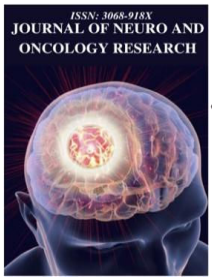

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Case Report

Neuropsychiatric Lupus Associated with Ischemic Stroke: A Case Report from Lome

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Abstract

Neuropsychiatric Systemic Lupus Erythematosus (NPSLE) is a severe manifestation of Systemic Lupus Erythematosus (SLE) that can affect multiple organs, including the central nervous system. Acute ischemic stroke is one of the most common neurological manifestations of SLE and may be due to a hypercoagulable state caused by antiphospholipid syndrome, embolism from endocarditis, atherosclerosis or cerebral vasculitis. We report the case of a 45-year-old man presenting with rapidly progressive left hemiplegia over three days. His history included two episodes of behavioral disturbances with visual hallucinations and dysautonomia, including erectile dysfunction, over the preceding six months. Examination revealed weight loss, confusion, left-predominant pyramidal signs, dysphagia, right-sided anhidrosis, frontal syndrome, left cranial nerve VI palsy and vertical gaze paralysis. Brain MRI showed multiple T2 and FLAIR hyperintensities in the right thalamo-hypothalamic region, posterior limb and knee of the internal capsule and right mesencephalo-pontine region. Laboratory investigations revealed elevated ESR (90 mm/h), reduced creatinine clearance (28.7 mL/min) and 24-hour proteinuria >0.5 g/24 h. Cerebrospinal fluid analysis showed hyperproteinorachia without pleocytosis. Antinuclear antibodies were positive at 1:160 (speckled). Based on the American College of Rheumatology criteria (score 11), the diagnosis of neuropsychiatric lupus was established, revealed by an ischemic stroke due to an anterior choroidal artery infarction. Infectious encephalitis and multiple sclerosis were excluded. The patient was treated with corticosteroids and antiplatelet therapy.

Keywords: Neuropsychiatric Lupus; Ischemic Stroke; Togo

Introduction

Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease that can severely affect multiple organs, including the nervous system, resulting in Neuropsychiatric Systemic Lupus Erythematosus (NPSLE) [1]. Manifestations of SLE in the Central Nervous System (CNS) range from subtle cognitive dysfunction to acute confusional states, psychosis, seizure disorders and stroke [2]. Diagnosis often requires a multidisciplinary approach with multiple assessments, including laboratory tests, imaging and neuropsychological evaluations [1]. The true prevalence of NPSLE is unknown, but published estimates suggest it affects between 12% and 95% of patients with SLE [2]. In Togo, few studies have been published on NPSLE: a hospital-based clinical study reported an SLE prevalence of 0.19% and a case of NPSLE presenting with lupus headache and right-sided hemicorporeal paresthesia has been described [3,4]. We report this case of neuropsychiatric lupus characterized by a striking and polymorphic clinical presentation.

Case Report

A 45-year-old right-handed male merchant was admitted for a rapidly progressive left-sided motor deficit over three days. His personal history was notable for an undocumented seizure in 2023. He is polygamous, has no surgical history and denies alcohol or tobacco use. There is a family history of inflammatory polyarthralgia in his mother and one sister. Six months prior to admission, the patient experienced a brief episode of psychotic behavior with visual hallucinations that spontaneously resolved within 48 hours. Two months before admission, he had a recurrence of behavioral disturbances and visual hallucinations, associated with progressive dysautonomia, including erectile dysfunction. The rapid onset of left-sided motor deficit ultimately prompted his family to seek medical care. On initial examination, he had a low-grade fever of 38°C, vital signs within normal limits, a weight of 53 kg, height 1.72 m (BMI 17.9 kg/m²) and multiple scattered hypopigmented macules. Neurological examination revealed a confusional state, bilateral pyramidal syndrome more pronounced on the left (muscle strength 0/5, proportional) and differential diagnoses included infectious and/or inflammatory encephalitis, stroke or metabolic/toxic encephalopathy. Cranial Computed Tomography (CT) with and without contrast showed no density abnormalities. Cerebrospinal Fluid (CSF) analysis revealed clear fluid, normal glucose, elevated protein at 1.8 g/L (normal 0.15-0.25g/L), 6 cells/µL (60 % neutrophils), negative culture, negative GeneXpert for Mycobacterium tuberculosis and oligoclonal band testing in CSF was not performed. Laboratory tests showed a normal complete blood count, elevated Erythrocyte Sedimentation Rate (ESR) at 90 mm/h (normal <20 mm/h), serum creatinine 29 mg/L corresponding to an estimated Glomerular Filtration Rate (eGFR, MDRD) of 28.7 mL/min/1.73 m², 24-hour proteinuria of 588.8 mg (normal <150 mg/24h), serum protein electrophoresis with alpha-1 at 8.3% (normal 2.9-4.9%), urea 1.05 g/L (normal 0.15-0.45), hypercholesterolemia, normal calcium and negative HIV and Treponema pallidum serologies. Empiric treatment for infectious encephalitis was initiated. One week later, his condition worsened with dysphagia, right-sided anhidrosis, frontal syndrome, left abducens nerve palsy, vertical gaze palsy and systemic signs of infection. Brain Magnetic Resonance Imaging (MRI) performed 15 days after admission showed multiple non-systematized T2 and FLAIR hyperintensities (right thalamo-hypothalamic region, genu and posterior limb of the internal capsule, external capsule and right mesencephalo-pontine region), isointense on T1, with enhancement of the right thalamo-hypothalamic lesion (Fig. 1-4). Antinuclear Antibodies (ANA) were positive by indirect immunofluorescence on HEp-20-10 slides at a titer of 1/160 with a speckled pattern. Testing for anti-Smith antibodies, double-stranded DNA (dsDNA), complement (C3/C4) and antiphospholipid antibodies was not performed due to financial constraints.

