



Case Series

Case Series: Catatonia in Systemic Lupus Erythematosus

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ABSTRACT

Neuropsychiatric symptoms of systemic lupus erythematosus (NPSLE) are a complex condition with varying prevalence rates, ranging from 37% to 95%. The American College of Rheumatology (ACR) issued the case definitions for NPSLE syndromes in 1999, but did not include catatonia, a behavioral syndrome characterized by unusual movement and immobility. Catatonia, a multifaceted syndrome, is characterized by the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) and requires a more general evaluation of neuropsychiatric symptoms. In this case series, we discuss two SLE cases where catatonia was a prominent neuropsychiatric feature, emphasizing the importance of recognizing catatonia as a possible complication, enhancing management of complex clinical scenarios, and timely intervention.

Keywords: Catatonia, Neuropsychiatric manifestations in SLE, SLE

INTRODUCTION

An important clinical challenge in diagnosing and managing the neuropsychiatric features of systemic lupus erythematosus (NPSLE) is its repercussions. The significant variation in NPSLE prevalence, reported to range from 37% to 95% in some case studies, highlights the scope and diversity of this condition.^[1]

The American College of Rheumatology (ACR) issued a case definition for the distinct neuropsychiatric features of systemic lupus erythematosus (SLE) in 1999. This helped healthcare professionals understand and address these symptoms.^[2] However, the framework of these case definitions portrays a patient's neuropsychiatric lupus symptoms as incomplete. The ACR definitions did not mention catatonia, a behavioral syndrome marked by unusual movement and immobility, which is particularly notable.^[3]

Despite this oversight, a few cases of catatonia in SLE have been reported. This underlines the need for a broader evaluation of the neuropsychiatric symptoms in lupus patients.^[4]

Catatonia is a complex symptom characterized by the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) through specific features for diagnosis. The DSM-5 requires the presence of three or more signs and symptoms from a defined list. These symptoms include agitation, mutism, posturing, mannerisms, grimacing, stereotypy, negativism, waxy flexibility, cataplexy, echolalia, and echopraxia.^[5]

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In this publication, we describe two SLE cases where catatonia was the main neuropsychiatric feature. These cases emphasize the need to recognize and acknowledge catatonia as a possible complication of SLE. Catatonia does not need to be explicitly mentioned in diagnostic criteria to be considered. Focusing on the various neuropsychiatric symptoms of lupus increases the chances of effectively managing complex clinical situations. This approach also facilitates timely treatment.

CASE SERIES

Case 1

A 16 years old boy with SLE came with a 2 weeks duration of history of clouded or altered level of consciousness, inability to rest or relax, not talking with anybody, even not responding on asking, but sometimes saying a few muddled words repeatedly that were clear, excessive crying, being easily frightened, holding both hands in an abnormal extended position for a while without any discomfort, and lying down with both hands keeping over his chest in 'namaskar' position. Two years earlier, he had also had a loss of hair, breathlessness, ulcers in the oral cavity, and also constitutional symptoms. With an ejection fraction of 40%, his 2D-echocardiogram showed left ventricular hypokinesia. Intravenous cyclophosphamide was started according to the National Institutes of Health (NIH) protocol. He was improving, however, and later lost the follow-up.

During the hospitalization, on examination, he was found to have altered sensorium, mutism, restlessness, waxy flexibility, and posturing. Additional investigations, such as the cerebrospinal fluid (CSF) examination, the lumbar puncture, both blood and urine cultures, and a computed tomography brain scan, returned normal findings. Laboratory examination revealed the anti-double-stranded DNA (dsDNA) of 104.3 IU/ml, the anti-N-methyl-D-aspartate (NMDA) receptor antibody level of 0.75, and normal complement levels for both C3 and C4.

His Systemic Lupus Erythematosus Disease Activity Index 2000 (SLEDAI-2K) score was 20 during admission. He was diagnosed with SLE along with neuropsychiatric features, i.e., catatonia, and according to NIH protocol, he was given intravenous methylprednisolone, cyclophosphamide, and lorazepam at a dose of 6 mg daily for catatonia.

This patient made progress and was sent home with a follow-up plan in place. At follow-up appointments, he did not experience any relapse of symptoms.

Case 2

A female patient, age 23, was brought to the hospital with

a history of fever, gum bleeding, and melena that lasted for a week. After pancytopenia was found during preliminary testing, a malignancy investigation was conducted. However, bone marrow aspiration and biopsy, along with other tests listed in Table 1, were normal. Due to persistent symptoms, the patient was then recommended to be referred to the rheumatology department.

