# ISCHEMIC STROKE IN SYSTEMIC LUPUS ERYTHEMATOSUS - A CASE REPORT

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**ABSTRACT**

Systemic lupus erythematosus (SLE) is an autoimmune disorder affecting many organs, including the central nervous system. Cardiovascular morbidity and mortality are frequent complications, stroke occurring in 3-20% of patients. In this paper we present the case of a 34-year-old woman known with SLE for 12 years, incompliant to the immunosuppressive therapy, that presented two large ischemic strokes, with fatal outcome. Although the overall prognosis for SLE patients has improved in recent decades, the premature cardiovascular disease remains a major cause of death in SLE, even when the traditional risk factors are under control.
**Key words:** stroke, ischemia, systemic lupus erythematosus.

## BACKGROUND

Systemic lupus erythematosus (SLE) is an autoimmune disorder that affects many organs, including the central and peripheral nervous system, cardiovascular morbidity and mortality being a frequent complication, particularly in females aged 35-44 years. Central nervous system (CNS) involvement in SLE was first described by Kaposi[1] in 1872, and the first reference of recurrent cerebral ischemia is made by Osler[2] in 1903. Based on the skin vasculitic changes found in SLE, Osler proposed that similar changes in the brain may be responsible for the neurologic impairments. In 1924, Liebman and Sacks[3] reported the endocarditis found in SLE patients, postulating that stroke may be caused by cerebral emboli from the heart and in 1963 Bowie et al.[4] described the association between the lupus anticoagulant and thrombosis. Although the disease was intensely studied in the last century, the mechanisms underlying the increased cardiovascular risk are not fully understood; many factors have been proposed to account for the increased risk of cardiovascular events: hypertension, diabetes mellitus, renal failure, increased homocysteine, chronic inflammation, endothelial cell injury, immune complex deposition, antiphospholipid antibodies and therapy (especially corticosteroid use).

## CASE REPORT

We report the case of a 34-year-old woman who was admitted to our clinic with confusion, psychomotor agitation, language disturbances and fever with subacute evolution ongoing for 48 hours. The patient was known with SLE from 1994 and was treated with Prednison (according to her medical records). She was incompliant to the treatment and interrupted her medication 3 months prior to her admission in our department.

The physical findings consisted in fever (39.1°C), hyperpigmentation, malar rash and cubital deviation of the hands.

The neurological examination revealed a conscious patient, agitated, with major global aphasia but no focal signs. She could not understand any order and was unable to say even the simplest words. She tried to communicate through writing but her attempts were unreadable and incomprehensible.

The complete blood cell count showed leukocytosis (16.600/ ul), a normal red blood cell count but with decreased hemoglobin (11,3 g/dl), decreased hematocrit (32,7%), decreased mean red blood cell volume (77,5 / fl) and decreased mean corpuscular hemoglobin concentration (26,8 / pg). There was also thrombocytopenia (103.000 / ul). The biochemical blood tests revealed high glucose

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levels (238 mg/dl). Other laboratory tests showed increased fibrinogen (475 mg/dl) and high levels of C-reactive protein (51 mg/L). On immunological testing, a high level of IgG (24 g/dl), an abnormal titer of anti DNA-antibody (1/160), the presence of lupic anticoagulant and low complement component 3 (C3) (39 mg/100 ml) were found. Positive tests for antibodies to SM nuclear antigen and anti-RNP and anti-Ro/SSA antibodies were also documented.

The MRI scan revealed two large areas with inhomogeneous signal intensity, predominantly with hypo signal on T1 sequences and hyper signal on T2 sequences; one lesion was situated in the right fronto-temporal area and the other in the left fronto-parieto-temporal region. Both lesions had a minimal contrast enhancement and mass effect. These images were suggestive for two large ischemic strokes with hemorrhagic transformation. Also, a minimal subarachnoid hemorrhage was present. Angio-MRI did not reveal any arteriovenous malformation or aneurismal dilatation (see figures 1, 2, 3, 4, 5, 6).

