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CASE REPORT

From Nerve to Autoimmunity: Acute Guillain-Barré Syndrome in a 4-Year-Old with Early-Onset Pediatric Systemic Lupus Erythematosus

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Objective: To report an unusual pediatric case of Guillain-Barré Syndrome (GBS) presented as the first manifestation of Systemic Lupus Erythematosus (SLE), highlighting diagnostic and clinical considerations.

Methods: We document the clinical presentation, laboratory findings, diagnostic investigations, and management of a 4-year-old boy who presented with progressive weakness and sensory deficits. Initial Electrophysiology and cerebrospinal fluid analysis reveal GBS diagnosis while, autoimmune and renal workup revealed an underlying SLE.

Results: We report a case of a 4-year-old boy who presented with progressive bilateral lower-limb weakness, absent deep tendon reflexes, sensory loss, and muscle weakness, confirmed as GBS through electrophysiological studies and cerebrospinal fluid analysis. Further investigations revealed thrombocytopenia, elevated antinuclear antibody titers, double-stranded DNA antibodies, proteinuria, and hematuria, leading to the diagnosis of SLE with GBS as the initial manifestation. The patient was referred for rheumatology and nephrology management and recovered from GBS but was diagnosed with SLE, complicated by membranous lupus nephritis (class V).

Conclusion: GBS can rarely present as the first neurological manifestation of pediatric SLE. Early recognition and a multidisciplinary approach are critical for effective management and improved outcomes.

Plain Language Summary: Guillain-Barré Syndrome (GBS) is a rare neurological complication of Systemic Lupus Erythematosus (SLE), with limited literature in pediatric cases. We report a case of a 4-year-old boy who presented with progressive bilateral lower-limb weakness, absent deep tendon reflexes, sensory loss, and muscle weakness. Tests confirmed GBS, and further blood and urine tests revealed SLE. The patient was referred for rheumatology and nephrology management and recovered from GBS but was diagnosed with SLE, complicated by membranous lupus nephritis (class V). This case emphasizes that physicians should consider SLE when pediatric patients present with acute GBS, so that treatment can be effective, and multidisciplinary care can be started.

Keywords: case report, systemic lupus erythematosus, guillain-barré syndrome, pediatric

Introduction

Guillain-Barré syndrome (GBS) is considered an acute, immune-mediated disease affecting the peripheral nerves and leading to progressive limb weakness with decreased or absent reflexes.[1] Most cases in pediatric patients follow gastrointestinal or respiratory infection, with an approximate incidence of 1.51 per 100,000 in children under 15 years old annually.[1]

Based on electrophysiological findings, GBS is subdivided into acute inflammatory demyelinating polyradiculoneuropathy (AIDP), which is considered the most reported type in western countries, and acute motor axonal neuropathy (AMAN), which is more observed in the pediatric population.[2]

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Pediatric SLE, or juvenile-onset SLE, is a rare autoimmune condition that affects approximately 15–20% of all SLE patients and often leads to significant disability.[3,4] Compared to adult-onset SLE, pediatric SLE presents unique challenges in diagnosis and management due to its broader spectrum of clinical presentations.[5] Renal involvement is common among the pediatric population with proteinuria, hematuria, and hypertension, being among the earliest and most common clinical manifestations.[6] Hematological abnormalities are reported in children as well, including; thrombocytopenia, leukopenia, and anemia.[6] In addition to renal, dermatologic, hematologic, and musculoskeletal manifestations, neuropsychiatric manifestations, though uncommon, are also observed.[3] These neuropsychiatric manifestations are associated with poor outcomes and a lack of clearly defined management guidelines.[7]

Among the neuropsychiatric manifestations, Guillain-Barré Syndrome (GBS) is considered particularly rare.[8] Defined by the American College of Rheumatology, GBS in the context of SLE is classified as an NPSLE syndrome affecting the peripheral nervous system.[9] It can occur as a rare initial presentation or a late manifestation of the disease, complicating the clinical course of the disease.[9]

