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*Case Report*

# A Case Report of Female Systemic Lupus Erythematosus and Cerebral Lupus as The Complication : Diagnosis and Treatment

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## Abstract

**Background :** Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease that affects multiple systems in the body. Because clinical manifestations can appear in different organs, the complications are diverse and can be quite severe, one of which is cerebral lupus or neuropsychiatric systemic lupus erythematosus (NPSLE). It includes neurological and psychiatric syndromes in SLE patients where other causes have been ruled out. We reported a case of SLE manifestation with cerebral complication involvement. This case report aims to provide insights and expect to offer an understanding into the clinical presentation, diagnosis, and management of a patient with cerebral lupus.
**Case Presentation :** 18-year-old Indonesian woman with complaint of sudden seizure was referred to the Emergency Unit of Dr. Ramelan Naval Central Hospital Surabaya. She had previously been diagnosed with SLE back in 2022 and consistently does a monthly checkup in the internal medicine clinic at Dr. Ramelan Naval Central Hospital Surabaya. Initial examinations revealed signs of infection, slight electrolyte imbalances, and a flare phase of SLE, but no abnormalities in imaging tests. She received initial treatments of loading phenytoin along with mecobalamin injection, vitamin B6, methamizole, and cefobactam. Over the course of her hospital stay, with no further seizures, she was discharged with medication for continued treatment and a scheduled follow-up.
**Conclusion :** This case of cerebral lupus is rare. SLE can damage the blood brain barrier (BBB), causing neuropsychiatric complications.

**Keywords :** Autoimmune diseases, Neuropsychiatric Systemic Lupus Erythematosus, Systemic Lupus Erythematosus.

## INTRODUCTION

Systemic Lupus Erythematosus (SLE) can be defined as a chronic, multi-system autoimmune disease with various systemic manifestations that involve almost all tissues and organs.[1,2] The annual incidence of SLE in Asia ranges from 2.8 to 8.6 per 100,000 people per year, with the prevalence varying from 26.5 to 103 per 100,000 individuals.[3] Several classification criteria has been developed over time. there are three classification criteria for SLE that can be used: ACR 1997, SLICC 2012, and EULAR/ACR 2019.[4,5] Because clinical manifestations can occur in various organs, complications of SLE are diverse and can be severe, one of which is cerebral lupus or neuropsychiatric systemic lupus erythematosus (NPSLE).

Cerebral lupus encompasses various psychiatric and neurological manifestations due to SLE involvement in the nervous system, where other causes have been excluded.[6–9] Neuropsychiatric manifestations of SLE range from mild symptoms such as headaches, anxiety, depression, psychosis, and pseudodementia, to more severe conditions such as seizures, stroke, or coma.[6,7,10] These manifestations usually occur early in the course of SLE, with a higher incidence in young women.[11] Cerebral lupus presents unique challenges in diagnosis.[8,9] There are no specific criteria for diagnosing NPSLE, but they can be assisted by using clinical, serological, immunological, electrophysiological, and neuroimaging studies to eliminate other comparative diagnoses.[12,13]

We present a case of a patient with Systemic Lupus Erythematosus (SLE) complicated by cerebral lupus. Additionally, this case report is expected to offer an understanding of the clinical characteristics, diagnosis, and treatment of patients with SLE complicated by cerebral lupus.

