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Clinical features of Guillain-Barré syndrome and chronic inflammatory demyelinating polyradiculoneuritis associated with SLE

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ABSTRACT

Objective We report on the clinical characteristics, treatments and outcomes of Guillain-Barré syndrome (GBS) and chronic inflammatory demyelinating polyradiculoneuritis (CIDP) associated with SLE.

Methods Patients treated at Peking Union Medical College Hospital between January 2004 and November 2021 who fulfilled the diagnostic criteria for SLE and GBS/CIDP (n=9) were included. Clinical presentations, lab results, treatment regimens and prognoses were retrieved and analysed.

Results Six patients were diagnosed with SLE and GBS, while three were diagnosed with SLE and CIDP, with the average age at diagnosis of 38.6±18.2 years. SLE disease duration ranges from 1 week to 36 years, and the courses of GBS and CIDP range from 1 week to 2 months and from 2 months to 15 months, respectively. All patients exhibited either or both limb paresthesia and weakness, other neurological symptoms include dysphagia, peripheral facial nerve palsy and respiratory and cardiac arrest. The median cerebral spinal fluid white blood cell count and protein level were 0.002×10^9/L ($0–0.006\times10^9$/L) and 0.79 g/L (0.57–7.09 g/L), respectively. All patients received glucocorticoid and immunoglobulin therapy. Seven patients received cyclophosphamide, and seven patients received intrathecal injections of methotrexate and dexamethasone. Two patients had complete resolution, five experienced marked improvements and two failed to improve with treatments.

Conclusion SLE-associated GBS/CIDP may manifest regardless of disease systemic activity. Clinical features may differ from that of pure GBS/CIDP, and treatment often requires immunosuppressants, making differential diagnosis crucial, especially for patients with GBS/CIDP presenting as the first manifestation of SLE.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Guillain-Barré syndrome (GBS) and chronic inflammatory demyelinating polyradiculoneuritis (CIDP) are rare but serious complications of SLE, previously characterised primarily based on case reports.

WHAT THIS STUDY ADDS

⇒ Among nine patients with SLE-associated GBS/CIDP, neurological symptoms were diverse and irrespective of SLE systemic activity. Cytoalbuminologic dissociation was apparent in all cases, and all tested antiganglioside antibodies were negative. Treatment included corticosteroid pulse therapy, cyclophosphamide and intrathecal injections in addition to classic GBS/CIDP treatment regimes, and the prognosis is generally favourable.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study adds to the current knowledge of the clinical characteristics of the disease and suggests that clinical features and treatment may differ from that of pure GBS/CIDP, calling for more research on the topic.

INTRODUCTION

SLE is a chronic inflammatory autoimmune disease that may affect various important organs or systems including the kidneys, the haematological system, the nervous system, etc, significantly affecting patients' prognosis and quality of life.[1] Neuropsychiatric SLE (NPSLE) is a common but potentially devastating complication of SLE, with literature reporting an occurrence varying from 21% to 95%.[2] A total of 19 neuropsychiatric syndromes were described according to the American College of Rheumatology (ACR) nomenclature and case definitions from 1999, which could be subclassified into 12 central and 7 peripheral manifestations.[3]

Common presentations of NPSLE among Chinese patients include seizures, acute confusional state and cerebrovascular disease, which were found in 33.6%, 25.3% and 15.5% of patients, respectively.[4] On the other hand, acute inflammatory demyelinating polyradiculoneuritis, the most common subtype of Guillain-Barré syndrome (GBS), was seen in only 1.5% of NPSLE patients. Chronic inflammatory demyelinating polyradiculoneuritis

(CIDP) may be an even more uncommon presentation, with a previously reported incidence of 0.2%.[5] Despite the low occurrence, GBS and CIDP are serious complications of SLE that warrant further investigation. Few studies have previously reported on the clinical characteristics and outcomes of SLE-associated GBS and CIDP based on a small sample size or review of published case reports.[5 6]

This study reports on the clinical characteristics of GBS and CIDP in a retrospective cohort of nine SLE patients from Peking Union Medical College Hospital (PUMCH), intending to improve understanding of the disease, provide clues for early diagnosis, as well as improve patient prognosis.

