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Case Report

Seizures and Guillain-Barré Syndrome Leading to the Discovery of Systemic Lupus: Case of an Adolescent in Lomé and Review of the Literature

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Abstrat

Background: Systemic lupus erythematosus (SLE) is a chronic idiopathic autoimmune disease. Guillain-Barré syndrome (GBS) is an acute monophasic immune-mediated polyradiculoneuropathy. Case presentation: We report the case of a 16-year-old patient, who presented in November 2023 a flu-like syndrome with diarrhea and abdominal pain. Then he had visual hallucinations and psychosis, followed by generalized tonic-clonic seizures. Urine toxicology assay was positive, a syndrome of hepatic cytolysis and cholestasis were noted. Toxic encephalopathy and hepatopathy were mentioned and he was transferred to a psychiatric ward where he persisted with an infectious syndrome, hallucinations, delirium and then flaccid tetraplegia of ascending evolution with dyspnea and respiratory distress. In January 2024, there was tetraplegia, abolished deep tendon reflexes (DTR) in all 4 limbs, hypoesthesia in all 4 limbs. Cerebrospinal fluid examination found albumin -cytological dissociation. Magnetic resonance imaging of the spinal cord was normal. He received 500 mg of intravenous methylprednisolone for 3 days then a per os relay with prednisolone 50 mg in addition to azathioprine (Aza) 50 mg x2/day and functional rehabilitation. The electroencephalogram revealed signs of non-structural generalised epilepsy in March 2024. The electroneuromyogram found a neurophysiological pattern of acute polyradiculoneuropathy in the recovery phase. In the evolution, an antinuclear antibody assay (indirect immunofluorescence) came back positive at a titer of 1/160, with a speckled appearance. On the basis of the American College of Rhumatology (ACR) with a score of 19, the diagnosis of SLE was retained and background treatment with Aza 50 mg/day continued. At the end of May 2025, there persisted a predominantly distal tetraparesis, an abolition of DTR. Conclusion: Although rare, GBS can be the mode of revelation of SLE. This SLE-GBS association is often serious and can be life-threatening.

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Keywords

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1. Introduction

Systemic lupus erythematosus (SLE) is a chronic idiopathic autoimmune disease that affects various organs, including the central nervous system (CNS) and peripheral nervous system (PNS) [1]. Guillain-Barré syndrome (GBS) is defined as an acute monophasic immune-mediated polyradiculoneuropathy, usually post-infectious, and is the leading cause of acquired neuromuscular paralysis worldwide [2]. The diagnosis of GBS is made based on clinical features, supported by laboratory findings and electrophysiology. Intravenous immunoglobulin (IV Ig) and plasma exchange remain the main therapeutic modalities, regardless of the electrophysiological subtype.

The presentation of severe GBS as the inaugural manifestation of SLE remains exceptional [3]. Reported cases of GBS associated with SLE worldwide are essentially clinical cases. Cases have been reported in the Maghreb [4, 5] but few in sub-Saharan Africa [6]. In Togo, studies on Guillain-Barré syndrome exist [7, 8]. We report the case of a 17-year-old adolescent in whom convulsive seizures followed by severe GBS revealed lupus disease. To our knowledge, this is the first case of this kind reported in Togo.

2. Methods

The study was approved by our local ethics committee and oral informed consent was obtained from the patient for the publication of this case report.

3. Case Presentation

In December 2023, a 16-year-old right-handed student and football player was hospitalized for initial generalized tonico-clonic seizures.

His personal history is poor: there is no notion of sickle cell disease, asthma, no cardio- cerebrovascular risk factors, no surgical history. There is no report of alcohol, tobacco or drug intoxication but there is an almost daily consumption of energy drinks for 3 years. His family history includes epilepsy and high blood pressure on the maternal side, a father who die in a road accident in June 2024, he is the 2nd of a family of 4 children of which the youngest is epileptic with a notion of long-term biological inflammatory syndrome. There is no consanguinity of the parents.