Positive results from rheumatological investigations included an increased level of anti-dsDNA of 530 IU/ml and a speckled 3+ anti-nuclear antibody (ANA) on the HEp-2 assay. Antiphospholipid antibody (APLA) levels were within normal limits. The patient was scheduled to start immunosuppression and received pulse methylprednisolone.

Then, the patient was directed to the psychiatric department for a clouded or altered level of consciousness, reduced speaking or responding gradually, repeating some words but not coherently, and after a few days, no verbal speaking, not even nodding or replying to anyone, and decreasing food intake day by day. During hospitalization, on examination, the patient presented with altered sensorium, repeated a few words, reduced speech with time and interaction with family members, inability to recognize family members, reduced oral intake, mutism, grimacing, posturing, and waxy flexibility. She also had a seizure with generalized tonic-clonic seizures (GTCS).

The cerebrospinal fluid (CSF) test and brain magnetic resonance imaging were both normal, and the culture came back negative. All microorganisms were found to be absent from the urine and blood cultures. Additionally, viral markers came back negative. The results of the human immunodeficiency virus, hepatitis b surface antigen, and hepatitis c virus screening were negative. Tests for fungal culture, CSF for Indian ink preparation, CSF Multiplex polymerase chain reaction, and the viral encephalitis panel came back negative. The X-ray of the chest was also normal. The levels of C3 and C4 were both low. Table 1 contains a list of the lab parameters. Consultations in neurology and psychiatry were requested. The patient's catatonia satisfied the DSM-5 criteria. Her score on the SLEDAI-2K was 30.

According to the NIH regimen, cyclophosphamide was started in response to the catatonic presentation and the high disease activity. To control the catatonia, lorazepam (6 mg/day) in divided doses was recommended along with antiepileptic medication that included phenytoin (20 mg/kg). After two weeks in the hospital, her condition improved, and she was released.

The patient had fever, seizures, and jaundice (total/direct bilirubin: 14.2/12.5 mg/dl, transaminases: 568/142). At the second visit, which was five months later, the patient

displayed catatonic symptoms, including posturing, stupor, and grimacing. The patient had also stopped follow-up for five months. 530 IU/ml was the anti-dsDNA titre.

Investigations for autoimmune hepatitis and viral screening were also normal. However, due to financial constraints, the level of anti-NMDA receptor antibodies could not be

Table 1: Summary of laboratory & clinical characteristics of the cases .

Differentials	Case 1	Case 2
Patient's gender	Male	Female
Age of presentation	16 years	23 years
Disease duration (yrs)	1.5 yrs	2.5 yrs
Catatonic features	Posturing, mannerisms, stupor, negativism, stereotypy, mutism, cataplexy, waxy flexibility, echolalia, and echopraxia	Grimacing, waxy flexibility, mutism, and posturing
Hb (Haemoglobin) (gm %)	8.2 gm%	8.6 gm%
Total leukocyte count (per cumm)	4270 per cumm	3460 per cumm
Differential count (per cumm)	Neutrophils- 72, Lymphocytes- 12	Neutrophils- 86.6, Lymphocytes- 11.5
BUN (mg/dL)	26 mg/dL	38 mg/dL
Creatinine(mg/dL)	0.64 mg/dL	0.72 mg/dL
Na+ (mEq/L)	131 mEq/L	141 mEq/L
K+ (mEq/L)	3.5 mEq/L	3.9 mEq/L
SGOT (mg/dL)	28 mg/dL	38 mg/dL (1st visit), 470 mg/dL (2nd visit)
SGPT (mg/dL)	32 mg/dL	28 mg/dL (1st visit), 247 mg/dL (2nd visit)
Urine routine	Normal	Albumin 2+
24 hr Urine protein (mg /day)	0.4 mg/day	0.5 mg/day
ANA (IIF)	Homogenous 2+	Speckled 3+
Anti nuclear antibody (ANA) profile	Ribosomal P	Negative
Anti-double-stranded DNA (Anti-dsDNA) (IU/mL)	104.3 IU/mL	530 IU/mL (2nd visit)
C3 (mg/dl)	92 mg/dL	43 mg/dL
C4 (mg/dl)	36 mg/dL	4 mg/dL
Antiphospholipid antibody	ACL IgG/IgM:24.4/25.3 B2GPI IgG/IgM:7/27	ACL IgG/IgM:30/36 B2GPI IgG/ IgM:2.92/4.72
Anti-NMDA receptor antibody	0.75	Could not be assessed because of financial condition
Lupus anticoagulant	Negative	Negative
Cerebrospinal fluid (CSF) analysis	Normal	Normal
SLEDAI-2K score*	20	30
MRI (brain)	Normal	Chronic haemorrhages in the posterior parietal region of the brain
Management	Cyclophosphamide, Steroids, Lorazepam	Cyclophosphamide, Steroids, Lorazepam