METHODS

Participants and data collection

Patients treated at PUMCH between January 2004 and November 2021 who fulfilled the diagnostic criteria for SLE and GBS/CIDP with available inpatient medical records were included in the study. Medical records were retrieved from the department of medical records at PUMCH. SLE was diagnosed according to the 1997 ACR classification criteria or the 2012 Systemic Lupus International Collaborating Clinics classification criteria for SLE.[7 8] GBS was diagnosed based on the criteria developed by the National Institute of Neurological and Communicative Disorders and Stroke in 1978 and updated in 1990.[9] CIDP was diagnosed based on the 2005 European Federation of Neurological Societies/Peripheral Nerve Society Guideline.[10] Patients with other known causes of peripheral neuropathy, such as primary disease of the nervous system, infections, medications, etc, were excluded from the study.

Study design

Anonymous clinical data were obtained from the participants during their hospital stays. Eligible patients diagnosed with SLE and GBS/CIDP were retrospectively recruited into the cohort. Clinical presentations, lab results, treatment regimens and prognoses were collected and analysed.

Statistical analysis

Data analyses were performed using SPSS V.22.0. Descriptive statistics were used to analyse baseline characteristics. Categorical variables are expressed as proportions (%) and continuous variables are expressed as mean (M)±SD or median (range).

RESULTS

Demographics and basic characteristics

From the 6870 patients diagnosed with SLE during the study period, a total of nine eligible patients were enrolled in the study (table 1), of whom seven were female (77.8%), and the average age at diagnosis was 38.6±18.2 years. Among these patients, six cases were diagnosed with SLE and GBS, while three cases were diagnosed with SLE and chronic inflammatory demyelinating polyradiculoneuropathy. The disease duration of SLE ranges from 1 week to 36 years, with a median duration of 5 months. The course of GBS ranges from 1 week to 2 months, while the course of CIDP ranges from 2 months to 15 months. The median disease durations for GBS and CIDP were 1 month and 3 months, respectively.

Clinical manifestations and laboratory examination features

With regard to clinical presentations, six patients (66.7%) had both paresthesia and weakness in the bilateral limbs, two patients (22.2%) had limb weakness, while one patient (11.1%) had limb paresthesia. Other clinical symptoms include dysphagia (two patients), peripheral facial nerve palsy (two patients) and respiratory and cardiac arrest (one patient) (table 2). Besides the classic symptoms of GBS and CIDP, other NPSLE manifestations include generalised tonic–clonic seizures (one patient), and EMG evidence of CNS involvement (one patient). Non-neurological manifestations included alopecia (three patients), oral ulcers (two patients), rash (two patients), joint involvement (four patients), anaemia

Table 1 Demographics and basic characteristics

Case no.	Gender	Age (years)	Peripheral neuropathy	SLE disease duration	GBS/CIDP disease duration	Interval from SLE to GBS/CIDP
1	Female	29	GBS	5 months	2 weeks	4 months
2	Female	44	GBS	14 years	2 weeks	14 years
3	Male	62	GBS	1 week	1 week	Initial presentation
4	Female	36	CIDP	6 years	3 months	6 years
5	Female	15	GBS	2 months	2 months	Initial presentation
6	Female	50	CIDP	4 years	2 months	4 years
7	Female	21	GBS	1 month	1 month	Initial presentation
8	Male	24	GBS	1 month	1 month	Initial presentation
9	Female	66	CIDP	36 years	15 months	35 years

CIDP, chronic inflammatory demyelinating polyradiculoneuritis; GBS, Guillain-Barré syndrome.