The underlying pathogenesis link between SLE and GBS is still not fully understood, but generalized immune system activation and targeting of peripheral nerve antigens in susceptible individuals, as well as, immune cross-reactivity, are on top of the theory explaining this rare association.[10]

Currently, there are no universally approved treatment guidelines for managing GBS in the context of SLE.[11] However, literature suggests that various immunosuppressive and immunomodulatory therapies, such as glucocorticoids, plasma exchange, intravenous immunoglobulin (IVIG), mycophenolate mofetil, and cyclophosphamide, have been effective in controlling symptoms and improving patient outcomes.[12] This emphasizes the need for a tailored approach in managing such cases, especially in the pediatric population, taking into consideration the severity and progression of both conditions.

In this case report, we present a rare occurrence of GBS as the initial presentation of underlying pediatric SLE. This case highlights the importance of taking into consideration an autoimmune etiology in pediatric patients presenting with GBS, particularly in those with suggestive clinical features or a family history of autoimmune diseases. Our report aims to contribute to the existing literature on this rare presentation, emphasizing the diagnostic challenges and the need for a multidisciplinary approach to management.

Case Presentation

History

A previously healthy 4-year-old boy presented to our hospital with a 4-week history of progressive bilateral lower-limb weakness. The symptoms began with difficulty climbing stairs, which progressed to fatigue during physical activity a few days later. The patient had no recent respiratory tract or gastrointestinal infections, no recent vaccinations or travel history, and no history of trauma, medication intake, or toxin exposure (such as arsenic, mercury, thallium, or paralytic shellfish poisoning). He had received all vaccinations per the national vaccination program, including against poliovirus. The patient's family history was unremarkable.

Examination

On admission, the patient was conscious and alert, with vital signs within normal ranges. Physical examination revealed absent deep tendon reflexes in the lower limbs, loss of sensation in the upper and lower limbs, a positive Gower sign, and symmetrical proximal muscle weakness in the lower limbs, with a strength of grade 4 according to the Medical Research Council scale for muscle strength.[13,14] There was no muscle fasciculation, clonus, ataxia, or enlarged lymph nodes, and the Babinski sign was negative. The cranial nerve examination was also unremarkable, and no skin rash, purpura, or petechiae were observed.

Investigations and Treatment

Initial complete blood count revealed a noticeable decrease in platelet count (Table 1). The erythrocyte sedimentation rate (ESR) was elevated at 145 mm/h, while C-reactive protein (CRP) level was within the normal range. A blood film

Table 1 Longitudinal Analysis of Laboratory Results Across Time Intervals

Test (Unit)	At Admission	Day 7 PA	Day 28 PA	Day 38 PA	Day 72 PA	Day 128 PA	Reference Range
Complete blood count							
Haemoglobin (g/dL)	11.9	11.7	12.3	11.9	12.4	12	9.5–14
WBC (× 10^3 per µL)	5.47	5.87	5.6	21.6	5.66	7.36	5.5–11
Platelet count (× 10^3 per µL)	17	159	21	162	73	321	150–450
Metabolic Panel							
Vitamin B12 (pmol/L)	331	N/A	N/A	N/A	N/A	N/A	181–795
Calcium (md/dL)	8.9	9.3	9.2	9.3	8.8	8.6	8.4–10.5
Potassium (mmol/L)	3.51	3.89	4.01	3.89	3.61	3.72	3.2–5.7
Creatinine (mg/dL)	0.25	0.31	0.28	0.3	0.33	0.39	0.3–0.7
Urea (mg/dL)	26	24	33	29	38	36	15–45
Creatine kinase (U/L)	89	N/A	N/A	N/A	N/A	N/A	75–230
Thyroid-stimulating hormone (mIU/L)	3.24	N/A	N/A	N/A	N/A	N/A	0.7–5.97
Free Thyroxine T4 (pmol/L)	13.82	N/A	N/A	N/A	N/A	N/A	12–21
Cerebrospinal fluid							
Protein (mg/dL)	73	N/A	N/A	N/A	N/A	N/A	15–60
WBC (× 10^6/L)	2	N/A	N/A	N/A	N/A	N/A	<=5
Red blood cell (x 10^6/L)	0	N/A	N/A	N/A	N/A	N/A	None
Glucose (mg/dL)	63	N/A	N/A	N/A	N/A	N/A	60–80
Urinalysis							
Protein	N/A	N/A	N/A	+3	+2	+2	Negative
WBC (cells/hpf)	N/A	N/A	N/A	3–5	1–2	0–2	<=5
RBC (cells/hpf)	N/A	N/A	N/A	Many	Many	6–8	<=5

