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Case Report **Medicine**

Successful Treatment of Lupus Encephalitis in an Elderly Woman at the "Polyclinique RIVIERA" of Bamako

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Abstract

Introduction: Recent epidemiological data demonstrate that disease flares often occur without apparent cause but there is evidence that certain environmental factors may trigger the disease such UV light, infections, certain hormones, and drugs. Here, we report a case of systemic lupus erythematosus flare with neuropsychiatric manifestations notably lupus encephalitis, lupus headache, movement disorder, mood disorder, anxiety and cardiovascular manifestation as pulmonary embolism triggered by malaria and urinary infection with Escherichia coli in an elderly Malian woman at the "Polyclinique REVIERA" of Bamako that had been successfully treated. ***Clinical Observation:*** An 79-year-old Malian female with history of systemic lupus erythematosus currently under azathioprine, arterial hypertension under candesartan, three episodes of erysipelas, chronic gastritis and a recent history of pulmonary embolism was hospitalized to the "Polyclinique REVIERA" of Bamako with a 5-days history an altered level of consciousness, temporospatial disorientation, broca's aphasia preceded by a 10-days history of nausea, vomiting, anorexia, epigastric pain, pain in joints and fever and a 1-month history of headache developed and persisted despite investigation and treatment associated with insomnia, anxiety, anhedonia, difficulty in concentrating and loss of energy. Neurologic examination was markedly for broca's aphasia, myoclonia, seizure, and the Glasgow scale was 07/15. Rheumatologic examination revealed pain and tumefaction left elbow joint, but no deformation of the joints. The dermatological examination noted erythematous-squamous placard on 1/3 of the legs, alopecia and intertrigo. The initial SLEDAI score assessment noted a very high activity of diseases with more than 20 points. A diagnosis of systemic lupus erythematosus flare with neuropsychiatric manifestations notably lupus encephalitis, lupus headache, movement disorder, mood disorder, anxiety and cardiovascular manifestation as pulmonary embolism triggered by malaria and urinary infection with *Escherichia coli* was considered. The treatment with prednisone at a dose of 1 mg per kilogram of body weight a day with 1-year tapering course associated with adjuvant treatments preceded by a bolus of 600 mg of methylprednisone was initiated. Azathiopurine at a dose of 75 mg a day was maintained. Hydroxycloroquine at a dose of 400 mg a day was added. Artesunate 120 mg and imipenem cilastatin 500 mg/500 mg were prescribed and adequately administered. Her anterior medications were continued such candesartan 16 mg and rivaroxaban 10 mg. The fifteenth hospital day SLEDAI score assessment noted a mild activity of diseases with 5 points. The patient was discharged with 2-week follow up visit appointment. ***Conclusion****:* Our case highlights the importance of discussing lupus encephalitis in any case of encephalitic syndrome, especially after having ruled out infectious and neoplastic causes.

Keywords: Lupus encephalitis, lupus flare, neuropsychiatric syndrome, neurolupus, encephalitic syndrome.

INTRODUCTION

Systemic lupus erythematosus (SLE) is an autoimmune disease in which the body's immune system mistakenly attacks healthy tissue in many parts of the body. Often there are periods of illness, called flares, and periods of remission during which there are few symptoms [1].

Recent epidemiological data demonstrate that disease flares often occur without apparent cause but there is evidence that certain environmental factors may trigger the disease such UV light, infections, certain hormones, and drugs [2].

Prevalence of systemic lupus erythematosus varies between countries from 20 to 70 per 100,000.

Women of childbearing age are affected about nine times more often than men. [3, 4]. Indeed, according to the American College of Rheumatology (ACR) case definitions for 19 neuropsychiatric syndromes in systemic lupus erythematosus, there is a high prevalence of neuropsychiatric manifestations in a population-based sample of patients with systemic lupus erythematosus but most neuropsychiatric syndromes were classified as minor [5].