Table 2 Clinical presentation and SLEDAI scores

Case no.	Neurological presentation	Other systemic presentations	SLEDAI Score (on admission)
1	Limb weakness, plantar hyperalgesia	Alopecia, oral ulcers, arthritis, proteinuria (2.74 g/24 hours)	14
2	Limb weakness and paresthesia, radicular pain in upper limbs, dysphagia	Leucopenia, rash	1
3	Limb weakness, facial paralysis, respiratory and cardiac arrest	Rash, proteinuria (1.0 g/L)	16
4	Limb weakness and paresthesia, facial paralysis, dysphagia	Alopecia, oral ulcers, joint pain, anaemia, nephrotic syndrome	7
5	Limb weakness and paresthesia, generalised tonic–clonic seizures	Thrombocytopenia, anaemia	9
6	Weakness in the proximal limbs, stocking-glove paresthesia	Arthritis, myositis, proteinuria (0.41 g/24 hours), thrombocytopenia	4
7	Limb paresthesia and itching, pain in distal limbs	Alopecia, proteinuria (0.3 g/L), leucopenia	2
8	Weakness in the lower limbs, numbness in the lateral side of the right leg, radicular pain in the back	proteinuria (0.7 g/24 hours)	6
9	Weakness in the proximal limbs	Arthritis, leucopenia, proteinuria (0.86 g/24 hours)	2

SLEDAI, Systemic Lupus Erythematosus Disease Activity Index.

(two patients), leucopenia (three patients), thrombocytopenia (two patients), nephrotic syndrome (one patient), proteinuria (six patients) and myositis (one patient). The median Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) score was 6, ranging from 1 to 16.

Table 3 Lab results and electromyography

Case no.	Cerebral spinal fluid			ANA	Other autoantibodies	Electromyography
	WBC (10^9/L)	PRO (g/L)	antiganglioside antibodies			
1	0.002	0.71	NEG	Homogeneous 1:320	Anti-dsDNA, anti-SSA, anti-SSB, anti-ribP, antinucleosome	Neurogenic lesion, SEP of the right lower limb, showed central damage above T12
2	0.002	0.79	NEG	Homogeneous speckled 1:320	Anti-dsDNA, anti-SSA, anti-ribP, antihistone, AMA-M2	Neurogenic lesion, abnormal SSR in lower limbs
3	0.005	7.09	NEG	Speckled 1:160	Anti-ribP	Unavailable
4	0	1.23	Unknown	Speckled 1:160	Anti-Sm, anti-RNP	Neurogenic lesion, myogenic changes (active phase)
5	0	1.23	Unknown	Speckled 1:1280	Anti-Sm, anti-U1RNP, ACL	Neurogenic lesion (primarily motor fibres)
6	0	0.57	Unknown	Speckled 1:640	–	Neurogenic lesion (motor fibres)
7	0.006	0.79	Unknown	Speckled 1:320	Anti-RNP, anti-ribP, anti-SSA, anti-Ro52, AMA-M2	Neurogenic lesion (primarily sensory fibres)
8	0	1.90	NEG	Dense fine speckled 1:160	Anti-Ro52, anti-β2GP1-IgM	No definite neurogenic or myogenic lesions, prolonged F-wave latency
9	0.004	0.72	NEG	Homogeneous 1:640	Anti-dsDNA, anti-SSA, anti-U1RNP, AMA-M2, antinucleosome, antihistone	Neurogenic lesion (motor fibres), motor conduction block

NEG, negative; PRO, protein; SEP, somatosensory evoked potentials; SSR, sympathetic skin response; WBC, white blood cell count.