Abbreviations: PA, post admission; WBC, white blood cell count; RBC, red blood cell count.

confirmed thrombocytopenia and was negative for other cellular abnormalities (Images are unavailable - test was done in an external institution and team was only able to retrieve the report). Despite the low platelet count, a lumbar puncture was performed, and cerebrospinal fluid (CSF) analysis revealed cytoalbuminologic dissociation, consistent with GBS (Table 2). Computed tomography (CT) of the head and non-contrast magnetic resonance imaging (MRI) of the spine were negative for hemorrhages, masses, and other structural abnormalities (Figure 1). Based on these findings and with reference to the Brighton criteria,[15] a diagnosis of GBS with immune thrombocytopenic purpura (ITP) was made, and intravenous immunoglobulin (IVIG) (1 g/kg/day) was administered for 2 days.

One week post-admission (PA), the platelet count had increased to 159 × 10^3 per µL, but there was no change in the patient's symptoms or physical examination findings. The patient was discharged and scheduled for regular outpatient clinic visits (Figure 2). At the first clinic visit (day 10 PA), the child had restored sensation to pain in both the upper and

Table 2 Cerebrospinal Fluid (CSF) Analysis

White blood cells	2 cells/µL (normal)
Red blood cells	0 cells/µL (normal)
Protein	73 mg/dL (elevated)
Glucose	63 mg/dL (normal)
Cytology	No malignant or abnormal cells
Interpretation: Cytoalbuminologic dissociation, consistent with Guillain–Barré syndrome	