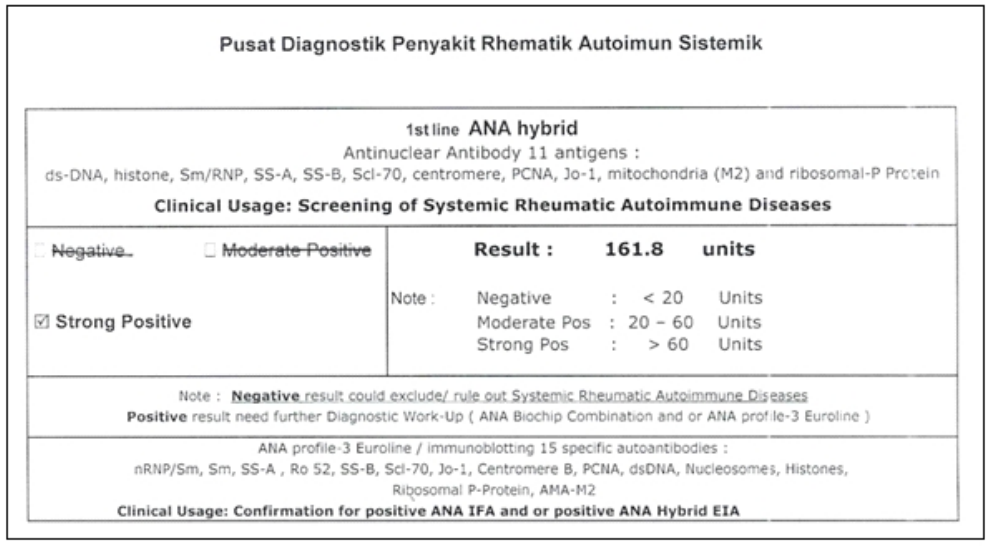
## CASE PRESENTATION

18-year-old Indonesian woman was brought to the emergency department of RSPAL Dr. Ramelan Surabaya with the chief complaint of seizure that lasted about 5 minutes, 30 minutes before she was admitted to the hospital. It occured throughout the whole body, with stiffening and jerking, without a foamy mouth nor bed wetting. She was unconscious right after the seizure stopped, then regained consciousness again. She had a history of frequent headeche (migraines), nausea, and vomiting. While in the emergency room, the patient experienced repeated seizures for the second time – a similar one, which was a tonic clonic for about 15 seconds.

The history of seizures was denied, but she has suffered from Systemic Lupus Erythematosus (SLE) for a year, as confirmed by Antinuclear Antibody (ANA) test, which resulted in a strong positive 161 units. The medication has been taken since that day, and the latest medications were Kamyfet 2 x 500mg, Fenofibrat 1 x 100mg, Folic acid 2 x 1 mg, Inpepsa 3 x 1 C, and Omeprazole 1 x 20mg which were routinely taken and controlled.

Upon arrival, her general appearance was weak, with blood pressure of 104/79 mmHg, heart rate of 120/min, temperature of 36,4°C, respiratory rate of 20 breaths per minute, and oxygen saturation of 97% free air. General status within normal limits. Normal neurological examination (no lateralization and meningeal sign was normal), psychiatric disorders were absent .

To establish the diagnosis in this patient, it is necessary to carry out several supporting examinations to exclude the possibility of other diagnoses that result in seizures. The results of the laboratory examination found leukopenia, neutrophilia, lymphocytopenia, anemia,



**Pusat Diagnostik Penyakit Rhematik Autoimun Sistemik**

1st line **ANA hybrid**
Antinuclear Antibody 11 antigens :
ds-DNA, histone, Sm/RNP, SS-A, SS-B, Scl-70, centromere, PCNA, Jo-1, mitochondria (M2) and ribosomal-P Protein
**Clinical Usage: Screening of Systemic Rheumatic Autoimmune Diseases**

☐ ~~Negative~~ ☐ ~~Moderate Positive~~ ☑ **Strong Positive**

**Result : 161.8 units**

Note : Negative : < 20 Units
Moderate Pos : 20 – 60 Units
Strong Pos : > 60 Units

Note : **Negative** result could exclude/ rule out Systemic Rheumatic Autoimmune Diseases
**Positive** result need further Diagnostic Work-Up ( ANA Biochip Combination and or ANA profile-3 Euroline )

ANA profile-3 Euroline / immunoblotting 15 specific autoantibodies :
nRNP/Sm, Sm, SS-A , Ro 52, SS-B, Scl-70, Jo-1, Centromere B, PCNA, dsDNA, Nucleosomes, Histones, Ribosomal P-Protein, AMA-M2
**Clinical Usage: Confirmation for positive ANA IFA and or positive ANA Hybrid EIA**