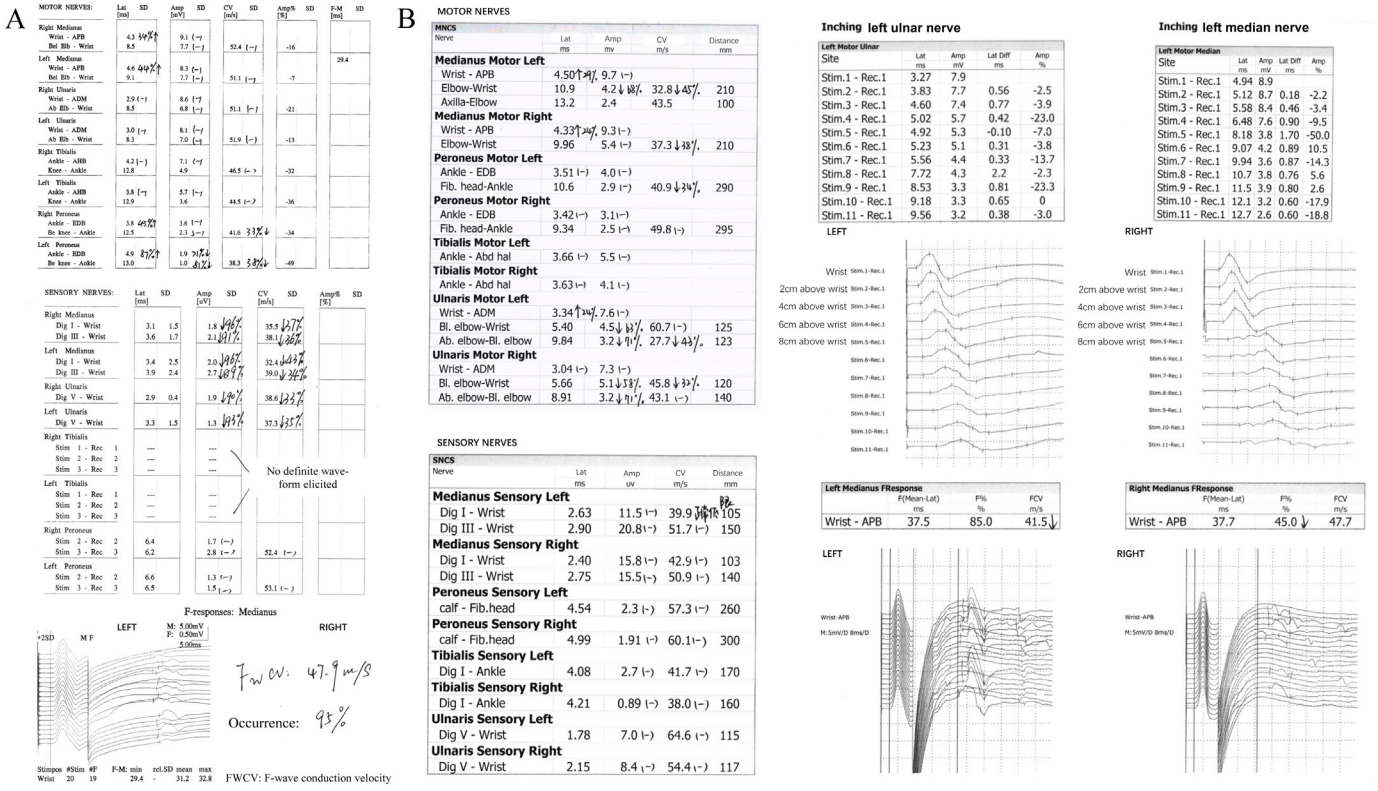

Figure 1 Electrophysiological studies of Guillain-Barré syndrome (GBS) and chronic inflammatory demyelinating polyradiculoneuritis (CIDP) patients. (A) Nerve conduction studies of a GBS patient (case 2) show prolonged distal motor latency, decreased motor nerve conduction velocity, decreased amplitude of sensory nerve action potential and prolonged F-wave latency. (B) Nerve conduction studies of a CIDP patient (case 9) show increased distal latency and decreased conduction velocity primarily in motor nerves. Inching studies reveal conduction blocks in both ulnar and median nerves. Conduction velocity and occurrence rate of F-waves were decreased. Needle electromyography results are consistent with neurogenic impairment in these patients (not shown here). ADM, abductor digiti minimi; AHB, adductor hallucis brevis; APB, abductor pollicis brevis; CV, conduction velocity; EDB, extensor digitorum brevis; FCV, F-wave conduction velocity; F-M, F-M interval=F-wave latency – M-wave latency.