This adolescent presented with flu-like symptoms in November 2023, followed by diarrhea, asthenia, and anorexia. After unsuccessful self-medication and the onset of abdominal pain, he consulted a first health center in early December 2023. His weight was 53 kg for a height of 1.60 m (body mass index 20.7), blood pressure 105/60 mmHg, and a fever of 38 ℃. An infectious pathology is suspected and a blood count was performed, which came back normal, and a search for plasmodium falciparum trophozoites in a peripheral blood smear (PBS) was negative. The renal function tests came back normal, and the liver function tests were disturbed with hepatic cytolysis syndrome. Abdominal ultrasound revealed significant diffuse intestinal aero-stercoral stasis, hepato-bilio-pancreatic and spleno-renal morpho-structure without abnormalities. He received antipyretic and antimalarial treatment with artesunate. On day 4 of hospitalization, he presented visual hallucinations, incoherent speech with psychomotor agitation which prompted a first referral. The parents consulted another health center. The agitation persisted requiring the introduction of injectable promethazine 50 mg x2/day. He manifested tonic -clonic convulsive seizures during the hospitalization with lateral biting of the tongue (first episode of this kind). The post-critical clinical examination revealed an alteration of the general condition, an examination of the digestive system and other systems without particularity. The brain CT returned normal. The urine toxicology assay performed came back positive for (ketamine, methylenedioxymethemphetamine, benzodiazepine), negative for (cocaine, cannabis, morphine, amphetamine, methamphetamine). The blood biology found an inflammatory syndrome with an erythrocyte sedimentation rate (ESR) at 40 mm, lymphopenia, a positive C - reactive protein, a negative PBS, a normal ionogram, a normal renal assessment, a syndrome of hepatic cytolysis and cholestasis. The hepatitis B, C, human immunodeficiency virus (HIV1 and HIV2) serologies were negative. He was put on clobazam 10 mg x2/day. Given the syndrome of hepatic cytolysis and cholestasis and the toxicology assessment appearing doubtful, encephalopathy associated with toxic hepatopathy was considered and he was referred to a psychiatric department for withdrawal.

During his stay in psychiatry, he persisted with an infectious syndrome at 38-39 ℃, visual hallucinations, and megalomaniacal delusions. The mother's additional interview later reported that during this hospitalization, he manifested paresthesias of the type of tingling of ascending evolution then a week later, a tetraparesis of ascending evolution which lasted a week complicated by dysphagia, tachycardia with elevated blood pressure figures. A second brain scan performed came

back normal, the biology found an ESR at 50 mm, a renal assessment, CRP negative, dosage of antistreptolysins (ASLO) negative, PBS negative. During hospitalization, treatment included diazepam 10 mg every 12 hours, metamizole 500 mg intravenous (IV) every 8 hours, enoxaparin 0.4 mg/day, omeprazole 40 mg/day, promethazine 50 mg/every 12 hours, vitamin B1 500 mg/day, vitamin B6 250 mg/day, gentamycin 160 mg for 5 days, ceftriaxone sulbatam IV 2g/day. The diagnosis was an infectious syndrome with elevated blood pressure complicating acute intoxication on a chronic background with ketamine and methylenedioxymethemphetamine. Given the unfavorable evolution, transfer to intensive care was indicated.

They consulted a third health center in early January 2024. During his non-medical transport, he developed dyspnea. He arrived there with respiratory distress that lasted 48 hours and required oxygen therapy. On examination, there was flaccid tetraparesis with a motor strength of 0/5 according to the Medical Research Council (MRC) Muscle Scale, abolished deep tendon reflexes (DTR) in all 4 limbs, tactile and pain hypoesthesia in all 4 limbs, regular heart sounds. The electrocardiogram of January 2024 noted a regular sinus tachycardia at 118 bpm. The pulmonary CT angiography returned normal. Magnetic resonance imaging of the spinal cord was normal. The kinetics of the liver assessment showed a cytolysis and cholestasis syndrome, an ESR at 65 mm, negative PBS. The neurological opinion concluded with Guillain-Barr ésyndrome, an electroneuromyogram (ENMG) was indicated as well as a lumbar puncture which was not honored. A bolus of 500 mg of intravenous methylprednisolone was performed for 3 days.