**Figure 1.** Past history of ANA Test result

TABLE 1
**Laboratory test result**

| Laboratory Test | Patient Test Results | Normal Range |
|---|---|---|
| **Complete Blood Count** | | |
| Leucocytes ($10^3$/µL) | 3.38 (L) | 4.00 – 10.00 |
| Eosinophils (%) | 1.40 | 0.5 – 5.0 |
| Basophils (%) | 0.4 | 0.0 – 1.0 |
| Neutrophils (%) | 82.50 (H) | 50.0 – 70.0 |
| Lymphocytes (%) | 12.50 (L) | 20.0 – 40.0 |
| Monocytes (%) | 3.20 | 3.0 – 12.0 |
| Haemoglobin (g/dL) | 7.70 (L) | 12 – 15 |
| Hematocrit (%) | 23.20 (L) | 37.0 – 47.0 |
| Erythrocytes ($10^6$/µL) | 2.73 (L) | 3.50 – 5.00 |
| MCV (fmol/cell) | 84.9 | 80 – 100 |
| MCH (pg) | 28.4 | 26 – 34 |
| MCHC (g/dL) | 33.4 | 32 – 36 |
| Trombosit ($10^3$/µL) | 143.00 (L) | 150 – 450 |
| MPV (fL) | 12.5 (H) | 6.5 – 12.0 |
| PDW (%) | 16.8 | 15 – 17 |
| PCT ($10^3$/µL) | 1.780 (H) | 0.108 – 282 |
| **Electrolytes** | | |
| Calsium (mg/dL) | 9.9 | 8.8 – 10.4 |
| Natrium (mEq/L) | 149.00 (H) | 135 – 147 |
| Kalium (mmol/L) | 2.95 (L) | 3.0 – 5.0 |
| Chlorida (mEq/L) | 111.4 (H) | 95 – 105 |
| **Chemical Chemistry Analyzer** | | |
| Random Blood Sugar (mg/dL) | 120 | < 200 |
| Quantitative CRP (mg/dL) | 127.0 (H) | < 10 |
| Procalcitonin (PCT) (mg/dL) | 0.62 (H) | < 0.5 |
| **Blood Gas Analysis (BGA) of Artery** | | |
| pH | 7.345 (L) | 7.35 – 7.45 |
| pCO2 (mmHg) | 34.9 (L) | 35 – 45 |
| PO2 (mmHg) | 290.6 (H) | 80.0 – 100.0 |
| HCO3 Act (mEq/L) | 18.8 | – |
| HCO3 Std (mEq/L) | 19.2 | 22 – 26 |
| BE (ecf) (mmol/L) | -7.1 | -2 s/d +2 |
| BE (B) (mmol/L) | -6.5 | – |
| ctCO2 (mmol/L) | 19.9 | – |

TABLE 1. *Continued...*

| Laboratory Test | Patient Test Results | Normal Range |
|---|---|---|
| O2 SAT | 99.6 | >95 |
| O2CT(mL/dL) | 15.1 | – |
| pO2/FI02 | 4.84 | – |
| pO2(A-a)(T) (mmHg) | 115.6 | – |
| pO2(A/a)(T) (mmHg) | 0.72 | – |
| Temp (C) | 36.0 | – |
| ctHb (g/dL) | 10.2 | – |
| FIO2 (%) | 61.0 | – |
| **Renal Function Test** | | |
| Creatinin (mg/dL) | 1.8 (H) | – |
| BUN (mg/dL) | 41 (H) | – |
| **Culture Test** | | |
| Left blood culture + Antibiotic sensitivity test | No bactery growth | |
| Right blood culture + Antibiotic sensitivity test | No bactery growth | |
| **Urinalysis** | | |
| URO | Normal | Normal |
| BLD | 3+ | Negative |
| BIL | Negative | Negative |
| KET | Negative | Negative |
| GLU | Negative | Negative |
| PRO | 2+ | Negative |
| pH | 7.5 | 5.0 – 7.5 |
| NIT | Negative | Negative |
| LEU | 2+ | 0 – 5 |
| CRE | 0.1 | Negative |
| ALB | Over | ≤ 0.02 |
| P/C | 2+ | <0.15 |
| A/C | 2+ | < 30 |
| S.G | 1.005 | 1.015 – 1.025 |
| COLOR | Yellow | – |
| CLOUD | 2+ | Negative |