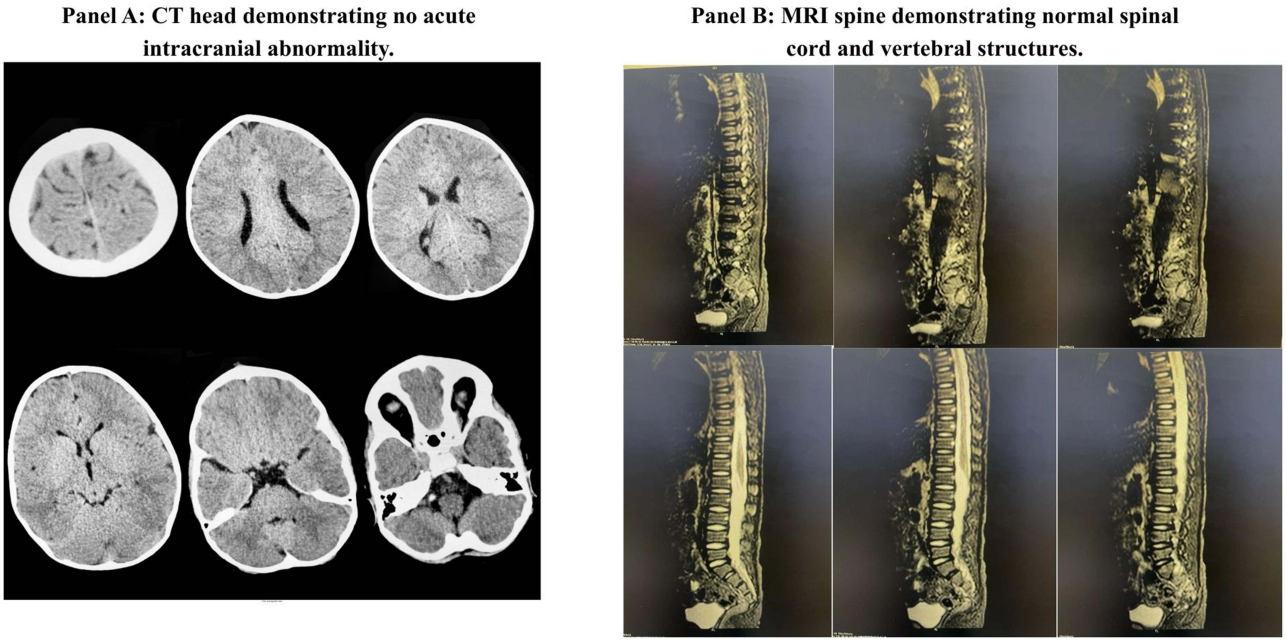

Figure 1 Diagnostic Evaluation, including: Panel (**A**) CT head demonstrating no acute intracranial abnormality, Panel (**B**) MRI spine demonstrating normal spinal cord and vertebral structures.

lower extremities. However, bilateral lower-limb weakness and the absence of deep tendon reflexes persisted. Another dose of IVIG (1 g/kg/day) was administered for 2 days, and the patient was referred to another hospital for a nerve conduction study (NCS). The NCS of the lower limbs revealed decreased compound muscle action potential amplitudes of both the peroneal and tibial nerves, with normal distal motor latencies and motor conduction velocities. Lower limb sensory studies revealed normal sensory nerve action potential amplitudes in both sural nerves, with normal peak latencies. No F-waves were recorded in the lower limbs, confirming the diagnosis of pure acute motor axonal neuropathy (AMAN), a variant of GBS affecting the lower limbs.

On day 28 PA, the patient exhibited progressive recovery, and the Gower sign was negative. However, the deep tendon reflexes were still absent, and the platelet count had dropped to 21 × 10^3 per µL. Another dose of IVIG (0.5 g/kg/day) was administered for 2 days. At the next visit (day 31 PA), despite treatment adherence, the platelet count did not improve as desired. Oral prednisolone (4 mg/kg) was prescribed for 5 days. One week later (day 38 PA), the patient was febrile with dysuria. A complete septic investigation was performed. Urinalysis showed +3 proteinuria, numerous RBCs, and urine casts with a normal count of white blood cells in the urine, while the platelet count was 162 × 10^3 per µL. Paracetamol and intravenous ceftriaxone were administered, leading to an improvement in the patient's condition. Figure 3 shows the patient's platelet count fluctuation over the course of admission and with the treatment provided.

At subsequent clinic visits (days 42, 49, 56, and 62 PA), platelet count readings were within acceptable ranges, but proteinuria and microscopic hematuria persisted. The patient was referred to the pediatric nephrology clinic for further investigation. A rheumatologic workup revealed positive antinuclear antibody (ANA) and anti-double-stranded (ds) DNA titres. ESR remained elevated (45 mm/h) with a low platelet count (11 × 10^3 per µL). Table 3 shows the results of other laboratory tests. Another dose of oral prednisolone (4 mg/kg) was administered for 5 days.

The diagnosis of SLE was confirmed based on five of the 17 Systemic Lupus International Collaborating Clinics (SLICC) group classification criteria: positive ANA and anti-dsDNA, thrombocytopenia, proteinuria, and neurological manifestations. Oral prednisolone was prescribed.[16]

A kidney biopsy performed months later at another hospital revealed a slightly cellular mesangium with scattered electron-dense deposits. The glomerular basement membrane was moderately thickened with numerous intramembranous and subepithelial deposits. The foot processes were effaced. Based on the classification system by the International