At the end of January 2024, they consulted a fourth health center where the diagnosis of Guillain-Barr é syndrome was also mentioned. Biology found an ESR at 85 mm, a liver function test still disturbed. Lumbar puncture found hyperproteinorachia at 1 g/l, normal glycorachia, and an absence of cells. He benefited from short-term corticosteroid therapy 500 mg of IV methylprednisolone for 3 days then a per os relay with prednisolone 50 mg in addition to azathioprine 50 mg x2/day and functional rehabilitation. The therapies were stopped by the patient after 1 month. The patient was lost to follow-up and seen again in March 2024. He reported paresthesias such as tingling and electric shocks in all 4 limbs and 2 more episodes of tonic - clonic seizures. On examination, there was tetraparesis with a motor strength of 3/5 in the upper limbs, 1/5 distally, 2/5 in the lower limbs, 1/5 distally, abolished osteotendinous reflexes in all 4 limbs, bilateral hypopalesthesia L2 to L5, preserved pain sensitivity, walking possible with human assistance and a cane. The EEG of March found EEG activity of non-structural generalized epilepsy. The ENMG (Tables 3 and 4, Figures 1 to 3) found a neurophysiological pattern of acute polyradiculoneuropathy in the recovery phase with collapsed motor amplitudes, conduction blocks, globally slowed motor conduction velocities, prolonged F-wave latencies, collapsed sensory amplitudes with slowed sensory conduction velocities. Gabapentin 300 mg x2/day was introduced in addition to functional rehabilitation sessions. At the end of May 2024, the adolescent was again hospitalized for 1 month in a health center in the interior of the country. This hospitalization was carried out at the request of a parent for better care. The investigations carried out revealed an accelerated ESR of 32 mm and hepatic cytolysis syndrome.

In September 2024, he was found to have 3+/5 tetraparesis in the upper limbs, 2/5 distally, 3/5 in the lower limbs, 2/5 distally, walking possible without a cane, and an ESR of 50 mm. Given this development at 10 months, the family history and the persistence of a biological inflammatory syndrome and associated neuropsychiatric manifestations, a systemic disease was considered and an indirect immunofluorescence assay of antinuclear antibodies was performed, which came back positive with a speckled appearance and a titer of 1/160. The specificities of anti-DNA and soluble antinuclear antibodies were not performed. Based on the criteria of the American College of Rheumatology with a score of 19 (fever: 2, thrombocytopenia: 4 leukopenia: 3, convulsion: 5, psychosis: 3, delirium: 2), the diagnosis of systemic lupus was retained and azathioprine 50 mg reintroduced at 50 mg/day after normalization of the liver function test. In January 2025, during the follow-up appointment, the patient did not show up, having regained almost complete autonomy, he would have become independent again since December 2024, walked without a cane and discontinued immunosuppressive treatment. In early May 2025, during a fever, he presented polyarthralgia, lower back pain and worsening of the deficit in the lower limbs to 3/5. At the end of May 2025, the examination noted bilateral steppage, predominantly distal tetraparesis, 4+ in the upper limbs, extension of the hand on the forearm at 3/5, 4+ paresis in the lower limbs, 2/5 in plantar flexion, DTR still abolished in all 4 limbs, no sensory deficit, the rest of the physical examination was unremarkable.

Table 1. *Patient's Main Blood Tests.*

	07-Dec	December 16	26-Dec	05-Jan	January 10	January 18	January 29	19- Feb	March 31	02 Sept	STANDARDS
AST	2.3 N	2.9 N		4.1 N	5.5 N	4.1 N	4 N	2.4 N	4 N		Less than 26, 38 or 37 IU/l
ALT	2.9 N	1.6 N		7.6 N	7.7 N	7.6 N	8.8 N	3.9 N	4.6 N		Below 29, 40 or

	07-Dec	December 16	26-Dec	05-Jan	January 10	January 18	January 29	19- Feb	March 31	02 Sept	STANDARDS
											41
GGT		1.6 N		3.5 N	2.5 N	3.5 N	4 N	Normal			[10-50]
PAL		1.4 N		2.9 N	1.0 N	2.9 N		1.7 N			Less than 280
Urea	0.28		0.40	0.3	0.22		0.36		0.28		[0.15-0.45g/l]
Creatinine	8.65	8	10	9	9		8		7		[07-14mg/ml]
D-dimers				953.55							N < 500 mg/ml
PBS	Negative	Negative	Negative	Negative							
CRP		96	< 5			< 5	< 5				N < 5 mg/l
ESR		40	50	65			85		32	50	< 20 mm
Blood sugar							1.08				0.7-1.10g/l
CPK							109				[24-190]
ASLO			<200								
CBC											
Hb	15.0	14		10.2	10.2		11.3	14.4			12-16g/dl
MCV	89.80	97.3		96	93		92.1	97.3			90-100 fl
MCHC	31.70	33.2		33.1	32		31.6	31.2			27-32 pg
Platelets	275000	264000		23100	98000		318000	264000			[150-400,000/mm^3]
Leukocytes	5400	3700		7656	5500		7300	3700			4000-10000/mm^3
Lymphocytes	1697	1034		696	1870		949	1147			1500-4000/mm^3
PNN	1955	2445		2500	3410		4820				1700-7000/mm^3
HIV serology, Hepatitis B, C		Negatives					Negatives		Negatives		
Ionogram		Normal									
Blood cultures						Negatives					
Multi-drug test		Positives									
CSF: Proteins							1.0 g/l				<0.5 g/l
Glucose							0.45g/l				0.50-0.70 g/l
Cytology							0 cells				0-5 cells
ANA (IFI)										1/160	< 1/160

