Acute parkinsonism in patients with systemic lupus erythematosus: a case report and review of the literature

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ABSTRACT

Purpose of the study: Parkinsonism in patients with systemic lupus erythematosus (SLE) is rare. This study reported a case of parkinsonism in SLE and reviewed the clinical features and outcomes of parkinsonism in SLE patients.

Methods: English language literature of parkinsonism in SLE patients was reviewed.

Results: There were 28 patients (19 adults and 9 children) with SLE and parkinsonism. Twenty-three patients were female. Of 26 patients whose disease duration was available parkinsonism occurred at SLE diagnosis and after SLE diagnosis in 6 and 20 patients, respectively. Twenty-five patients had active SLE. Hematologic, mucocutaneous and musculoskeletal systems were the 3 most common organs involved in SLE during parkinsonism onset. Rigidity, bradykinesia and resting tremor were the 3 most common parkinsonian symptoms. Compared with adults, child cases had significantly more psychosis (4 in 9 vs. 1 in 19, p = .026), seizures or psychosis (6 in 9 vs. 2 in 19, p = .005) and mutism (6 in 9 vs. none, p < .001). Brain magnetic resonance imaging (MRI) was abnormal in 13 of 24 patients. Eight of nine patients had abnormal single-photon emission computed tomography (SPECT) and 5 and 3 showed hypoperfusion and hyperperfusion, respectively. The outcomes were resolution, partial response and persistent symptoms in 17, 7 and 4 patients, respectively. The outcome was no different whether or not dopamine therapy was included to corticosteroids and/or immunosuppressive drugs.

Conclusions: Parkinsonism in SLE usually occurs during active SLE disease. Good response to corticosteroid and/or immunosuppressive drugs supports the immunologic mechanism in the pathogenesis.

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Introduction

Various neuropsychiatric SLE (NPSLE) manifestations are well recognized, and can range from mild moods and anxiety disorders to severe manifestations, including seizure and psychosis [1]. However, the only two cardinal features of this disease are seizures and psychosis, which are included in the SLE classification criteria [2]. Movement disorders are a rare manifestation of SLE. A recent review of 17 studies found a pool estimate of NPSLE and movement disorder prevalence of 56 and 1%, respectively [3]. Among movement disorders, chorea was the most commonly seen at 2% prevalence in SLE/anti-phospholipid syndrome (APLS) [4]. Other movement disorders included ataxia, hemiballism, myoclonus, torticollis, blepharospasm and dystonia also described occasionally [5–10]. Parkinsonism is very rare; as reported in only 1 of 37 pediatric and 1 of 41 adult SLE patients with central nervous system (CNS) involvement [6,7]. Furthermore, only chorea was listed as a movement disorder in the 1999 American College of Rheumatology (ACR) nomenclature for NPSLE, and reports mentioned that Parkinson’s disease and hemiballism were considerably rare [1].

This study reported a female SLE patient who developed acute parkinsonism during her 3rd NPSLE episode; and previously reported cases of parkinsonism in SLE patients also were reviewed. This study was performed in accordance with the ethical standards of the institutional and/or national research committee and the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards. It was
approved by the Ethic Committee of the Faculty of Medicine, Chiang Mai University (No. 7437/2020).

**Case report**

A 26-year-old woman was diagnosed SLE in 2011, when she presented with facial erythema, discoid rashes, oral ulcers, leukopenia, thrombocytopenia, nephritis, seizure, positive anti-nuclear antibody (ANA) and anti-double-stranded DNA (anti-dsDNA). She was treated initially with high dose corticosteroids and cyclophosphamide. The SLE clinical course was complicated by 2 episodes of each systemic salmonellosis and NPSLE. Her SLE was controlled with corticosteroid and mycophenolate mofetil. These medications were discontinued in March 2016, when full completed remission occurred. The patient had minor flare again in February 2018, which was controlled with prednisolone at 20 mg/d.

In November 2018, she presented with fever, chill and dysuria while taking prednisolone at 5 mg/d. General physical examination was unremarkable. A complete blood count, blood electrolytes, renal and liver function and chest radiograph were unremarkable. Urine analysis showed pyuria and +3 proteinuria. Blood and urine cultures grew non-typhoidal salmonella. Acute urinary tract infection was diagnosed and intravenous ceftriaxone was given. The fever disappeared in the following 3 d.