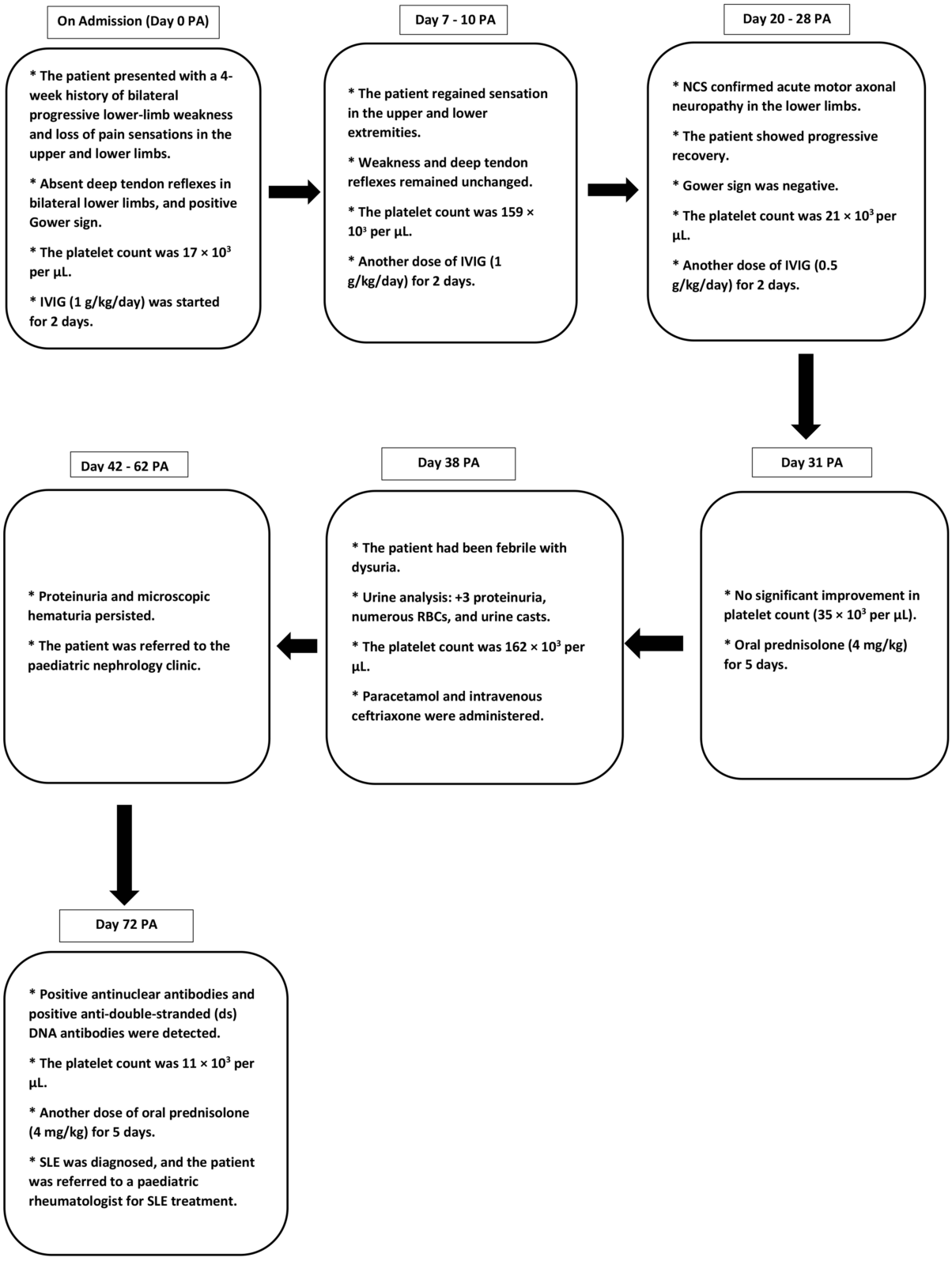

Figure 2 Summary of Events Following the Patient's Admission and Clinic Visits.