Based on additional test results, acute bacterial, tuberculous or syphilitic encephalitis were considered but ruled out due to normal CSF appearance, negative cultures, absence of antibiotic exposure, no socio-economic risk factors, negative GeneXpert and negative syphilis testing. Routine labs excluded uremic or hepatic encephalopathy and there was no toxic exposure suggesting toxic encephalopathy.

The clinical course, young age and dissemination of lesions in time and space raised the possibility of multiple sclerosis, but this was excluded based on only two lesion locations (periventricular and infratentorial) out of five proposed in the 2024 McDonald criteria, along with positive ANA.

Ischemic stroke was suspected due to the rapidly progressive neurological deficit and confirmed by FLAIR hyperintensities in the territory of the right anterior choroidal artery on MRI. After ruling out most differential diagnoses and based on the 2019 American College of Rheumatology (ACR) criteria with a score of 11, the diagnosis of neuropsychiatric lupus was established, revealed by an ischemic stroke due to an anterior choroidal right artery infarction.

Therapeutically, on hospital day 17, the patient received intravenous methylprednisolone 1 g daily for 5 days. Clinical evolution was favorable, allowing discharge on day 27. Oral prednisone at 60 mg/day was continued for 1 month, then tapered off, combined with aspirin 100 mg/day for secondary ischemic stroke prevention. After five months of therapy, clinical evolution remained favorable despite poor adherence. Confusional state and dysphagia resolved, oculomotor palsy improved and left-sided muscle strength increased from 0/5 to 4/5.

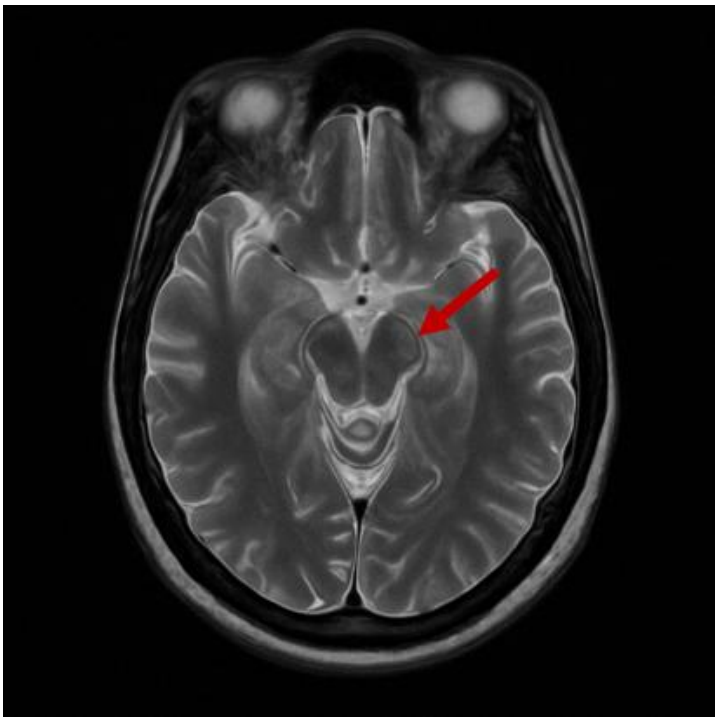

Figure 1: Axial T2-weighted brain MRI showing a hyperintense signal in the right mesencephalon.