Legend: AST: Aspartate amino- transferase, ALT: Alanine amino- transferase, GGT: Gamma- glutamyl transferase, CRP: C - reactive protein, PBS: peripheral blood smear, ESR: Sedimentation Rate, CBC: Complete Blood Count, CPK: Creatinine Phosphokinase ASLO: Antistreptolysin, Hb: Hemoglobin, MCV: Mean Cell Volume, MCHC: Mean Corpuscular Hemoglobin Content, PNN: Polymorphonuclear Neutrophils, CSF: Cerebrospinal Fluid, HIV: human immunodeficiency virus, ANA: Antinuclear Antibodies IFI: Indirect immunofluorescence on Hep 20-10 slides

Table 2. *Some cases of GBS as the initial manifestation of SLE.*

Citation (Actor/Year)	Age/Sex	Demonstration of the LES	Antibody	Kind of SGB	Treatment	Evolution	Background treatment
Uhunmwangho (2015)	26/F	Polyarthralgia; high blood pressure, proteinuria, lupus nephritis, generalized myxedema	ANA, dsDNA	Not realized	Cs per os, IV	Complete (Time not defined)	AZA, HQC
Boudanga (2023)	41/F	Generalized Alopecia Myxedema	ANA 1/1280 dsANA 300 IU/ml	AMAN	IVIg, CP (IV)	Complete	CS, HQC
Beshir (2021)	14/F	No symptoms before GBS	ANA 1/1280 in ANA 12 UI	AMAN	EP, (8), IG, IV, CS, IV, Rituximab	Complete	CS Oral
Yildiz (2015)	47/F	Alopecia, oral ulcer, anemia, leukopenia, thrombocytopenia,	ANA	AMAN	Ig IV, CS	Complete (14 months)	RTX, HQC
Wang (2024)	62/M	Malar rash, protein	ANA 1/160		MTX + intrathecal DXM	Not reported	Not reported
Fawzy (2022)	39/M	Arthralgia Epilepsy PRES syndrome	ANA, dsDNA ANA, Smith, antiSSa, Low complements		IV Ig, EP	Death	
Chaudhuri (1989)	40/M	Epilepsy, Leukopenia, Lupus nephritis	ANA in ANA	AIDP	EP, CS, CP	Partial (8 weeks)	
He (2025)	38/M	Malar rash, leukopenia, lupus pleurisy	ANA 1/1000 Rnp, Sm, Ro52, SSA, U1RNP, C3 low	AIDP	IVIg CS HCQ, MMF	Complete (18 months)	CS, HCQ, MMF
Present case	16/M	Psychosis, delirium, epilepsy, leukopenia psychosis	ANA	AIDP	CS, AZA	Partial (18 months)	AZA

LEGEND: ANA: Antinuclear antibodies; dsDNA: Anti-Double-Stranded DNA; Ig IV: Intravenous Immunoglobulin; HQC: Hydroxychloroquine; CS: Corticosteroids; RTX: Rituximab; AIDP: Acute Inflammatory Demyelinating Polyneuropathy; MMF: Mycophenolate mofetil; AMAN: neuroradiculopathy acute motor axonal syndrome; PE: plasma exchange; CP: Cyclophosphamide; RNP: Ribonucleoprotein; AZA: azathioprine; GBS: Guillain-Barr ésyndrome; SLE: Systemic lupus erythematosus; MTX methotrexate; DXM Dexamethasone SSA/ Ro: Soluble nuclear ribonucleoprotein A

4. Discussion

Neuropsychological manifestations of SLE or NPLSE are multiple neuropsychiatric manifestations directly linked to SLE [9]. There are 19 NPLSEs [10]. CNS manifestations include confusional syndrome, seizures, psychosis. PNS manifestations include mononeuropathy, plexopathy, GBS. NPSLE has a higher incidence in women, while the risk of seizures is more pronounced in men [9]. NPSLE is more common in African descendants and Asians compared to whites; however, the severity of NPSLE is more significant in the white race. Neuropsychiatric manifestations occur in the early stages of SLE and represent between 39 and 50% of patients with this disease [9]. In Cameroon, neuropsychiatric manifestations were inaugural of the disease in 37.0% of cases within the lupus population [11]. Seizures are common manifestations of NPLSE (7-20%) while GBS (0.08-1.2%) is rare [9].