The median white blood cell count in the cerebral spinal fluid (CSF) was 0.002×10^9/L, with a range of 0–0.006×10^9/L. The median CSF protein level was 0.79 g/L, ranging from 0.57 to 7.09 g/L. Serum and CSF antiganglioside antibodies were tested and were all negative in five patients. In terms of electromyography, neurogenic lesions were apparent in most cases, involving motor or sensory fibres, or both (table 3) (figure 1). MRIs of the brain were unremarkable in all nine patients. The antinuclear antibody pattern was speckled in five patients, homogeneous in two patients, homogeneous and speckled in one patient and dense fine speckled in one patient. Four patients also tested positive for anti-rRNP antibodies, while four patients tested positive for anti-U1RNP antibodies.

Treatments and outcomes

With regard to treatment, four patients (44.4%) received high-dose methylprednisolone pulse therapy, four patients (44.4%) received high-dose oral glucocorticoid treatment and one patient (11.1%) received low-dose prednisone maintenance therapy (5–7.5 mg once a day). Seven patients (77.8%) received oral or intravenous cyclophosphamide. All patients (100%) received intravenous immunoglobulin (Ig) therapy, with one patient (11.1%) receiving two rounds of intravenous Ig treatment. Seven patients (77.8%) received at least one round of intrathecal injection of methotrexate and dexamethasone. Clinical outcomes were determined based on comprehensive clinical assessments by a neurologist and a rheumatologist, as well as patient self-reports. One patient's (11.1%) disease had a self-limiting course, one patient (11.1%) achieved complete resolution with treatment, five patients (55.6%) had marked improvement of symptoms after treatment and two patients (22.2%) did not improve with treatments (table 4).

DISCUSSION

In this study, we described the basic characteristics, clinical features and treatment outcomes in a cohort of nine patients with SLE complicated with inflammatory demyelinating polyradiculoneuritis. GBS is a rare autoimmune disorder usually presenting as rapid-onset flaccid paralysis and numbness, with the highest incidence in patients aged 50–70 years.[11] CIDP has a progressive or relapsing-remitting course over 8 weeks, most common in patients between 40 and 60 years old.[12] GBS and CIDP

Table 4 Treatment and outcomes

Case	intravenous Ig	Glucocorticoid	Immunosuppressant	Intrathecal	Outcome
1	20 g once a day for 5 days	Methylprednisolone 1 g intravenously once a day for 3 days	Cyclophosphamide	Dex 10 mg+MTX 10 mg times three cycles	Complete resolution
2	20 g once a day for 5 days	Prednisone 5/7.5 mg once a day	–	–	Self-limiting course, complete resolution
3	20 g once a day for 5 days	Methylprednisolone 80 mg once a day	–	Dex 10 mg+MTX 10 mg times one cycle	No marked improvement
4	20 g once a day for 5 days	Prednisone 60 mg once a day	Cyclophosphamide	Dex 10 mg+MTX 10 mg times two cycles	No marked improvement
5	20 g once a day for 5 days	Methylprednisolone 1 g intravenously once a day for 3 days	Cyclophosphamide	Dex 10 mg+MTX 10 mg times two cycles	Marked improvement
6	20 g once a day for 5 days×2cycles	Methylprednisolone 48 mg once a day	Cyclophosphamide	–	Marked improvement
7	20 g once a day for 5 days	Methylprednisolone 1 g intravenously once a day for 3 days	Cyclophosphamide	Dex 10 mg+MTX 10 mg times one cycle	Marked improvement
8	25 g once a day for 5 days	Methylprednisolone 1 g intravenously once a day for 3 days	Cyclophosphamide	Dex 10 mg+MTX 10 mg times one cycle	Marked improvement
9	20 g once a day for 5 days	Methylprednisolone 80 mg once a day	Cyclophosphamide	Dex 10 mg+MTX 10 mg times three cycles	Marked improvement

Dex, dexamethasone; Ig, immunoglobulin; MTX, methotrexate.

are polyneuropathies due to demyelination, whose mechanistic associations with SLE are not entirely clear, but most likely have an immunological basis.[13] Most patients in our cohort were female, due to the predominantly female demographic of SLE, and though not significant, GBS seemed to afflict younger patients than CIDP.