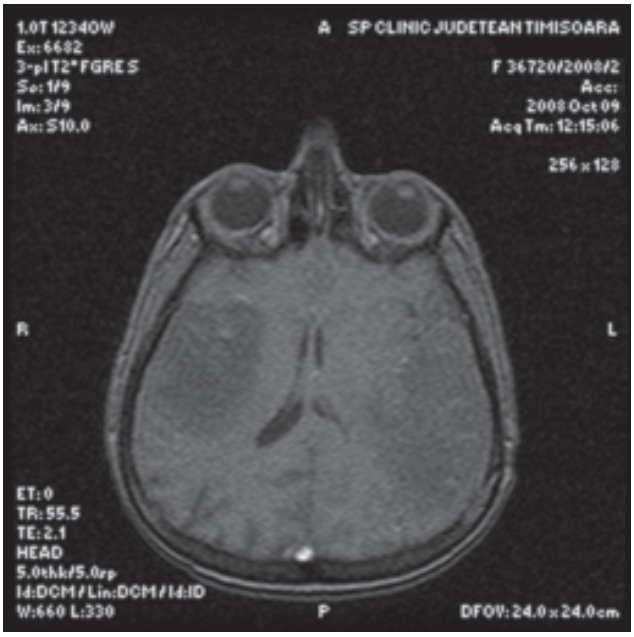


*Figure 1*

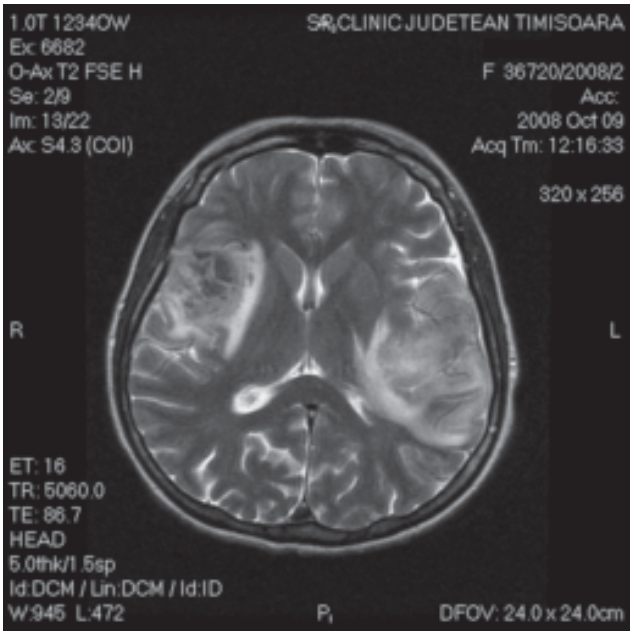


*Figure 2*

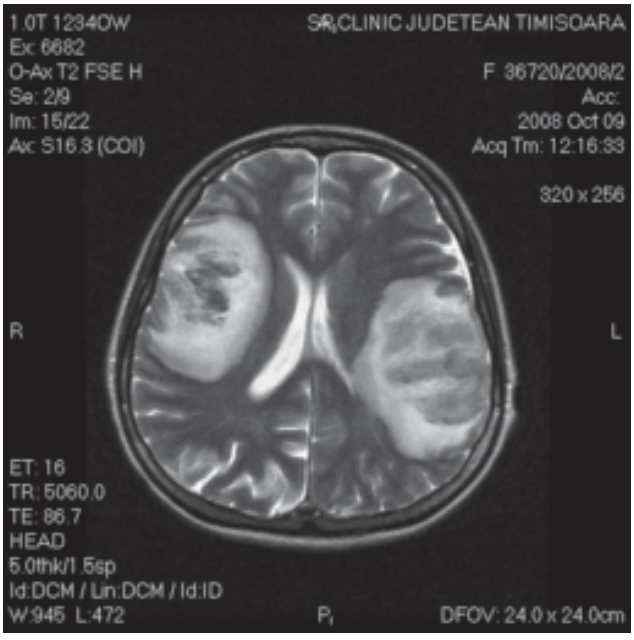


*Figure 3*

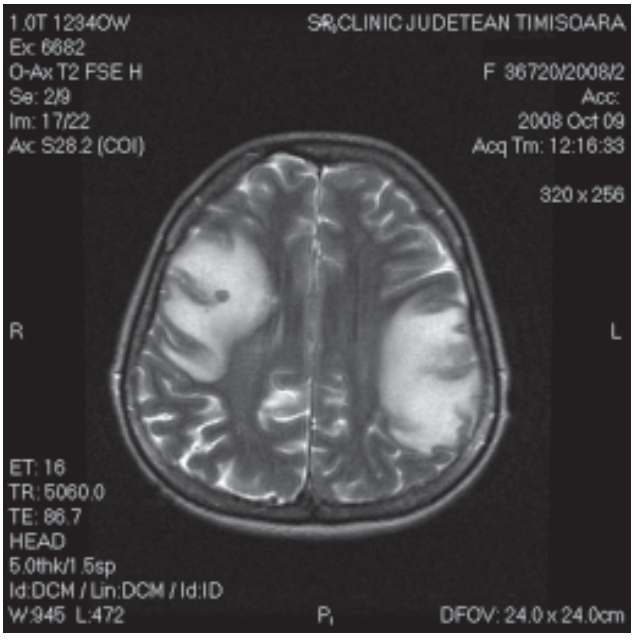


*Figure 4*

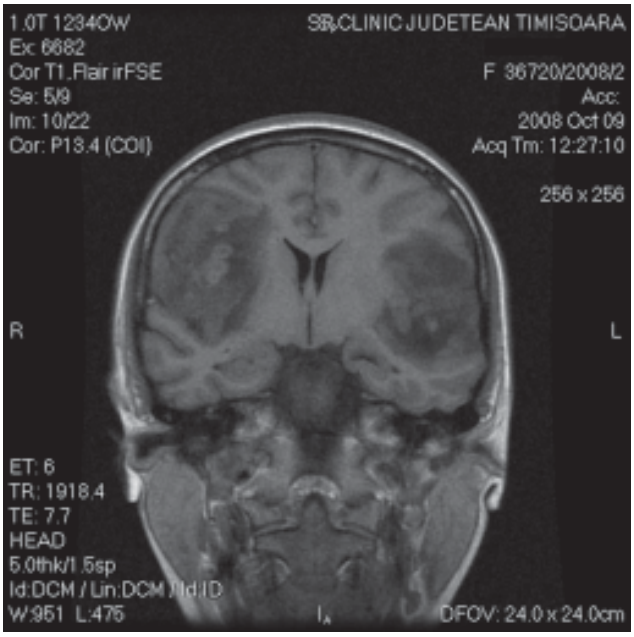


*Figure 5*

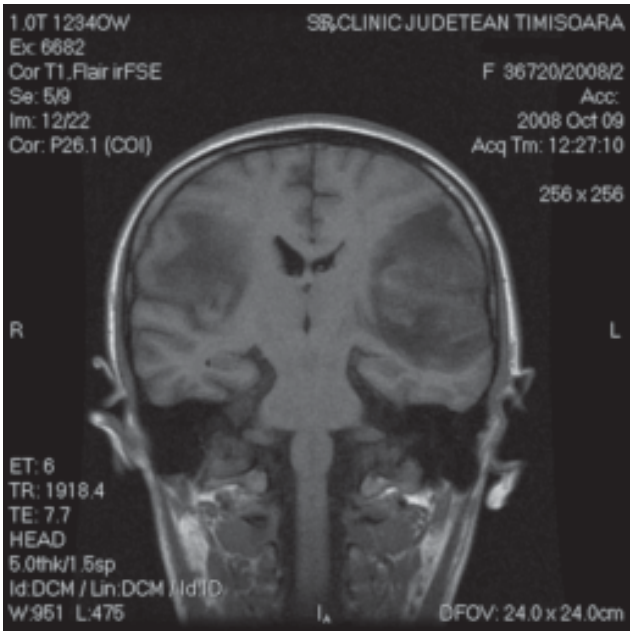


*Figure 6*

The patient received depletive, anticoagulant and immunosuppressant medication (Methylprednisolone "pulse-therapy" for 7 days followed by a cure of Cyclophosphamide) and antibiotics. Her status deteriorated progressively in the next week and she became comatose being transferred to the intensive care unit. The evolution was unfortunately unfavorable, the patient dying two weeks after the hospital admittance.

## CONCLUSIONS

Although the overall prognosis for SLE patients has improved in recent decades, with a 80-95% 10-years survival rate[5], stroke occurs in 3-20% of cases[6]. The neurological complications worsen the prognosis, the principle obstacle in long-term management of the disease being the patient's compliance with immunosuppressive drugs due to their adverse-effects. These patients have a high incidence of atherosclerosis, the premature cardiovascular disease being now a major cause of death in SLE, even after controlling for traditional risk factors. This suggests that both, the disease and the complications of SLE treatment are major causes of cardiovascular disease. Beside the treatment of traditional cardiovascular risk factors, better therapies that address the immune and inflammatory components of the development of atherosclerosis