In this adolescent, psychosis, convulsions and delirium were considered as manifestations of a withdrawal syndrome, but in reality they were neuropsychological manifestations of SLE. This observation is thus distinguished by diagnostic wandering, the repetition of additional examinations in search of an infectious etiology (6 searches for plasmodium falciparum all negative, 3 HIV, hepatitis C and hepatitis B serologies also negative, blood cultures negative). Thus the infectious etiology was always systematically tracked, with each time, an unsuccessful search. He also benefited from antimalarial and antibiotic treatment. In the exploration of the syndrome of hepatic cytolysis and cholestasis, the liver and biliary tract ultrasound was normal. Liver damage can also be seen in SLE in the form of disturbance of liver biological tests (cytolysis

or cholestasis greater than 4N) or abnormal liver histology [12].

Schematically, an alteration of the general condition preceded epileptic seizures and then GBS. The disturbance of liver enzymes throughout the evolution for 4 months was considered as an infectious but especially toxic hepatopathy despite an unacknowledged context of alcoholic intoxication or psychoactive substances. This initial positive multi-drug test was another element that confused the clinicians. In retrospect, the critical analysis of this multi-drug test shows us that it is a false positive test, that it is not ketamine but phencyclidine dosed in urine tests which is a dissociative agent structurally related to ketamine. In clinical practice, a false-positive urine drug screen can be caused by many xenobiotics: dextromethorphan, diphenhydramine, doxylamine, ibuprofen, imipramine, ketamine, meperidine, venlafaxine, bupropion, methylenedioxpyrolvalerone, and tramadol [13]. The adolescent self-medicated and is likely to have used ibuprofen and tramadol. In addition, promethazine, which is used in hospital, is part of a group of drugs called phenothiazines [14]. It can be used to relieve nausea and vomiting. It was used in this case as a hypnotic. Promethazine can cause false-positive urine drug screens for amphetamines or methamphetamines [14].

This observation highlights the problem of a rare disease with extremely rare manifestations in a country with limited technical facilities. First, the rarity of lupus itself and the rarity of its hepatic and neurological manifestations. The other problem is medical nomadism (stay in 6 health centers including two referral centers). The lumbar puncture was feared by the parents, finally performed with an albumin-cytological dissociation, serum/CSF isoelectrofocusing was not available. A crisis of confidence in the healthcare staff set in, justified by the label of a child consuming psychoactive substances that was stuck on their child, when in reality he was consuming energy drinks with the dream of becoming a professional footballer. This false label of a teenager addicted to psychoactive substances pursued him for more than a month, and this is why the referral from the psychiatric service to an intensive care unit was not honored because the parents thought that this diagnosis would be pursued and they then preferred to continue the hospitalization elsewhere. The tetraplegia was considered as generalized hypotonia resulting from a withdrawal syndrome, the episode of dyspnea was taken as a pulmonary embolism in this context of immobilization. It was only after a window of sedative drugs and by re-interviewing the family who confirmed a flaccid tetraplegia of ascending evolution over a few days that GBS was for the first time mentioned.