Here, we report a case of systemic lupus erythematosus flare with neuropsychiatric manifestations notably lupus encephalitis, lupus headache, movement disorder, mood disorder, anxiety and cardiovascular manifestation as pulmonary embolism triggered by malaria and urinary infection with Escherichia coli in an elderly Malian woman at the "Polyclinique REVIERA" of Bamako that had been successfully treated.

CLINICAL OBSERVATION

A 79-year-old Malian female was hospitalized to the "Polyclinique REVIERA" of Bamako with 5-days history an altered level of consciousness, temporospatial disorientation, broca's aphasia. These were preceded by a 10-days history of nausea, vomiting, anorexia, epigastric pain, pain in joints and fever. In addition, a 1-month history of headache developed and persisted despite investigation and treatment associated with insomnia, anxiety, anhedonia, difficulty in concentrating and loss of energy. She denied cough, mictional burning but pollakuria was reported. One month before the presentation, the pulmonary embolism was diagnosed by her cardiologist and treated with rivaroxaban. She had a long past history of systemic lupus erythematosus currently under azathioprine, arterial hypertension under candesartan, more than three episodes of erysipelas and chronic gastritis.

The physical examination revealed a temperature of 38.9°C, a heart rate of 115 beats per minute, a respiratory rate of 29 cycles per minute, and a Body Index Mass (BMI) of 25.68 kilogram per square meter. Neurologic examination was markedly for broca's aphasia, myoclonia, seizure, and the Glasgow scale was 07/15. Rheumatologic examination revealed pain and tumefaction left elbow joint, but no deformation of the joints. The dermatological examination noted erythematous-squamous placard on 1/3 of the legs, alopecia and intertrigo. The breath sounds were heard without bibasilar fine crackles. The digestive examination noted decayed teeth and presence of dentures.

Initial laboratory results were notable for a markedly elevated peripheral-blood white-cell count (16,200 cells per cubic millimeters, reference range: 4,000 to 10,000) with 90% neutrophils, 4% lymphocytes and 5% monocytes. The erythrocyte sedimentation rate (ESR) was 99 mm at the first hour (normal range, 0 to 29 millimeter) and the blood C-reactive protein level was 173 mg per liter (normal value, < to 6 mg per liter). The infectious assessment was remarkable. The thick drop examination was positive with 160 trophozoites per cubic millimeter. Bacterial urinary test isolated *Escherichia coli* was sensitive to colistin, imipenem and amykacin. Blood test for dengue was negative. The results of other laboratory studies are shown in table 1. Brain computed tomography scan revealed no cerebrovascular lesions. Cerebrospinal fluid examination, electroencephalogram and brain magnetic resonance imaging were not performed. The initial SLEDAI score assessment [6-8] noted a very high activity of diseases with more than 20 points (table 2).

A diagnosis of systemic lupus erythematosus flare with neuropsychiatric manifestations notably lupus encephalitis, lupus headache, movement disorder, mood disorder, anxiety and cardiovascular manifestation as pulmonary embolism triggered by malaria and urinary infection with *Escherichia coli* was considered.

The treatment with prednisone at a dose of 1 mg per kilogram of body weight a day with 1-year tapering course associated with adjuvant treatments preceded by a bolus of 600 mg of methylprednisone was initiated. Azathiopurine at a dose of 75 mg a day was maintained. Hydroxycloroquine at a dose of 400 mg a day was added. Artesunate 120 mg and imipenem cilastatin 500 mg/500 mg were prescribed and adequately administered. Her anterior medications were continued such candesartan 16 mg and rivaroxaban 10 mg.