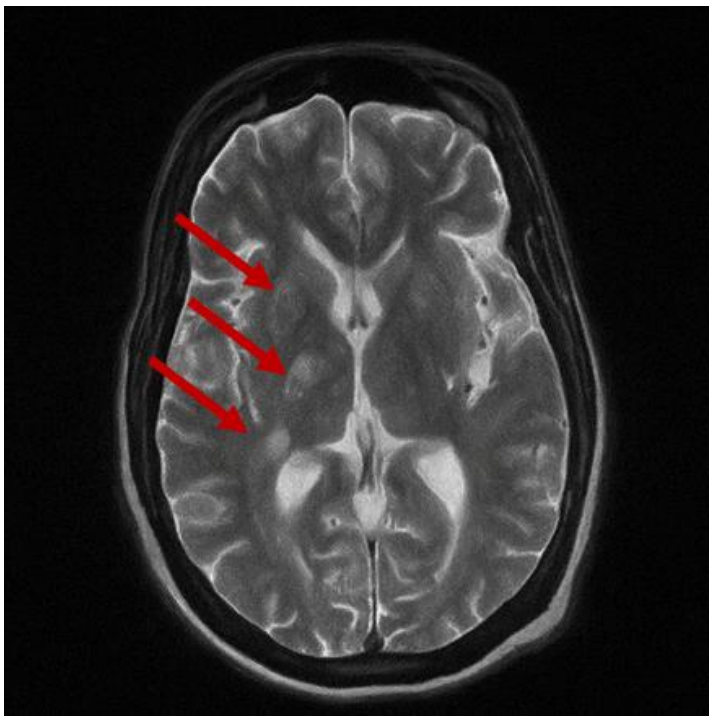

Figure 2: Axial T2-weighted brain MRI showing hyperintense signals in the right thalamus and posterior limb of the internal capsule.

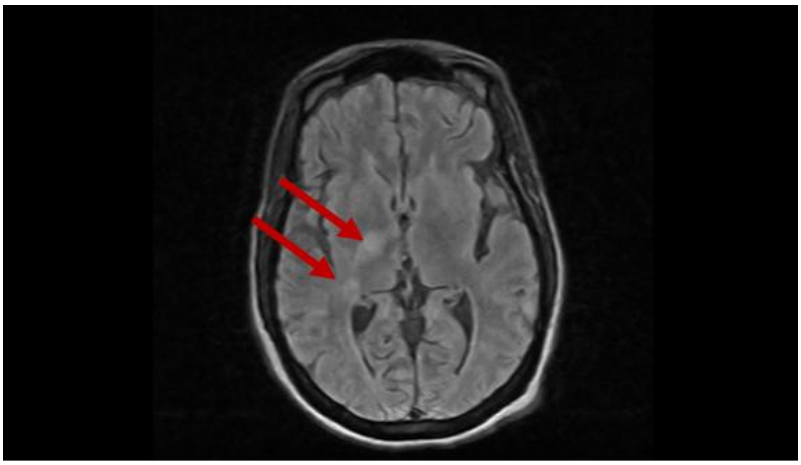

Figure 3: Axial FLAIR brain MRI showing hyperintense signals in the right thalamus and posterior limb of the internal capsule.

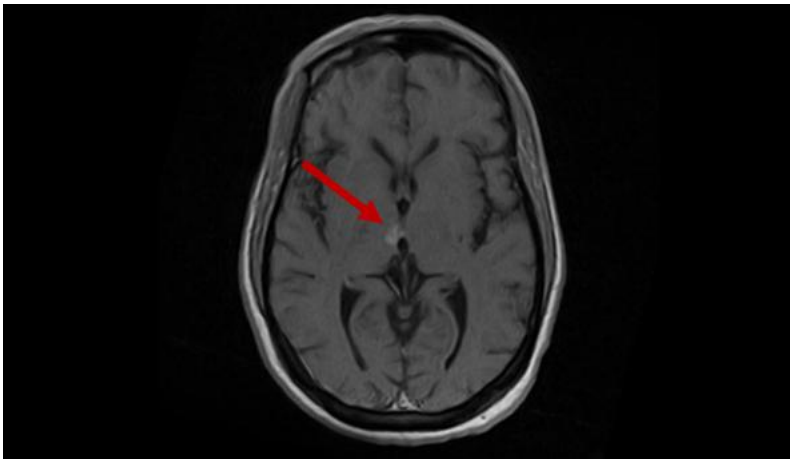

Figure 4: Axial T1-weighted brain MRI with gadolinium showing enhancement of the right thalamic lesion.