The occurrence of GBS in the patient was the event that changed the diagnostic approach. Based on the Brighton criteria [15], the patient presented the highest level of diagnostic certainty for GBS. The patient had symmetrical flaccid paralysis of the limbs, abolition of tendon reflexes and a monophasic course (peaking at 2 weeks). Cerebrospinal fluid showed albumin -cytological dissociation and ENMG revealed demyelination and axonal degeneration of the peripheral nerves of the limbs. Respiratory involvement was also observed. Differential diagnoses of chronic inflammatory demyelinating polyneuropathy (monophasic course without recurrence), myelitis (no sensory level, no sphincter disorder), metabolic/toxic/infectious neuropathies (no corresponding medical history, negative serological and etiological tests) were excluded. In adolescents, GBS was severe as reported several times in the literature [3]. Severe GBS is characterized by rapidly evolving limb weakness, dysphagia, respiratory distress requiring respiratory support, and severe autonomic dysfunction (malignant arrhythmias, labile hypertension, or hemodynamic collapse) [2]. ENMG at 2 months of flaccid tetraplegia demonstrated a pattern of acute inflammatory demyelinating polyneuropathy in the recovery phase. This is the most frequently reported electrophysiological form in SLE [16]. According to He et al, the presence of axonal lesions on electromyography increases the likelihood of severe disease development [3]. The occurrence of SLE complicated by severe GBS is rare, with severe manifestations involving respiratory failure being uncommon. He et al [3] in their systematic review of the literature covering cases reported since the seminal description of severe SLE-GBS with respiratory failure (period 1999-2024), identified 13 severe cases. In our case, seizures preceded GBS. Two cases of GBS have been reported with epilepsy in the literature. The first was under investigation when the patient's neurological status worsened with increasing asthenia and presented with tonic-clonic seizures [17]. In the second case, GBS was subsequently associated with PRES syndrome and their outcome despite optimal treatment was unfavorable [18].

Our observation nevertheless has some shortcomings: no dosage of the specificities of antinuclear antibodies (ANA), complements C3 and C4, anticardiolipin antibodies, antiganglioside antibodies because the explorations were the responsibility of the adolescent's family and due to the local technical platform limited the additional examinations. In the literature review of Coomes et al, the majority of patients with positive ANA were associated with another antibody, the speckled appearance on indirect immunofluorescence predominated followed by the homogeneous appearance [16]. Two cases of low ANA titer at 1/160 in the literature: in Wang et al, the antiribonuclear antibodies were positive [19], in Yildiz et al, the specificities of the ANA antibodies came back negative [20]. Another case in Wang with an average titer of 1/640, there was no other associated antibody [19]. The transient worsening of our patient's neurological condition in the context of discontinuation of background treatment with fever suggests an SLE flare-up, which is another argument supporting the diagnosis of SLE.

In the treatment of classic GBS, corticosteroids are not recommended: intravenous methylprednisolone does not improve neurological status and its association with intravenous immunoglobulin's is not beneficial. Oral corticosteroids may delay recovery or cause side effects [2]. However, in the specific clinical context of SLE comorbid with GBS, this treatment logic should be reexamined [3]. He et al advocate that

the possibility of SLE should be considered for GBS patients without clear triggering factors, young women, or those with an immune-related or family history [3] and that SLE should be part of the differential diagnosis of GBS because conventional therapies in GBS may not be adequate to treat SLE-GBS. According to current guidelines [21], when neuropsychiatric symptoms are highly correlated with SLE activity or specific neuropathies present with notable inflammatory features (axonal lesions), high-dose pulse corticosteroid therapy (500-1000 mg/day) should be used as the primary treatment. In severe cases [3], all patients received pulse corticosteroid therapy during acute disease progression. In some cases, efficacy was not significant despite early association with IV Ig, requiring the addition of immunosuppressants such as cyclophosphamide and azathioprine. He et al propose a hypothesis to be confirmed by multicenter randomized controlled studies: in comparison with the demyelinating lesions of peripheral nerves observed in classical GBS, SLE-GBS could involve a specific mechanism, mainly characterized by complement activation and immune complex deposition. The combined use of corticosteroids and immunosuppression may help manage the acute phase and reduce long-term risks by blocking T/B cell activation and cytokine storms.

Methylprednisolone were used and later azathioprine 50 mg/day was introduced. In Nigeria, Uhunmwangho et al [6] used Aza 50 mg. We did not have IV Ig or plasma exchanges which were also beyond the financial reach of the parents. In the literature [16], cyclophosphamide, rituximab, azathioprine were the most used immunosuppressants. Synthetic antimalarials (hydrochloroquine) were often associated with the treatment. Gabapentin for neuropathic pain in Beshir was also used in adolescents with good progress [22].

The 18-month evolution of GBS noted a partial recovery. Severe cases generally had a complete recovery (evolution achieved at 24 months) [3].

5. Conclusion

This observation reminds us that systemic diseases exist in Togo and coexist with infectious, toxic, metabolic, and cardiovascular diseases. The initial manifestations of these systemic diseases can completely confuse the clinician. Although rare, GBS can be the mode of revelation of SLE. This SLE-GBS association is often serious and can be life-threatening. In a context of limited technical platform, faced with GBS with dysautonomic signs, the persistence of a biological inflammatory syndrome associated with other neuropsychiatric manifestations and a family context of suspected autoimmune disease, we propose the systematic search for SLE. The treatment of this particular form of GBS is not very well codified but the cases reported in the literature use corticosteroids and immunosuppressants with good evolution, drugs available in sub-Saharan Africa.