*SLEDAI-2K score: The Systemic Lupus Erythematosus Disease Activity Index 2000 (SLEDAI-2K) UN: Blood urea nitrogen, Na+: Sodium Ion, K+: Potassium Ion, SGOT: Serum glutamic - oxaloacetic transaminase, SGPT: Serum glutamic - pyruvic transaminase, DNA: Deoxyribonucleic acid, NMDA: N-methyl D-aspartate, ANA (IIF): Antinuclear antibody detection (Indirect immunofluorescence), ACL: Analytical chemistry laboratory, IgG: Immunoglobulin G, B2GPI: Beta-2-glycoproteinI.

measured (Table 1). After receiving advice and 75% of the recommended dosage of cyclophosphamide, the patient's symptoms and liver function tests improved. She was instructed to continue taking the same drugs for the next one to two months to monitor the patient's catatonic features. The reason for the relapse was not adequately worked up because the patient then changed states and lost contact.

DISCUSSION

Catatonia is a syndrome associated with schizophrenia, but it can also occur in various psychiatric and non-psychiatric medical conditions. Non-psychiatric causes may include infections, whether systemic or related to the central nervous system, stroke, delirium, neuroleptic malignant syndrome, nonconvulsive status epilepticus, and anti NMDA receptor encephalitis.^[5]

The neuropsychiatric symptoms of SLE are difficult to treat, leading to serious health issues and increased risk of death. Neuropsychiatric SLE catatonia is a rare but serious form of SLE that needs quick identification and management. Catatonia can appear at any stage of SLE progression, either as an early symptom or during a lupus relapse.^[6]

The cases reported showed the typical signs of catatonia found in SLE. These signs included mutism, grimacing, posturing, and waxy flexibility. Both cases met the DSM-5 criteria for diagnosing catatonia. We also ran tests to exclude other causes. We measured disease activity using the SLEDAI-2K score. Both patients improved with high doses of corticosteroids and immunosuppressants like cyclophosphamide, along with supportive care using benzodiazepines.^[7]

These results align with existing literature on pediatric catatonic lupus. Frago-Loyo *et al.*^[7] described two SLE cases with catatonia that responded to corticosteroids, cyclophosphamide, and lorazepam. Grover *et al.* reported on a 22-year-old woman with SLE who showed catatonic features during a disease relapse. She received treatment with lorazepam, steroids, and cyclophosphamide.^[8]

Boeke *et al.* presented two cases of SLE with catatonia and reviewed over 35 additional cases from the literature.^[9] In both situations, doctors provided immunosuppressive therapy for the underlying SLE. They managed symptomatic catatonia with benzodiazepines in 81% of patients and with electroconvulsive therapy in 38%. It's crucial to consider catatonia in NPSLE and treat it as an autoimmune disease. This includes addressing factors that may worsen the condition and providing relief through benzodiazepines and electroconvulsive therapy (ECT) for cases that do not improve with standard treatments. This research enhances

the understanding and management of catatonia in NPSLE.^[9]

In another case series, Sundaram *et al.* reported two cases of SLE with catatonia. Both patients improved with immunosuppressive therapy.^[10] The first case involved fever, pancytopenia, a TEN-like rash, and later developed catatonia along with macrophage activation syndrome. The second case showed symptoms of acute cutaneous lupus erythematosus, alopecia, fever, polyarthralgia, nephritis, cytopenia, and catatonia. They reviewed the literature and found 37 reported cases so far. The ages of these patients ranged from 14 to 46 years. Catatonia typically occurred between one month and 13 years after the onset of SLE, although it could be the first symptom in some instances. Both patients initially displayed psychiatric features. Those who developed catatonia did not always have specific clinical signs or lab results, but nephritis was rare, appearing in only two cases. There were six positive cases for anti-ribosomal antibodies. Early detection and treatment with immunosuppression, along with benzodiazepines and/or ECT, were crucial for effective management.^[10]

In this instance, the second case showed a relapse at five months, even after initial improvement in our study. Breilinski *et al.* also noted this tendency for relapse, reporting recurrence rates of up to 50% in pediatric catatonic SLE patients. Therefore, careful follow-up is essential.^[11]

CONCLUSION

Both cases support the current evidence for catatonia in SLE patients, which is a serious but rare neuropsychiatric issue. Early diagnosis, strong immunosuppression therapy, supportive care, and long-term monitoring are essential for improving outcomes in these vulnerable patients. It's also important to work with a multidisciplinary team and follow treatment guidelines, especially due to the risk of recurrence. Further research to identify predictive factors and the best maintenance treatments could offer valuable insights.

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