in SLE patients are also necessary[7]. Nevertheless, there are no large-scale clinical trials that could provide a base for some firm recommendations for primary prevention. Such trials are vital in order to provide a rational basis for evidence-based strategies that will enable us to improve the morbidity and mortality of patients with SLE.

## REFERENCES:

1.**Kaposi MK.** – Neue Beitraege zur Kenntniss des Lupus Erythematosus. Arch Dermatol Syph. 1872; 4: 36-78.

2.**Osler W.** – On the visceral manifestations of the erythema group of skin diseases. *Trans Assoc Am Physicians.* 1903; 18: 599-624.

3.**Liebman E, Sacks B.** – A hitherto undescribed form of valvular and mural endocarditis. *Arch Intern Med.* 1924; 33: 701-737.

4.**Bowie EJW, Thompson JH, Pascuzzi CA, Owen CA.** – Thrombosis in systemic lupus erythematosus despite circulating anticoagulants. *J Lab Clin Med.* 1963; 62: 416-430.

5.**Cervera R, Khamashta MA, Font J, Sebastiani GD, Gil A, Lavilla P, Aydintug AO, Jedryka-Goral A, de Ramon E, Galeazzi M, Haga HJ, Mathieu A, Houssiau F, Ruiz-Irastorza G, Ingelmo-M, Hughes GRV** – The European Working Party on Systemic Lupus Erythematosus. Morbidity and mortality in systemic lupus erythematosus during a 5-year period: a multicenter prospective study of 1000 patients. *Medicine.* 1999; 78: 167-175.

6.**Futrell N, Millikan C.** – Frequency, etiology and prevention of stroke in patients with systemic lupus erythematosus. *Stroke.* 1989; 20: 583-591.

7.**Manzi S.** – Systemic lupus erythematosus: a model for atherogenesis? *Rheumatology.* 2000; 39: 353-359.