Abbreviations

SLE	Systemic Lupus Erythematosus
GBS	Guillain-Barré Syndrome
CNS	Central Nervous System
PNS	Peripheral Nervous System
IV Ig	Intravenous Immunoglobulin
PBS	Peripheral Blood Smear
CT	Computed Tomography
DTR	Deep Tendon Reflexes
CRP	C reactive Protein
HIV	Human Immunodeficiency Virus
ENMG	Electroneuromyogram
EEG	Electroencephalogram
ESR	Erythrocyte Sedimentation Rate
ANA	Antinuclear Antibodies
DNA	Deoxyribonucleic Acid
NPLSE	Neuropsychological Manifestations of Systemic Lupus Erythematosus
Aza	Azathioprine
CSF	Cerebrospinal Fluid

Author Contributions

Adama Mawulikplimi Ephoevi-ga: Conceptualization, Data curation, Funding acquisition, Methodology, Resources, Writing – original draft

Kokou Mensah Guinhouya: Funding acquisition, Validation, Writing – review & editing

Lehleng Agba: Funding acquisition, Validation, Writing – review & editing

Komla Nyinevi Anayo: Funding acquisition, Validation

Bitankade Kabassem: Funding acquisition, Validation

Kossivi Apetse: Funding acquisition, Validation, Visualization

Komi Assogba: Funding acquisition, Visualization

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Mofou Belo: Funding acquisition, Visualization

Agnon Koffi Balogou: Funding acquisition, Visualization

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare no conflicts of interest.