thrombocytopenia, increased creatinine, increased Blood Urea Nitrogen (BUN) levels, hematuria, proteinuria, pyuria, slight hypernatremy, slight hypokalemia, hyperchloremia, high quantitative C-Reactive Protein (CRP), and increased procalcitonin (PCT). Chest X-ray and Magnetic Resonance Imaging (MRI) were within normal limits. Therefore, by using the SLICC (Systemic Lupus International Collaborating Clinics) damage index

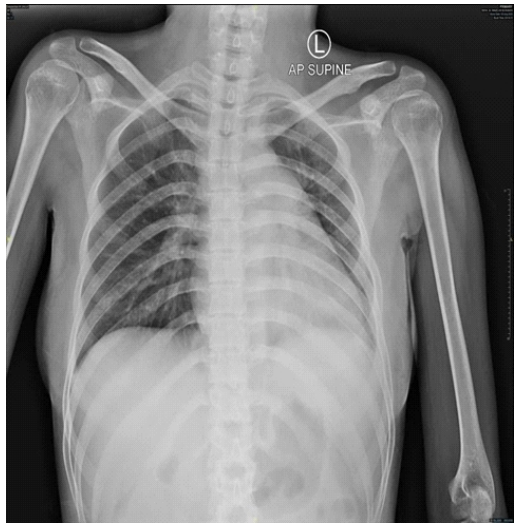



**Figure 2.** Chest X-ray within normal limit

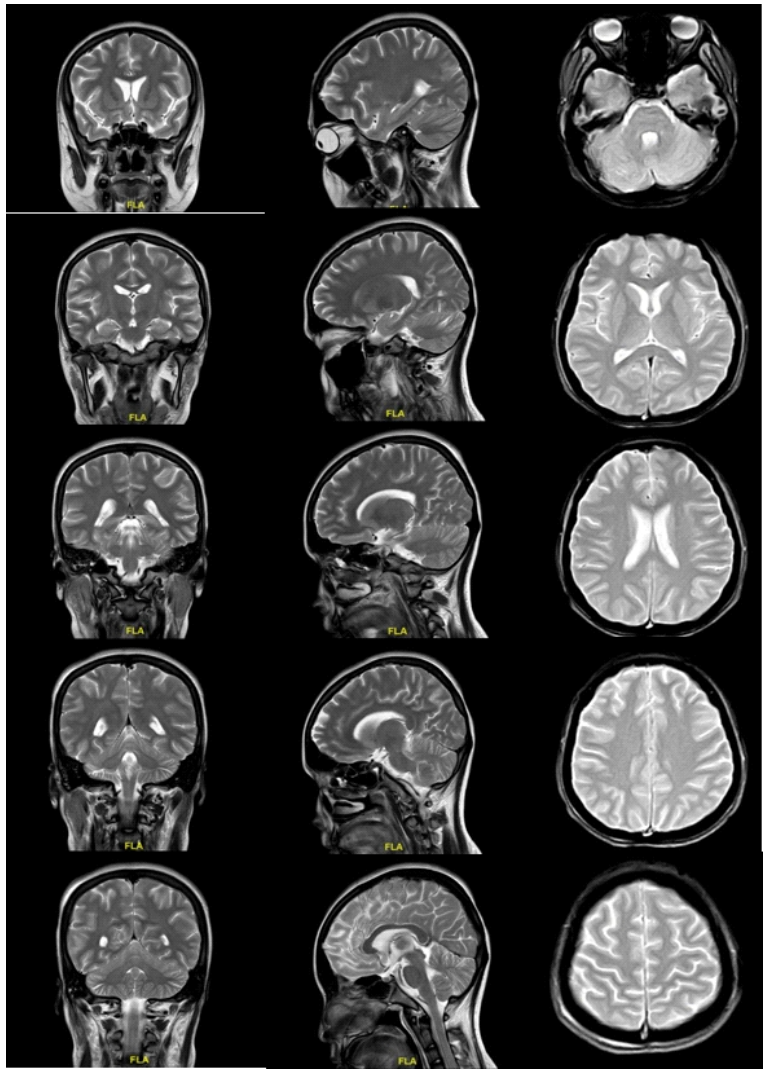



**Figure 3.** Magnetic Resonance Imaging (MRI) head/brain. Non-contrast MRI does not appear abnormality. Brain parenchyme does not show abnormality, no visible infarct/bleeding