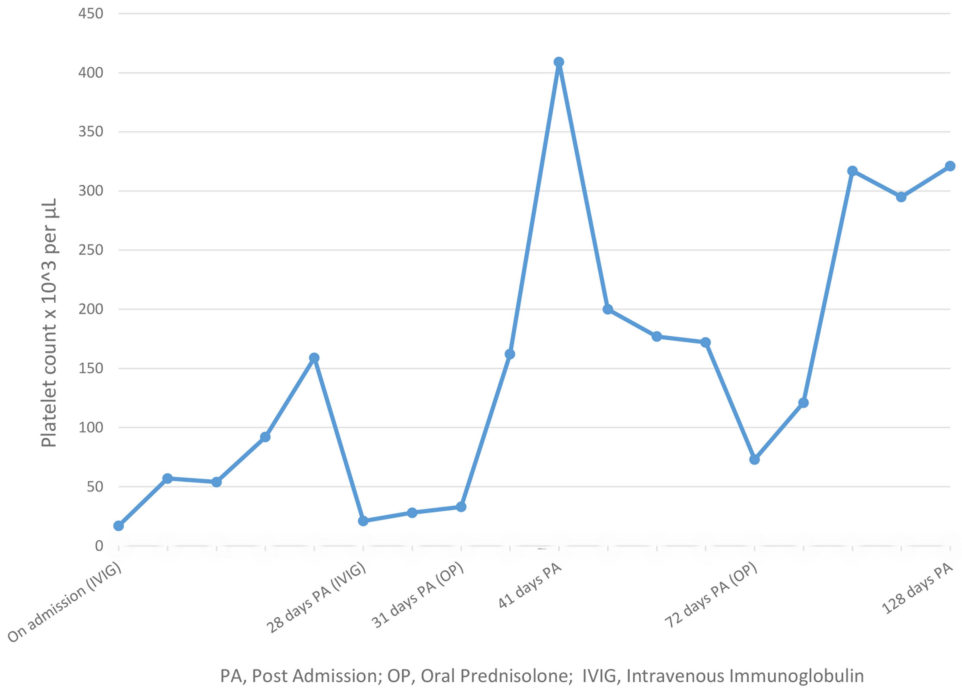

Figure 3 Patient Platelet Count Results Showing Fluctuations with Intravenous Immunoglobulin and Oral Prednisolone Administration.

Society of Nephrology (ISN),[17] these findings suggested membranous lupus nephritis (class V). The patient was referred to pediatric rheumatology and nephrology clinics for appropriate treatments (The original biopsy images were not retrievable as the procedure was performed externally; findings are based on the documented pathology report).

Table 3 Laboratory Results at Day 68 Post-Admission

Test	Results	Reference Range
Autoimmune serology		
Erythrocyte sedimentation rate	45 mm/h	≤ 10
Anti-DNA (DS) antibody	1:20	Negative
Antiscleroderma-70 Antibodies	Negative	0.0–0.9 AI
Smith antibody	Negative	0.0–0.9 AI
Anti-ribosomal P antibody	Negative	0.0–0.9 AI
Sjogren's SS-A (Ro) antibody	Negative	0.0–0.9 AI
Sjogren's SS-B (La) antibody	Negative	0.0–0.9 AI
Anti-Centromere B Antibodies	Negative	0.0–0.9 AI
Anti-RNP antibodies	Negative	0.0–0.9 AI
ANA (Antiuclear AB)	Speckled 1:320	Negative
Cytoplasmic anti-neutrophil cytoplasmic antibodies (cANCA)	Negative	Negative
Perinuclear anti-neutrophil cytoplasmic antibodies (pANCA)	Negative	Negative

(*Continued*)

Table 3 (Continued).

Test	Results	Reference Range
Anti-histone antibodies	Negative	0.0–0.9 U
Anti-Cardiolipin IgG	3.57 GPL Units	0.0–9.9 GPL Units
Anti-Cardiolipin IgM	8.28 MPL Units	0.0–9.9 MPL Units
Antiphospholipid IgG	1.64 U/mL	0.0–9.9 U/mL
Antiphospholipid IgM	3.15 U/mL	0.0–9.9 U/mL
Complement C3	90 mg/dL	80–193 mg/dL
Complement C4	24.4 mg/dL	14–57 mg/dL
Rheumatoid factor	< 20.0 IU/mL	≤ 30 IU/mL
Urine studies		
Protein random urine	28.9 mg/dL	0–12 mg/dL
PH	6	4.6–8.0
Glucose	Negative	Negative
Protein	+1	Negative
White blood cells	0–2	0 to 5/HPF
Red blood cells (RBCs)	Many	0 to 5/HPF
Casts	Granular cast	
Haemoglobin	+3 ≥ 50	Negative
Relevant blood tests		
Platelet count (x 10^3 per µL)	11	150 – 450

