of vaccination coverage in the population) contribute essential information to the paucity of knowledge about CNS infection-related GBS among children [20].

The average duration of GBS at 5.8 days after CNS infection fits into the immunological chronology of post infectious autoimmunity, and molecular mimicry is the probable mechanism [21]. Preponderance was in viral etiologies (65.4%), which is in accordance with the world trends in CNS infection of the pediatrics, in particular in vaccinated groups [22]. It is interesting to note that cases of bacterial GBS (all of which were ICU patients, at least in terms of pre-GBS hospitalization) imply a more aggressive course, but our limited sample size prevented statistical significance.

The spectrum of clinical was heterogeneous as almost half of them had lower-limb weakness, and 23% acquired life-threatening respiratory failure. This is unlike in adult GBS where more than 80% of them advance to upper limbs [23]. Greater cranial nerve involvement (38.5%) is similar to results in the pediatric literature, which may be due to differences in myelination of nerve fibers and immune response in the developing central nervous system [24].

The problem of diagnoses was eminent. Repeat LP demonstrated classic albuminocytological dissociation only in 54% which is a reflection of interference by the continuing CNS inflammation. It highlights the fact that GBS diagnosis here would have to be based on integrated clinical, CSF and electrodiagnostic evidence-not CSF but rather CSF-only [25]. This 31% protein increase in the unprotected also criticizes the dogma that protein elevation in the CSF is critical in diagnosis, also reiterating the recent Brighton criteria that multimodal confirmation is important [17].

AIDP prevailed (73%), which is in line with Western cohorts [26]. Nevertheless, the 27% form of axons (AMAN/AMSAN) are higher than the standard Western rates (<15%) but equivalent to those of Asia and the Middle East [11, 12], indicating regional pathogen or genetic determinants to GBS phenotype.

Intervention according to international standards: IVIG in 88.5 percent, since plasmapheresis was not available- it is a frequent restriction in low-resource countries [27]. Nevertheless, having zero mortality is a demonstration of sufficient supportive care, such as the provision of timely ventilation. Nevertheless, they only had 42% of the patients recovering on discharge- a low figure compared to the 60-70% normally reported [8]. This can indicate dichotomous CNS/ peripheral injury or retarded IVIG in certain cases.

Clinical implications of the study

The clinical implications of our findings include**:**

- Neurological follow-up must be common to all CNS infections of children.
- Repetition LP 7days later can indicate changing GBS-pattern CSF.
- Diagnosis requires early EMG/NCS (after day 7).
- IVIG is not to be delayed even in the case of atypical CSF.

Limitation and future direction

It has limitations such as retrospective design, small sample size, non-long-term follow-up, and absence of anti-ganglioside antibody testing, which is now widely used as a subtyping method of GBS. Future multicenter prospective studies should assess GBS incidence post-CNS infection, validate clinical prediction tools, and explore biomarkers (e.g., anti-GM1, anti-GQ1b) for early identification. Given global disparities in neurological care, context-specific GBS management algorithms are urgently needed.

Conclusions

This study establishes a strong association between infection of the central nervous system (CNS) and the occurrence of Guillain-Barre Syndrome (GBS) later in children below the age of 13 years. Viral CNS infections as well as bacterial CNS infections can lead to the development of autoantibody against peripheral nerves, which induces immune-mediated polyradiculoneuropathy. Affected children have a progressive ascending weakness which starts in the lower extremities and may progress to respiratory failure, clinically. Due to timely diagnosis and management, early neurological assessment is necessary. Diagnosis is dependent on clinical presentation, cerebrospinal fluid examination indicating that the patient had albuminocytologic dissociation and electrodiagnostic results indicating that the patient has acute neuropathy. Combining intravenous immunoglobulin (IVIG) treatment with intensive supportive care results in positive outcomes and zero mortality, although some patients have long-term recovery and deficit of disability. The article identifies the necessity of the increased clinical awareness, the regular neurological follow-up, and timely intervention in order to alleviate the severe complications. Minor pediatric patients should be identified with CNS infections as possible triggers of autoimmune neuropathies to enhance better early detection and prognosis.

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None

Conflicts of Interest

None declared.

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