**Entry criterion**
Antinuclear antibodies (ANA) at a titer of ≥1:80 on HEp-2 cells or an equivalent positive test (ever)

↓

If absent, do not classify as SLE
If present, apply additive criteria

↓

**Additive criteria**
**Do not count a criterion if there is a more likely explanation than SLE.**
**Occurrence of a criterion on at least one occasion is sufficient.**
**SLE classification requires at least one clinical criterion and ≥10 points.**
**Criteria need not occur simultaneously.**
**Within each domain, only the highest weighted criterion is counted toward the total score§.**

| Clinical domains and criteria | Weight | Immunology domains and criteria | Weight |
|---|---|---|---|
| ***Constitutional*** | | ***Antiphospholipid antibodies*** | |
| Fever | 2 | Anti-cardiolipin antibodies OR | |
| ***Hematologic*** | | Anti-β2GP1 antibodies OR | |
| Leukopenia | 3 | Lupus anticoagulant | 2 |
| Thrombocytopenia | 4 | ***Complement proteins*** | |
| Autoimmune hemolysis | 4 | Low C3 OR low C4 | 3 |
| ***Neuropsychiatric*** | | Low C3 AND low C4 | 4 |
| Delirium | 2 | ***SLE-specific antibodies*** | |
| Psychosis | 3 | Anti-dsDNA antibody* OR | |
| Seizure | 5 | Anti-Smith antibody | 6 |
| ***Mucocutaneous*** | | | |
| Non-scarring alopecia | 2 | | |
| Oral ulcers | 2 | | |
| Subacute cutaneous OR discoid lupus | 4 | | |
| Acute cutaneous lupus | 6 | | |
| ***Serosal*** | | | |
| Pleural or pericardial effusion | 5 | | |
| Acute pericarditis | 6 | | |
| ***Musculoskeletal*** | | | |
| Joint involvement | 6 | | |
| ***Renal*** | | | |
| Proteinuria >0.5g/24h | 4 | | |
| Renal biopsy Class II or V lupus nephritis | 8 | | |
| Renal biopsy Class III or IV lupus nephritis | 10 | | |

**Total score:**

↓

**Classify as Systemic Lupus Erythematosus with a score of 10 or more if entry criterion fulfilled.**

**Figure 4.** ACR/EULAR Criteria of SLE 2019[17]

to establish the diagnosis, where the ANA-IF test was positive as the immunologic criteria and for the clinical criteria in which, seizures, severe persistent headache, hematuria, proteinuria, pyuria, thrombocytopenia, and leukopenia were found, all indicated the activity of SLE in this case. Supported by neuropsychiatry systemic lupus erymathosus guidelines from American College of Rheumatology, we could diagnose that the seizures and severe persistent headache were caused by the NPSLE.

The patient was consulted to a neurologist who gave the advice of administering Phenytoin 100 mg x 7 ampoules using a syringe pump for 15 minutes; 3x1 ampoules were subsequently given; and if the seizures recur, Diazepam 1 ampoule was to be given slowly intravenously; as well as Mecobalamin injection of 500 mcg 1x1, Vitamin B6 per oral 1x1 tablet, and Metamizole 3 x 1000 mg IV for the headache complaints. Regarding the history of SLE, she was consulted to an internist who gave the advice of administering Methylprednisolone 250 mg in NaCl 0.9%. 100 cc used up in 1 hour for 3 days and Cefobactam 2x2 gr IV drip in NaCl 0.9% 100 cc used up in 1 hour. Because the results of the laboratory examination found that the potassium level was below normal, KCL 25 meq was given in NaCl 0.9% 500 cc (8 drops per minute). For complaints of nausea and vomiting, she was given omeprazole 2 x 40 mg IV and inpepsa 3 x 1 C.