All patients experienced the classic symptoms of either paresthesia or weakness in the bilateral limbs. Cytoalbuminologic dissociation was apparent in all cases, as in those with pure GBS. High CSF protein levels in GBS patients were reported to be associated with greater disease severity.[14] One of our cases had a CSF protein level of 7.09 g/L, and the patient experienced severe complications with no marked improvements after treatment. However, due to insufficient sample size, we could only speculate that the correlation holds for GBS associated with SLE. On the other hand, while previous statistics suggest that CSF antiganglioside antibodies were positive in approximately half of GBS and CIDP patients,[15] the antibodies were negative for all tested patients in our cohort. Existing studies are contradictory regarding the role of antiganglioside antibodies in the diagnosis of NPSLE,[16–18] and previous cases of SLE-related GBS or CIDP did not report on antiganglioside antibodies.[5 19] Therefore, it remained unclear whether antiganglioside antibodies are useful markers in the diagnosis of SLE-related GBS and CIDP.

Although a diagnosis of GBS or CIDP is not difficult to make based on clinical symptoms and test results, more in-depth investigations may be needed to look for signs of systemic illness such as SLE. In our cohort, four of the six GBS patients (cases 3, 5, 7 and 8) had GBS as the initial presentation of SLE, with varying severities of general symptoms, including proteinuria, rash, alopecia, anaemia, leucopenia and thrombocytopenia. The systemic symptoms may be subtle and non-specific, hence detailed history and further testing were particularly crucial. Meanwhile, our cohort also demonstrates that GBS or CIDP may develop years, even decades, after initial SLE diagnosis (cases 2, 4, 6 and 9), with only mild-to-moderate systemic activity based on SLEDAI scores. Several cases of GBS and CIDP as the initial presentation of SLE or without systemic activity have been reported in the literature, suggesting that high disease activity is not required for the development of GBS and CIDP.[20–22]

The current first-line therapy for GBS includes intravenous Ig and plasmapheresis, while treatment for CIDP usually includes corticosteroids and intravenous Ig.[23 24] For SLE-related GBS and CIDP, treatment for primary disease should also be initiated in addition to first-line therapy for GBS and CIDP. A previous systemic review has suggested that most SLE-related GBS patients had a favourable response to cyclophosphamide, which is not effective for patients with pure GBS.[25] Among the six GBS patients in our cohort, four were treated with

corticosteroid pulse therapy, with a generally favourable outcome. One GBS patient (case 2) had mild systemic disease activity and self-limiting GBS course at the time of treatment, hence did not require pulse therapy. The other GBS patient (case 3) had respiratory and cardiac arrest on transmission to our centre and had multiple complications including pneumonia and sepsis, hence intravenous pulse therapy was not administered at the time. Additionally, intrathecal injections of dexamethasone and methotrexate, which could potentially benefit NPSLE patients, were administered empirically as local treatment for radicular inflammation.[26] Therefore, in general, pulse therapy and intrathecal injections may be beneficial and should be attempted given no contraindications, but further investigations are required to determine the optimal treatment.

In conclusion, GBS and CIDP are rare but serious complications of SLE, which may manifest regardless of the systemic activity of the primary disease and may have various presentations. Clinical test features may differ from that of the pure form of GBS and CIDP, warranting further studies on how to distinguish them effectively. Treatment of SLE-related GBS and CIDP also differs from pure GBS and CIDP, often requiring immunosuppressants and corticosteroid pulse therapy, making differential diagnosis crucial for initiating treatment, especially for patients with GBS or CIDP presenting as the first manifestation of SLE.

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