Discussion

SLE is an autoimmune disease characterized by the widespread formation of autoantibodies against the cell nucleus, leading to variable clinical presentations.[18] Neurological manifestations in SLE can range from mild headaches and confusion to more severe conditions such as stroke and myelopathy. The American College of Rheumatology defines neurological and psychiatric manifestations related to SLE as NPSLE syndromes.[19] The prevalence of NPSLE varies significantly, ranging from 4% to 91%. This wide variation is attributed to differences in clinical manifestations, varying selection criteria, and the diversity of study populations.[20–22]

GBS is a rare manifestation of SLE and an NPSLE syndrome that affects the peripheral nervous system. It can occur either simultaneously with the initial SLE presentation or later in the course of the disease.[19,23] The exact pathophysiology underlying this association remains unclear. One possible mechanism involves the formation of anti-myelin sheath autoantibodies due to the massive immunological response in SLE.[24] Another proposed mechanism is the involvement of the vascular system in SLE, leading to small-vessel vasculitis changes and ischemic demyelination.[20]

In the preliminary presentation of a young child with bilateral progressive lower-limb weakness, absent deep tendon reflexes, loss of sensation, and inability to walk, differential diagnoses included GBS (acute inflammatory demyelinating polyneuropathy), subacute combined degeneration of the spinal cord associated with vitamin B12 deficiency, central nervous system tumors, and electrolyte disturbances (eg, potassium). Poliomyelitis and peripheral nerve toxicity were excluded based on the child's vaccination history and lack of toxin exposure.[25]

Table 4 Guillain-Barré Syndrome in Pediatric Systemic Lupus Erythematosus: A Summary

Study ID	Age	Gender	GBS is First Presentation of SLE	Clinical Presentation	Management	Need for Respiratory Support	Outcome
Miyagawa et al 2000[34]	**13**	**Female**	**No** The diagnosis of SLE was made when the patient was 7 years old	• Flaccid quadriplegia. • Absent reflexes. • Gradual involvement of cranial nerves following admission.	• PLE. • IVIG at 0.4 g/kg. • High-dose glucocorticoids.	**Yes** Hospitalised for 27 days in the paediatric intensive care unit (PICU) where ventilation was required	**Complete recovery**
Beshir et al 2022[35]	**14**	**Female**	**Yes** Patient was previously healthy	• Progressive ascending weakness for 4 weeks. • Diplopia. • Dysphagia.	• 14 sessions of PLE. • IVIG at 2 g/kg. • High-dose glucocorticoids. • Rituximab. • Mycophenolate mofetil. • Hydroxychloroquine.	**Yes** Endotracheal intubation and ventilation "Complications related to ventilator-associated *Streptococcus pneumoniae* pneumonia"	**Complete recovery**
Reddy et al 2019[36]	**9**	**Female**	**Yes** The patient had a history of an erythematous rash over the cheeks for 1 month prior to presentation.	• Bilateral lower limb pain. • Difficulty walking. • PICU admission due to rapid deterioration of symptoms (difficulty swallowing, loss of gag reflex, decreased speech output, loss of head control, and respiratory failure).	• IVIG at 2 g/kg. • High-dose glucocorticoids. • Cyclophosphamide. • Rituximab.	**Yes** Patient Needed ventilation for 32 days.	**Complete recovery**
Parvaneh et al 2019[37]	**12**	**Male**	**Yes** Positive SLE history (22-year-old sister). Mother had previous recurrent abortions.	• Lower extremity weakness. • Bilateral lower limb pain. • Difficulty walking.	• IVIG. • High-dose glucocorticoids. • Cyclophosphamide. • Hydroxychloroquine. • Symptoms did not improve with IVIG alone. • Recurrent relapse and admissions • Symptoms improved significantly with methylprednisolone, hydroxychloroquine, and cyclophosphamide treatment	**No**	**Complete recovery**

Abbreviations: PLE, Plasma exchange; IVIG, Intravenous Immunoglobulin.