On the 3rd day of hospitalization, the patient experienced an improvement in complaints, and there was no seizure period. The next day, she underwent an electroencephalogram (EEG) examination, and the results were normal recording of EEG. No epileptiform waves or abnormal slowdown. Some drugs were still given, but the dose was reduced or stopped. Methylprednisolone was given 1 x 125 mg IV until day 5, then lowered to 1 x 62.5 mg. Cefobactam was still given at

the same dose until day 10.

The patient's condition was stable when she was discharged from the hospital and given Phenytoin capsul 3x1, Vitamin B6 2x1 tabs, KSR 2x1 tabs, Omeprazole 2x 20 mg, Methylprednisolone 16 mg 1- 1-0, Kamyfet 3x 500 mg per oral, and Xepazym 2x1 tabs for home consumption. She was recommended to return for a control visit in the hospital a week later.

## DISCUSSION

Cerebral lupus includes neurological and psychiatric syndromes observed in patients with SLE where other causes have been excluded. The manifestations usually occur at the beginning of the SLE journey, with a higher incidence in young women and is the leading cause of morbidity.[8] A three- year prospective study conducted by Magro-Checa *et al*., 2023 of 370 SLE patients with no prior history of Central Nervous System (CNS) involvement determined that CNS involvement was rare in SLE patients, covering only 7.8 per 100 person-years.[11] It occurs because of the damage of the Blood Brain Barrier (BBB) so that the lymphocytes that are inflammatory cells enter the brain and produce cytokines (like IL-6) and autoimmune antibodies (anti-NMDAR and anti-RP) responsible for neuronal damage through signal pathway induction will worsen inflammation as well as initiate the entry of calcium that leads to apoptosis.[14]

According to a series of definitions of 19 Neuropsychiatric Systemic Lupus Erythematosus Syndrome (NPSLE) and its diagnostic criteria from the American College of Rheumatology (ACR), less than 40–50% of incidences are caused by the underlying CNS lupus activity. Further studies showed that Lupus CNS is at least as common in children as it is in adults. A three-year prospective study of 370 SLE patients with no previous history of CNS involvement determined that clinically severe CNSparticipation is rare in patients with SLE, covering only 7.8 per 100 people-years.[11] Patients with cerebral lupus have varying clinical manifestations, making it a unique challenge in diagnosis enforcement. This disease affects the central nervous system, causing aseptic meningitis, seizures, anxiety syndrome, psychosis, or peripheral nervous systems, with myasthenia gravis, mononeuritis, autonomic neuropathy, or polyneuropathies.[15] These manifestations range widely from mild symptoms such as headaches, altered mental status, anxiety, depression, psychosis and pseudodementia, to more serious conditions such as seizures, strokes, or coma. These manifestations most often appear in the first year of SLE diagnosis.[6,7,10] Focal syndromes are mostly neurological, whereas diffuse syndrome is mostly psychiatric. Cerebrovascular disorders and epileptic seizures are found in 5–15% of NPSLE patients. Cognitive impairments, mood disturbances, state of acute confusion, or peripheral neuropathy are found only in 1–5% of patients, while psychosis, myelitis, unconscious movement of extremities and facial muscles, and aseptic meningitis are very rare.[16] As in this patient, we can find the clinical manifestations are more related to the focal manifestation in CNS which are seizures and severe headache.

Diagnosing NPSLE is often difficult because doctors have to rule out alternative causes, such as infections and tumors, before they can establish a diagnosis. There are no laboratory or radiological biomarkers to establish a diagnosis, but they can be assisted by using clinical, serological, immunological,

TABLE 2
**SLICC (Systemic Lupus International Collaborating Clinics)[18]**

| Clinical Criteria | Immunologic Criteria |
|---|---|
| Acute cutaneous lupus | ANA |
| Chronis Cutaneous lupus | Anti-DNA antibodies |
| Oral or nasal ulcers | Anti-Sm antibodies |
| Non-scaring alopecia | Antiphospholipid antibody |
| Arthritis | Low complement (C3, C4, CG50) |
| Serositis | Direct Coombs' test |
| Renal | (do not count in the presence of hemolytic anemia) |
| Neurologic | |
| Hemolytic anemia | |
| Leukopenia | |
| Thromovytopenia (<100.000/mmc) | |