Four days after admission, she complained of light headache. Physical examination showed mild alteration of consciousness, impaired cognitive function, slow speech with monotonous voice, mild tremor (resting, postural and kinetic type), rigidity and bradykinesia of both arms, which was more severe in the left one. She also had slowness of gait. There was no facial weakness. All of the motor was grade V, with negative Babinski’s sign and clonus. There was no stiffness of the neck. Acute parkinsonism was diagnosed. Additional investigation showed 2.6 g of 24-h urine protein, low serum complements, high positive anti-dsDNA antibodies and hypertriglyceridemia. Anti-cardiolipin (ACL) antibodies and lupus anti-coagulants (LAC) were negative. Cerebrospinal fluid (CSF) examination showed protein at 42 mg/dL, glucose at 53 mg/dL (blood glucose 101 mg/dL), and 1 polymorphonuclear cell/cu.mm. Electroencephalogram (EEG) showed diffuse slowing of background activity consistent with diffuse cerebral dysfunction. Magnetic resonance imaging (MRI) of the brain showed T2/FLAIR hyperintensity bilaterally in the basal ganglia. Serum and CSF for autoimmune encephalopathy profiles showed a negative result.

Intravenous dexamethasone at 20 mg/d was given, but the parkinsonian symptoms did not improve after 3 d of treatment. The treatment was switched to a 3-d course of intravenous pulse methylprednisolone (IVMP) at 1000 mg/d, together with a single dose of intravenous cyclophosphamide (IVCY) at 1000 mg. Levodopa/carbidopa at 300/75 mg/d also was prescribed. One week later, the patient’s slow speech improved markedly, but not the tremor, rigidity or bradykinesia. She was discharged with prednisolone at 40 mg/d and levodopa/carbidopa at 300/75 mg/d.

A repeat MRI at 4 months showed complete resolution of previous abnormal findings. Mycophenolate mofetil at 2 gm/d was initiated after a 6-month course of IVCY, when her nephritis came into complete remission. The medication was tapered gradually to prednisolone at 2.5 mg/d and mycophenolate mofetil at 500 mg/d. Her parkinsonian symptoms showed progressive improvement and disappeared in October 2019.

**Statistical analysis**

The IBM SPSS computer program version 23.0 (IBM SPSS Statistics, Armonk, NY) was used for statistical analysis. Continuous variables were presented as mean ± standard deviation (SD) and categorical variables as percent. The Student’s t-test and Wilcoxon rank-sum test were used to determine the differences between two independent samples of continuous variables, and the Chi-square test or Fisher exact test was used to determine the differences among the categorical variables, where appropriate. A p value < .05 was considered as a statistically significant difference.

**Literature review of parkinsonism in SLE patients**

In a review of English language literature, 27 cases of parkinsonism in SLE patients with adequate information on SLE and parkinsonism were identified [7,11–33]. Including this patient, a total of 28 cases of parkinsonism in SLE (19 adults and 9 children) were analyzed (Supplementary Table 1). Twenty-three patients were female. Of the reported patients, 17 were Asian, six European, three South American, one African and one Australian. The mean ± SD age of the patients was 33.46 ± 20.08 years. Of 26 patients, with available disease duration, parkinsonism occurred simultaneously with SLE diagnosis in six cases, while parkinsonism developed after SLE diagnosis in the remaining 20, with mean duration of 2.25 ± 4.14 years. Parkinsonism occurred in three patients when SLE was
quiescent. SLE manifestations during parkinsonism episodes were hematologic in 15 cases, mucocutaneous in 11, musculoskeletal in 9, seizure or psychosis in 8, renal in 5, cutaneous vasculitis in 4, and cardiopulmonary in 3. Constitutional symptoms including fever, weight loss and malaise were not uncommon. ANAs were positive in 27 patients tested. Anti-dsDNA, anti-phospholipid (APL) antibodies (including ACL antibodies and LAC) and low complement levels were observed in 16 of 22, 6 of 14 and 15 of 17 patients, respectively, who had been tested or mentioned. Other serology, including anti-SSA, anti-SSB, anti-Smith antibodies (anti-Sm), anti-ribonucleoprotein antibodies (anti-RNP) and anti-neutrophilic cytoplasmic antibodies (ANCA) was reported occasionally as positive. Two patients, one had anti-ribosomal P protein antibodies and another had anti-dopaminergic cell antibodies in their serum.

The main clinical features of parkinsonism, including masked face, resting tremor, rigidity, bradykinesia or akinesia, postural instability and gait difficulty were reported in 13, 20, 25, 22, 10 and 18 patients, respectively. Asymmetrical involvement was noted in 10 patients. Alteration of consciousness, cognitive dysfunction, headache, abnormal speech and dystonia were noted in 14, 7, 6, 14 and 3 patients, respectively. Mutism was noted in six patients, of which all were juvenile SLE. Emotional disturbance (restlessness, agitation, anxiety, depression and self-injury) and focal neurological deficits (ptosis, diplopia, hemiparesis and facial weakness) have been mentioned occasionally. Brisk deep tendon reflex and positive extensor plantar response (Babinski’s sign) were noted in 8 of 14 and 7 of 12 patients, respectively (Supplementary Table 2).