Appendix

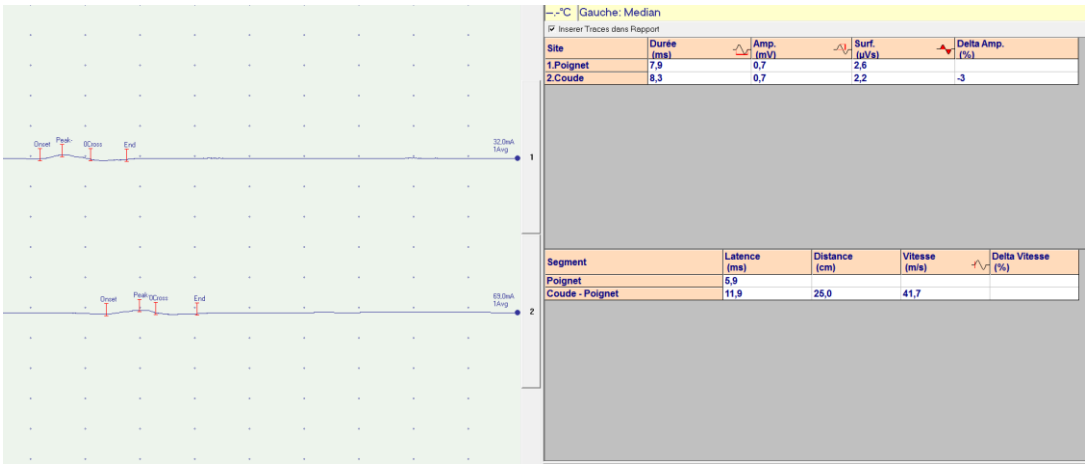

Figure 1. *Right median motor nerve conduction showing conduction block.*

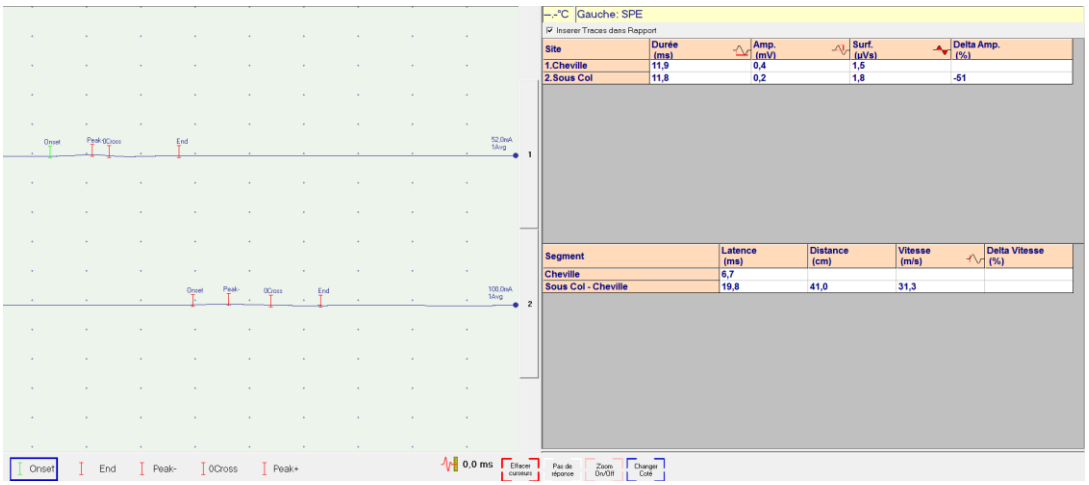

Figure 2. *Left fibular motor nerve conduction showing conduction block.*

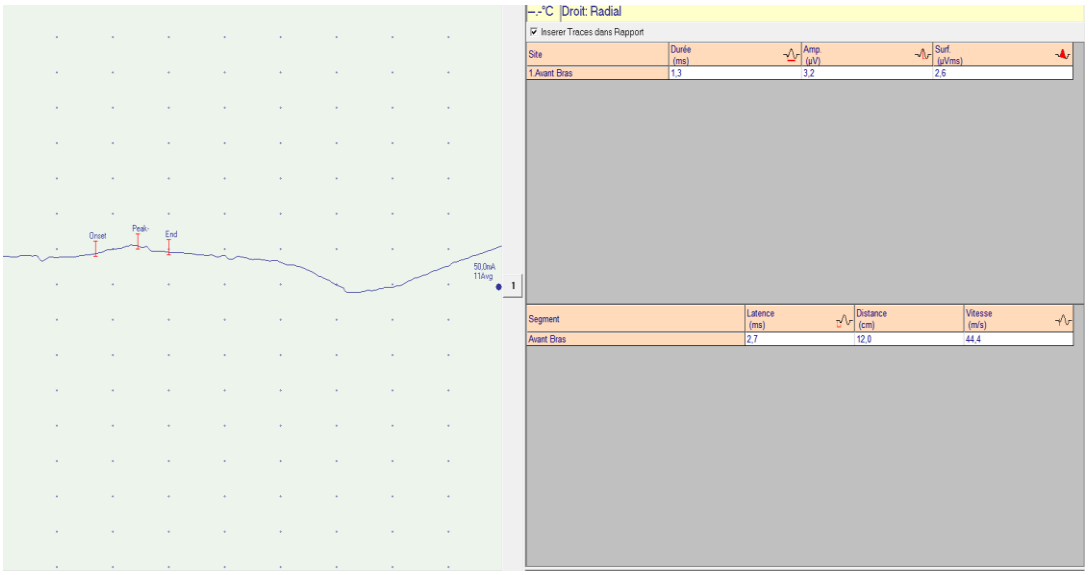

Figure 3. *Left ulnar sensitive nerve conduction showing collapse of amplitudes.*

Table 3. *Electroneuromyographic examination of motor conduction showed collapsed motor amplitudes, conduction blocks, globally slowed motor conduction velocities.*

Motor nerve conduction			
Nerves	**Distal latency (ms)**	**Amplitude (mV)**	**Speed (ms)**
Right median	4.0	1.7	42.6
Left median	5.9	0.7	41.7
Right ulnar	3.3	1.2	43.8
Left ulnar	3.9	2.8	46.0
Right common peroneal	6.7	0.4	31.3
Left common peroneal	8.8	No potential	28.6
Right tibial	8.7	3.1	31.3
Left tibial		3.8	

Table 4. *EMMG examination: sensory conduction showed collapsed sensory amplitudes with slowed sensory conduction velocities.*

Sensory nerve conduction (orthodromic pathway)			
Nerves	**Distal latency (ms)**	**Amplitude (mV)**	**Speed**
Right radial	2.7	3.2	44.4
Left radial	2.2	6.3	54.5
Right median	24	16.2	50.0
Left median	21	18.6	57.1
Right ulnar	24	8.2	37.5
Left ulnar	21	18.4	57.1
Right sural	40	3.5	30.0
Left sural		No potential	

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Research Field

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