On the fifteenth hospital day, general and neurological signs markedly improved. The patient was afebrile. The blood white-cell count fell to 2, 100 cells per cubic millimeter after 10 day antibiotic courses. The erythrocyte sedimentation rate (ESR) was 29 mm at the first hour (normal range, 0 to 29 millimeter) and the blood C-reactive protein level was 19 mg per liter (normal value, < to 6 mg per liter). Albumin was 32.7 g per liter (normal range, 39.7 to 49.4). 24 hours' proteinuria was normal. Chemical and cytological urine test was unremarkable for red-cells, white-cells, protein, but cylindria testing was not possible. A blood tests for anti-native DNA antibody was 1.09 UI per milliliter (normal value, < to 30). Additional serologic testing for C3 and C4 were negative, but CH50 was not tested. The fifteenth hospital day SLEDAI score assessment [6, 7, 8] noted a mild activity of diseases with 5 points (table 2). The patient was discharged with 2-week follow up visit appointment.

Table 1: Laboratory studies on admission and on the fifteenth hospital day

Variables	On admission	On the fifteenth hospital day	Normal range/ normal value
Hematological testing			
Hemoglobin (g per deciliter)	11.1	9.4	12 to 16
Mean corpuscular volume	83.1	79.6	80.0 to 100.0
Mean corpuscular hemoglobin concentration (pictogram)	31.1	29.0	27.0 to 32.0
Blood white-cell count (cell per cubic millimeter)	16,200	2,100	4,000 to 10,000
Neutrophils count (cell per cubic millimeter)	14,000	1,100	2,000 to 7,000
Lymphocyte count (cell per cubic millimeter)	600	800	800 to 4,000
Platelet count (cell per cubic millimeter)	247,000	407,000	150,000 to 500,000
Biochemical testing			
Erythrocyte sedimentation rate at the first hour (millimeter)	99	29	0 to 29
Blood C-reactive protein (mg per liter)	173	27	< to 6
Calcemia (mmol per liter)	1.86	1.94	2.2 to 2.9
Kaliemia (mmol per liter)	3.76	3.20	3.50 to 5.5
Natremia (mmol per liter)	135.6	143	135 to 145
Creatinine (µmol per liter)	78.6		60 to 123
Urea (mmol per liter)	4.76		2.50 to 8.30
Glyceamia (g per deciliter)	1.16		0.80 to 1.26
Hémoglobine gluquée	6.3		≤ to 6.5
ALAT (UI per liter)	26.0		0.0 to 33.0
ASAT (UI per liter)	38.1		0.0 to 32.0
Taux de prothrombine (%)	97.0		70 to 100
Immunological testing			
Anti-native DNA antibody (UI per milliliter)	1.09		< to 30.00
C3 (g per liter)	0.41		0.8 to 1.8
C4 (g per liter)	0.43		0.16 to 0.48

Table 2: SLEDAI score assessment on admission and on the fifteenth hospital day

Parameters	On admission	On the fifteenth hospital day
Recent onset seizure (yes= 8 No=0)	Yes	No, resolved
Psychosis (yes= 8 No=0)	Yes	No, resolved
Organic brain syndrome (yes= 8 No=0)	No	No
Visual disturbance (yes= 8 No=0)	No	No
New onset sensory or motor neuropathy involving cranial nerves (yes= 8 No=0)	No	No
Lupus headache (yes= 8 No=0)	Yes	No, resolved
New onset stroke (yes= 8 No=0)	No	No
Vasculitis (yes= 8 No=0)	No	No
Arthritis (yes= 4 No=0)	Yes	Yes, improved
Myositis (yes= 4 No=0)	No	No
Heme-granular or RBC urinary casts (yes= 4 No=0)	No performed	Not performed
Hematuria (yes= 4 No=0)	No	No
Pyuria (yes= 4 No=0)	No	No
Proteinuria (yes= 4 No=0)	No	No
Inflammatory-type rash (yes= 2 No=0)	No	No
Alopecia (yes= 2 No=0)	Yes	No, improved
Oral or nasal mucosal ulcers (yes= 2 No=0)	No	No
Pleuritic chest pain with pleural rub/effusion or pleural thickening (yes= 2 No=0)	No	No
Pericarditis (yes= 2 No=0)	No	No
Low complement (yes= 2 No=0)	Not	No
Increased anti-native DNA antibody (yes= 2 No=0)	Not performed	No
Temperature > 38°C(yes= 1 No=0)	Yes	No
Platelets < 100×10^9 per liter	No	No
Blood white-cells < 3×10^9 per liter	No	Yes