Eight of 19 patients, who had CSF examination, showed abnormal results (4 had increased cells [11–32 cells/cm.mm] and 5 increased CSF protein [62–354 mg/dL]). Two patients had anti-ribosomal P protein, one of which also had anti-dsDNA in their CSF. EEG on 8 of 11 patients was abnormal, and all of them showed generalized diffuse slow background activity. Of seven patients, who had computed tomography (CT) scan of the head, one showed abnormality of increased intensity at the basal ganglia, one brain atrophy with multiple infarctions and one basal ganglia calcification. Eleven of 24 patients, who had MRI of the brain, showed non-specific findings. The remaining 13 patients, who had abnormal MRI, showed increased signal intensity on T2/FLAIR in the brain, in particular, bilaterally at the basal ganglia and external capsule. Other abnormal lesions were observed at the thalamus, hippocampus, frontal gurus, periventricular area, central semiovale, brain stem and spinal cord. Of nine patients, who had single-photon emission computed tomography (SPECT), one was normal, five showed hypoperfusion at the basal ganglia, internal capsule, lentiform nucleus or parietal temporal lobe and three showed hyperperfusion at the basal ganglia. Dopamine transporter (DAT) imaging using SPECT was performed in one patient and was normal. Positron emission tomography (PET) scan was performed in two patients, of which one was normal, and one showed brain functional abnormality (Supplementary Table 2).

The clinical features of adult and child parkinsonism in SLE patients were compared and are shown in Table 1. Overall, the clinical manifestations and serological findings among the two groups were similar, except that the child patients had significantly more psychosis (44.44 vs. 5.26%, \( p = .026 \)), seizures/psychosis (66.67 vs. 10.53%, \( p = .005 \)), mutism (66.67 vs. 0%, \( p < .001 \)), brisk deep tendon reflexes (100 vs. 33.33%, \( p = .031 \)), and positive extensor plantar response (100 vs. 28.57%, \( p = .028 \)).

Treatment of parkinsonism associated with SLE varied widely (Table 2). Twenty-three patients received initial treatment with corticosteroids (high dose corticosteroids or IVMP and/or immunosuppressive drugs (cyclophosphamide or IVCY) or immunomodulation therapy (plasma pheresis, plasma exchange, intravenous immunoglobulin or rituximab). Dopamine therapy also was used as initial treatment in combination with the treatments above. CNS symptoms, e.g. seizure, psychosis, cognitive dysfunction, dystonia, headache and abnormal speech improved or disappeared in most of the cases. The outcomes of parkinsonian symptoms were complete resolution in 17 patients, partial response in 7 (one due to poor compliance), and persistent symptoms in 4. There were no significant differences in the treatment outcomes among patients who received dopamine therapy and those who did not in terms of symptom resolution and partial response vs. persistent symptoms (13 in 16 patients vs. 11 in 12 patients, \( p = .613 \)), or those with symptom resolution vs. partial response or persistent symptoms (9 in 16 vs. 8 in 12, \( p = .704 \)). Of 24 patients, whose duration of treatment was available, the mean ± SD duration of response to treatment was 0.95 ± 1.21 months.

**Discussion**

Of the 28 patients in this report, parkinsonism occurred in 90% of the SLE patients with active disease. Parkinsonism occurred together with other
clinical manifestations that led to SLE diagnosis in approximately one-fourth of the cases, while it occurred in the remainder after SLE diagnosis. Resting tremor, bradykinesia and rigidity were among the main clinical features seen in parkinsonism in SLE patients, which was similar to those seen in non-SLE patients. Other neurological features, including seizures, psychosis, alteration of mental status, headache and focal neurological deficits, supported the fact that parkinsonism was part of CNS involvement in SLE patients. Of interest, SLE children with parkinsonism had significantly more psychosis, seizures/psychosis, mutism, brisk deep tendon reflexes and positive extensor response than adult patients.

The pathogenesis of parkinsonism in SLE patients has not been understood clearly. A reason might be

Table 1. Comparison of clinical features in parkinsonism between adult and child patients with SLE.

| Age, in years (mean ± SD) | Total (n = 28) | Adults (n = 19) | Children (n = 9) | p Value |
|--------------------------|---------------|----------------|----------------|--------|
| 33.46 ± 20.08 | 42.95 ± 17.53 | 13.44 ± 2.30 | <.001 |
| Sex, female, n (%) | 23 (82.14) | 14 (73.68) | 9 (100.00) | .144 |
| Onset of parkinsonism from SLE diagnosis, in years (mean ± SD) | 2.25 ± 4.14 | 3.00 ± 4.92 | 0.875 ± 0.77 | .197 |
| (n = 20) | (n = 14) | (n = 7) |

Table 2. Outcome of parkinsonism in SLE according to initial treatment.