TABLE 3
**Neuropsychiatry manifestations in systemic lupus erymathosus; Adapted from Guidelines for the definition of neuropsychiatric nomenclature from American College of Rheumatology[16]**

| Clinical Criteria | Central Nervous System (CNS) | Peripheral Nervous System (PNS) |
|---|---|---|
| Diffuse manifefstation | Acute confusional state | – |
| | Anxiety disorder | |
| | Cognitive dysfunction | |
| | Mood disorders | |
| | Psychosis | |
| Focal manifestations | Aseptic meningitis | Guillain-Barre syndrome |
| | Cerebrovascular disease | Autonomic disorder |
| | Demyelinating syndrome | Mononeuropathy, single/multiple |
| | Headache | Myasthenia gravis |
| | Movement disorder | Neuropathy, cranial |
| | Myelopathy | Plexopathy |
| | Seizures | Polyneuropathy |

electrophysiological, and neuroimaging studies to eliminate other comparative diagnoses because most of the outcomes of support examinations yield results without specific abnormalities.[12,13] Currently, serological tests are not accurate enough to establish the diagnosis of NPSLE and/or to assess the severity of the disease. Autoantibodies, which are a hallmark of lupus, may also be useful in functioning as biomarkers. In addition to autoantibodies, it is also possible to explore molecules, which circulate in blood and/or Cerebrospinal Fluid (CSF) but so far the findings of CSF are also non-specific and only serve to rule out other etiologies.[19] Neuroimaging can be used to identify CNS involvement in a noninvasive manner in SLE. Compared to CT, MRI is a more sensitive imaging modality to detect intracranial abnormalities and assess the chronicity and evolution of these abnormalities. MRI is the current gold standard of radiology used in assessing patients with SLE, but about 50% of NPSLE patients do not have detectable abnormalities.[8,12] As with patients in this case report, supporting examination results from MRI found normal results with no specific anomalies and laboratory examinations found no signs of infection which leading to infection of the central nervous system, no abnormalities for the EEG examination.

The European League Against Rheumatism (EULAR) issued a consensus recommendation for the management of NPSLE stating that neuropsychiatric manifestations in patients with SLE should be first assessed and treated in the same way as in patients without SLE, including routine symptomatic therapy and psychological interventions. Current practice is mostly symptomatic and includes the use of antipsychotic drugs, antidepressants, and anti-anxiety medications to treat psychiatric symptoms as well as antiepileptic drugs to treat seizures, and immunosuppressive agents (e.g., corticosteroids, cyclophosphamide, azathioprine, mofetil mycophenolate) to suppress the systemic inflammatory response.[17,20] As in this case, the patient was given therapy for seizures first before knowing the cause of the seizures due to the emergency state by giving Phenytoin 100mg x 7 ampoules using a syringe pump for 15 minutes; 3x1 ampoules were subsequently given. Regarding the SLE history, she was prescribed methylprednisolone as the immunosuppressive agent. Then other supportive therapy are also given based on the complaints experienced and the findings of the examination such as mecobalamin, vitamin B6, metamizole, cefobactam, KCL, omeprazole, and inpepsa.

## CONCLUSSION

A case of coexistence of systemic lupus erythematosus (SLE) with involvement of the brain has been reported in 18-year-old female patient who has suffered from SLE for a year with the clinical manifestation of tonic clonic seizure. The diagnosis of this case was established after a series of examinations so we could exclude other possible diagnosis. The management in this case was the same as what is written in the guideline, the first therapy given was the emergency therapy for seizures accompanied by symptomatic and immunosuppressive therapy. The

patient's condition improved after therapy. Further research is needed to determine the vary clinical manifestations of cerebral lupus or NPSLE, further examination findings, and the therapy because single-case observations have limitations.

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