Discussion

The prevalence of NPSLE in SLE is estimated at 56.3% [5]. Similar data on NPSLE are reported in Africa. Adelewo, et al., in Nigeria reported a frequency of 51.6%, while Mapouré, et al., in Cameroon found a frequency of 55.5% [6,7]. Headache is the most frequent manifestation of neuro-lupus, occurring in 28.3% of cases [8]. Psychosis, peripheral neuropathy, acute confusional state and severe cognitive dysfunction are less frequent syndromes, with an occurrence rate of 3-5% [8]. Chorea or movement disorders, aseptic meningitis, cranial neuropathy, mononeuritis multiplex, demyelinating syndrome, Guillain-Barré syndrome, autonomic disturbances, myasthenia gravis and plexopathy are rare syndromes (<1-2%) [8]. NPSLE can precede the onset of lupus or occur at any time during its course [9].

The neurological and psychiatric symptoms in this patient were polymorphic and varied, including a non-documented seizure one year before other manifestations, left hemiplegia, confusional state, oculomotor disorder, dysautonomia and recurrent behavioral disturbances. It is possible that the autoimmune disease in this patient began with this seizure or even earlier; the precise onset of the disease is difficult to determine given the absence of prior investigations.

Single tonic-clonic seizures are common and related to SLE activity, typically appearing early in the disease course and recurrence is rare in SLE patients [10]. If recurrence occurs, it generally happens within the first year [11]. Mapouré, et al., found that NPSLE was present at the initial diagnosis of SLE in 37.0% of patients and occurred within the first year in 18.5% of cases [7]. When NPSLE was initial, CNS involvement was dominant, with demyelinating syndrome in 27.8%, headache in 27.5% and seizures in 10.0% of cases [7]. In the absence of specific diagnostic biomarkers, the clinical context in this patient suggested differential diagnoses of infectious encephalitis (specific or common pathogens), which were ruled out due to negative tests; inflammatory encephalitis was also considered due to the markedly elevated erythrocyte sedimentation rate; stroke was considered due to focal neurological deficit and by MRI signal abnormalities.

In sub-Saharan Africa, where the causes of meningoencephalitis are predominantly infectious, diffuse neurological syndromes generally do not reflect active CNS lupus [12]. Patel, et al., report that factors associated with lupus psychosis include prior neuro-lupus events, male sex, younger age at SLE diagnosis (per 10 years) and African ancestry [1]. Acute ischemic stroke is one of the most common neurological manifestations of SLE, affecting 3-20% of patients and may be due to a hypercoagulable state caused by antiphospholipid syndrome, embolism from endocarditis, accelerated atherosclerosis or cerebral vasculitis [1].

MRI signal abnormalities are common in lupus. Authors report that 69% of patients showed MRI abnormalities, whether neuropsychiatric signs were present (73.3%) or absent (64.3%) [13]. These abnormalities include posterior demyelinating lesions associated with enlarged perivascular spaces [13]. The 2019 EULAR/ACR classification criteria for SLE, with a sensitivity of 96.1% and specificity of 93.4%, require at least one positive ANA as an entry criterion, followed by additive weighted criteria across seven clinical domains (constitutional, hematologic, neuropsychiatric, mucocutaneous, serosal, musculoskeletal, renal) and three immunologic domains (antiphospholipid antibodies, complement proteins, SLE-specific antibodies), weighted from 2 to 10 points [14]. Patients accumulating ≥10 points are classified as having SLE [14]. This patient achieved 11 points and was therefore classified [14].

The use of glucocorticoids and other immunosuppressants has been studied as potential therapy for NPSLE. Prednisone is a synthetic glucocorticoid converted to active prednisolone in the liver, with moderate potency and a short half-life of approximately 3-4 hours; Prednisone is generally administered orally at doses of 0.5-1 mg/kg/day for NPSLE. Aspirin is a Non-Steroidal Anti-Inflammatory Drug (NSAID) with analgesic, antipyretic, anti-inflammatory and antiplatelet effects. It can be used in NPSLE to prevent thrombotic events [15].

The patient received intravenous methylprednisolone 1 g daily for 5 days. Clinical evolution was favorable, allowing discharge on day 27. Oral prednisone at 60 mg/day was continued for 1 month, then tapered off, combined with aspirin 100 mg/day for secondary ischemic stroke prevention.

This observation highlights an underdiagnosed condition in sub-Saharan Africa, which may be explained by the reliance on prior SLE diagnosis, limited access to specific investigations and socioeconomic factors.

Conclusion

This case emphasizes that in the presence of polymorphic neurological manifestations accompanied by laboratory evidence of systemic inflammation, systemic autoimmune diseases should be investigated. Given that Systemic Lupus Erythematosus (SLE) is the autoimmune disorder with the most frequent neuropsychiatric manifestations, it should be considered first. Screening for Antinuclear Antibodies (ANA) by indirect immunofluorescence is recommended and, whenever possible, should be complemented by testing for anti-double-stranded DNA (anti-dsDNA) and Extractable Nuclear Antigen (ENA) antibodies.

Conflict of Interest

The investigators declare no material or financial conflict of interest related to this study.

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