| Type of treatment | Resolution (n = 17) | Partial response (n = 7) | Persistence (n = 4) |
|------------------|---------------------|------------------------|--------------------|
| Corticosteroids monotherapy (n = 3) | 0 | 2 | 1 |
| Corticosteroids + dopamine therapy (n = 6) | 2 | 2 | 2 |
| Corticosteroids + IS/IM therapy (n = 9) | 8 | 1 | 0 |
| Corticosteroids + IS/IM + dopamine therapy (n = 5) | 5 | 0 | 0 |
| Dopamine monotherapy (n = 5) | 2 | 2 | 1 |
| Dopamine based treatment | | | |
| Dopamine therapy included (n = 16) | 9 | 4 | 3 |
| Dopamine therapy not included (n = 12) | 8 | 3 | 1 |

IS: immunosuppressive drugs; IM: immunotherapy (plasma pheresis, rituximab, intravenous immunoglobulin)

Table 2. Comparison of clinical features in parkinsonism between adult and child patients with SLE.

| Total (n = 28) | Adults (n = 19) | Children (n = 9) |
|---------------|----------------|----------------|
| 27/27 (100.00) | 18/18 (100.00) | 9/9 (100.00) | .103 |
| 16/22 (72.73) | 11/16 (68.75) | 5/6 (83.33) | .103 |
| 6/14 (42.86) | 3/10 (30.00) | 3/4 (75.00) | .103 |
| 15/17 (88.24) | 11/13 (84.62) | 4/4 (100.00) | .103 |

*Number of positive tests/number tested; ANA: anti-nuclear antibodies; anti-dsDNA: anti-double-stranded DNA; APL/LAC: anti-phospholipid antibodies/lupus anti-coagulants
due to the limited number of patients as well as paucity of complete neurological investigations. However, as most cases occurred during active SLE, the pathogenesis of parkinsonism might be similar to that of NPSLE and both vascular and immunologic mechanisms might be involved [34–36]. The vascular hypothesis might be due to vasculitis, which was supported by the abnormal increase in intensity at the basal ganglia and substantia nigra, as seen in MRI and the presence of widespread vasculitis at the substantia nigra and hypoperfusion, as observed at approximately 63% in SPECT (Supplementary Table 2). Unfortunately, approximately 37% of the patients showed hyperperfusion at the basal ganglia in SPECT. Whether the difference in perfusion findings at the basal ganglia reflects the different stage or pathogenic mechanism of parkinsonism in SLE patients was unclear.

The immune mechanism was supported by the presence of anti-dopaminergic cell antibodies (anti-DA) in serum (but not in the CSF) of a female SLE patient with rapidly progressive Parkinson’s disease [19], the presence of IgG bound to the cell surface of live neurons with dopaminergic properties [16] and the presence of anti-ribosomal P protein in serum and CSF of SLE patients with parkinsonism [16,30,32]. The presence of anti-ribosomal P protein has been shown to associate with neurological involvement in CNS lupus [37,38]. As a majority of parkinsonism cases occur during active SLE disease, and most of the patients showed good response to corticosteroids and immunosuppressive or immunomodulatory therapy, underlying the immune mechanism in the pathogenesis of the disease was supported.

Overall, the treatment of parkinsonism in SLE patients had favorable outcomes, as 85.71% of patients had complete resolution or showed partial symptom improvement. Whether the treatment response was due to corticosteroids and immunosuppressive drugs or immunomodulator therapy was unclear, as most patients also received dopamine therapy. However, the outcome among those who received dopamine therapy was no different from those who did not. It is notable that four of five patients, who received dopamine monotherapy, showed complete resolution or partial improvement in parkinsonism symptoms. Corticosteroids and immunosuppressive drugs or immunomodulator therapy are usually used to treat active SLE, but can improve parkinsonism that is a manifestation of NPSLE.

Of interest, a nationwide population-based report from Taiwan found an inverse relationship in the developmental risk of Parkinson’s disease in SLE patients, when compared with an age and a sex-matched control of non-SLE patients [39]. The risk was lower among the longer follow-up and increasing age of SLE patients. Unfortunately, the mechanism that could explain the inverse association between Parkinson’s disease and SLE was unclear.

Conclusions
This report reviewed parkinsonism in patients with SLE. Parkinsonism in SLE is a rare neuropsychiatric (NP) manifestation in SLE patients. It usually occurs in patients with active SLE. The response to corticosteroids and immunosuppressive drugs or immunomodulator therapy supported the immunological process in the pathogenic mechanism. Physicians are encouraged to perform complete neurological examination in patients who have new onset of NPSLE in order to detect this rare but treatable condition and prevent further morbidity.

Disclosure statement
No potential conflict of interest was reported by the author(s).

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