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IVIG and plasma exchange (PLE) are established treatments for GBS, with a preference for IVIG according to recent recommendations.[26] There are no specific recommendations for the use of glucocorticoids alone for isolated GBS.[12] However, our patient's platelet count improved significantly with corticosteroid pulse therapy, aligning with current guidelines that suggest using pulse glucocorticoids in patients with SLE presenting with inflammatory neuropsychiatric manifestations (Figure 2).[22,27] In addition to IVIG, PLE, and glucocorticoids, some adult patients with SLE and GBS have been treated with mycophenolate mofetil and/or cyclophosphamide.[8,28–30] Despite initial profound sensory loss, normal sensory findings were observed on nerve conduction studies (NCS), likely due to the IVIG treatment administered before the NCS.

A retrospective analysis by Xianbin et al (2015) included 4924 patients with SLE, of whom only 73 had peripheral nervous system involvement, with just one case of GBS.[31] A systematic review of nine articles identified only 2 GBS cases among 1463 SLE patients.[32] Additionally, Bortoluzzi et al (2019) reported no GBS cases in a retrospective cohort study of 1224 SLE patients.[33] To date, only three cases of GBS as the initial presentation of pediatric SLE have been reported, with one additional case in a previously diagnosed pediatric patient with SLE, Table 4 represents summarized review of pediatric SLE/GBS cases.

Compared to our patient who did not need respiratory support, three patients reported the need for mechanical ventilation. 34–36 In addition, Miyagawa et al, Beshir et al, and Reddy et al reported involvement of cranial nerves in the initial presentation of their patients, 34–36 which was not observed in ours and Parvaneh et al papers.[37] Regarding the recovery of patients, all studies reported complete recovery.[34–37]

It is important to note that juvenile-onset SLE can present with severe conditions as the first manifestation, including GBS and neuropsychiatric conditions such as Macrophage Activation Syndrome and Severe Thrombocytopenia.[38,39]

There is a shortage of studies exploring the association between GBS and SLE, particularly in the pediatric population. Among the four reported pediatric cases, severe manifestations were associated with female sex, including severe weakness and the need for respiratory support. In contrast, male patients did not show signs of respiratory insufficiency and did not require ventilation. Further studies are needed to assess the association between GBS and SLE more thoroughly and to explore the potential effect of sex on symptom severity (Table 4).

Although this case highlights the unusual associations between SLE and GBS in the pediatric patients, being a single-case report with a limited follow-up duration, restricts the ability to generalize the findings. In addition, the comparison with other reported cases is limited due to differences in the variables used. However, the detailed description of this case, starting from the presentation, clinical course, treatment strategies and outcomes, provides insights for physicians encountering similar presentations.

Conclusion

Guillain-Barré syndrome (GBS) is a rare neuropsychiatric manifestation of systemic lupus erythematosus (SLE). Clinicians should consider SLE in patients presenting with GBS, particularly those with a family history of autoimmune diseases, proteinuria, hematuria, or low platelet counts. Early suspicion of this association is critical, as timely diagnosis and initiation of immunosuppressive therapy can hugely improve neurological outcomes and overall prognosis Recent guidelines recommend pulse glucocorticoids for inflammatory neuropsychiatric symptoms in SLE, which could improve patient outcomes.

This case emphasizes the importance of detection and a multidisciplinary approach in managing atypical neurological presentations that may mask an autoimmune pathology.

Abbreviations

GBS, Guillain-Barré syndrome; SLE, systemic lupus erythematosus; NPSLE, Neuropsychiatric SLE; IVIG, Intravenous immunoglobulin; AMAN, acute motor axonal neuropathy.

Data Sharing Statement

All data generated or analyzed during this study are included in this published article.

Ethical Approval

Institutional review board approval of Al-zarka Governmental Hospital was acquired for this publication according to the policies of Jordanian Ministry of Health.

Consent

Written informed consent was obtained from the patient's legal guardian for publication of this case report.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflicts of interest in this work.

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