In summary	31 points/105 indicating a very high activity of disease (despite some paraclinical tests are not performed)	05/105 indicating mild activity of disease (despite some paraclinical tests are not performed)

DISCUSSION

This case report describes a pushed of systemic lupus erythematosus with neurological manifestations (lupus encephalitis and neuropsychiatric syndrome in systemic lupus erythematosus as lupus headache, movement disorder, mood disorder, anxiety) and cardiovascular manifestation (pulmonary embolism) triggered by malaria, urinary infection with *Escherichia coli* revealed by encephalitic syndrome, febrile syndrome, recent cardiovascular event.

Soo et al. describe a case of Striatal Lupus Encephalitis revealed by in bilateral ocular occlusive vasculitis, acute right-sided hemiparesis and facial asymmetry and confirmed Magnetic resonance imaging (MRI) disclosing bilateral symmetrical T2-weighted and fluid-attenuated inversion recovery (FLAIR) hyperintense signals in the basal ganglia, consistent with striatal lupus encephalitis. Authors declare that this case underscores the importance of a comprehensive diagnostic approach, integrating clinical, serological, and neuroimaging findings to differentiate striatal lupus encephalitis from other neuropsychiatric conditions associated with SLE [9].

Kano et al. in 2009 report a case of limbic encephalitis associated with systemic lupus erythematosus revealed by general fatigue, seizures and memory loss and confirmed by Magnetic resonance imaging of the brain showing a high signal area in the mesial temporal lobe bilaterally and the negativity of antibodies to antineuronal antibodies (anti-Hu, anti-Ta and anti-Ma) and antibodies to voltage-gated potassium channels and tumour marker [10].

Clearly, the lupus encephalitis should be defined as a sub-nosological entity of encephalitis exclusively explained by systemic lupus erythematosus flare i.e. encephalitis secondary to systemic lupus erythematosus. In our case, despite brain magnetic resonance imaging, cerebrospinal fluid examination and antibodies to antineuronal antibodies were not performed, the lupus encephalitis is considered because of encephalitic syndrome (headache, disorientation, loss of consciousness, speech disorder, seizure), lupus flare with very high activity of disease, absence of other central nervous system disease that could explained, general and neurological signs markedly and rapidly improved with lupus flare treatment.

The prognosis is typically worse for men and children than for women; however, if symptoms are present after age 60, the disease tends to run a more benign course. Early mortality, within five years, is due to organ failure or overwhelming infections, both of which can be altered by early diagnosis and treatment [11]. The present case is described in elderly Malian woman.

The cause of SLE is not clear [1]. It is thought to involve a combination of genetics and environmental factors [4]. Among identical twins, if one is affected there is a 24% chance the other one will also develop the disease [1]. Female sex hormones, sunlight, smoking, vitamin D deficiency, and certain infections are also believed to increase a person's risk [4]. Malaria and urinary infection with Escherichia coli are the two triggering factors of lupus flares in our case.

Despite some encouraging results for anti-double-stranded DNA antibodies, anti-C1q antibodies, B-lymphocyte stimulator and tumour necrosis factor-like weak inducer of apoptosis, none of the biomarkers stood out from the others as a potential gold standard for flare prediction [12]. These predictive biomarkers are not searched in our patient.

CONCLUSION

Our case highlights the importance of discussing lupus encephalitis in any case of encephalitic syndrome, especially after having ruled out infectious and neoplastic causes. It should be considered a priori in cases of lupus flare-ups with encephalitic syndrome. Lupus encephalitis, a rare and severe neurological manifestations of systemic lupus etrythematosus, should not go unnoticed by physicians managing this case, internist, rhumatologist and neurologist. The early diagnosis of lupus flare improves the pronosis and the disease burden.

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