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Left-hemisphere ischemic stroke as a complication of cutaneous lupus erythematosus

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ABSTRACT

We describe the case of a 33-year-old patient with cutaneous lupus erythematosus admitted to the Department of Neurology due to the weakness of right limbs and speech disorders after night sleep. On admission, neurological examination revealed: no verbal contact; mixed aphasia with motor predominance; wide, symmetrical and reactive pupils; conjugate gaze palsy in a horizontal direction; right-sided hemianopsia and right-sided pyramidal tract syndrome with right limbs paralysis and bilaterally positive Babinski's sign. Magnetic resonance imaging revealed changes in the type of fresh ischemia in the left hemisphere of the brain – in the temporal lobe / basal ganglia on the left side hyperintensive areas were visible. As a result of the therapy and rehabilitation, the patient's general and neurological condition was improved. The patient was discharged home with recommendations in good general condition.

Keywords: cutaneous lupus erythematosus, ischemic stroke

INTRODUCTION

Lupus erythematosus (LE) is associated with a wide range of cutaneous pathologies. Cutaneous symptoms are frequent manifestations of LE, and for some cutaneous LE (CLE) subtypes, they can occur in the absence of signs of systemic disease. CLE is up to three times more common than systemic lupus erythematosus (SLE) [1, 2].

As in the case of SLE, multifactorial etiology of CLE is currently suggested, including autoimmune induction, genetic susceptibility and immune system disorders [1]. CLE symptomatology consists of a wide range of dermatological symptoms, which may or may not be associated with the development of systemic disease [1]. The goal of CLE dermatological treatment is to prevent the formation of new lesions and improve the appearance of the skin by combining patient education, topical therapies and systemic agents. Education about heat, sun and avoiding drugs are essential recommendations [3].

SLE is one of the strongest known risk factors for cardiovascular disease (CVD) [4-6]. CVD is one of the most important causes of death in patients with SLE. The risk of developing both clinical CVD and subclinical atherosclerosis is increased in SLE patients. However, this increase is not fully explained by traditional cardiovascular risk factors (such as hypertension or smoking), and therefore it is suggested that immune system dysfunction also contributes to CVD risk in SLE patients [7]. Studies in multiple units have shown that SLE patients have significantly higher prevalence of atherosclerotic plaque than healthy people [8-11]. Compared with people without SLE, there is also an increased incidence of ischemic stroke in SLE patients [5, 12-15]. The prevalence is in the range of 2-15% [5, 16, 17]. In turn, cerebrovascular events account for 20-30% of deaths in patients with SLE [18, 19].

The study by Krishnan et al. showed that patients with SLE had an approximately 2-fold higher risk of hospitalization because of stroke compared to patients without SLE [19]. Another study revealed that women with SLE were hospitalized with stroke almost 9 times more frequent than the general population [20]. Strokes usually occur within the first five years of the onset of the disease [21]. Therefore, modifiable risk factors (such as hypertension, hyperlipidemia, obesity or smoking) should be treated to target [22].

In this article, we wanted to analyze the case of a patient with CLE and ischemic stroke.

CASE REPORT

A 43-year-old patient was admitted to the Department of Neurology due to the weakness of right limbs and speech disorders after night sleep. The patient additionally reported hypertension, type 2 diabetes mellitus, hypothyroidism and cutaneous lupus erythematosus.

On admission, neurological examination revealed:

- the patient was conscious, circulatory and respiratory efficient with a blood pressure of 140/80 mm Hg and a regular heart rate of about 80 beats per minute;
- no verbal contact; mixed aphasia with motor predominance;
- commands fulfilled selectively;
- negative meningeal symptoms;
- wide, symmetrical and reactive pupils;
- conjugate gaze palsy in a horizontal direction;
- right-sided hemianopsia;
- right-sided pyramidal tract syndrome (facial-limbs) with right limbs paralysis and Babinski's sign present on both sides;
- NIH Stroke Scale (NIHSS): 19 points, Modified Rankin Scale (mRS): 3 points.

The results of laboratory tests at the time of admission to the department are presented in Table 1. In computed tomography (CT) scan of the head performed in the urgent mode, no radiological features of acute stroke were found. Magnetic resonance imaging (MRI) revealed changes in the type of fresh ischemia in the left hemisphere of the brain – within the temporal lobe / basal ganglia on the left side hyperintensive areas were visible in T2 / FLAIR sequences with diffusion restriction as in the case of massive fresh ischaemia up to 40x75 mm (Figures 1–3). A moderate mass effect caused by the described changes was also visible. In addition, the intensity of grey matter and white matter brain signals were normal. Following intravenous administration of a contrast agent, no areas of pathological signal intensification were found.

In ultrasound Doppler examination carotid and vertebral arteries flows were within normal limits. 24-hour Holter blood pressure measurement did not show elevated blood pressure values. Holter electrocardiography (ECG) and echocardiography was also with no abnormalities.

The patient was consulted twice by an internal medicine doctor. The consulting psychologist recommended further observation of the emotional state. As a result of the therapy and rehabilitation, the patient's general and neurological condition was improved. On the day of discharge home, the patient walked without help, had persistent motor aphasia and slight paralysis of right limbs. The patient was discharged with recommendations in good general condition (Table 2).

Table 1. The results of laboratory tests at the time of admission to the department

Parameter	**Value**
ALT [U/L]	53,9
AST [U/L]	39
Total bilirubin [μmol/l]	8,9
CHOLESTEROL CAŁKOWIT [mmol/l]	4,27
CRP quantitatively [mg/l]	13,55
APTT [sec]	21,1
D-dimer [μg/ml]	0,27
fT4 [ng/dl]	1,6
Glucose [mg/dl]	138,6
Serum urea [mmol/l]	2,2
K^+ [mmol/l]	3,99
Na^+ [mmol/l]	141
Troponin T [ng/ml]	0,004
TSH [μIU/ml]	2,96
PT [sec]	11,7
INR	0,9
HCT [%]	31,9
RBC [$x10^6$/μL]	2,88
HGB [g/dl]	10,9
WBC [$x10^3$/μL]	8,25
MCV [fL]	110,8
MCH [pg]	37,8
MCHC [g/dl]	34,2
PLT [$x10^3$/μL]	234

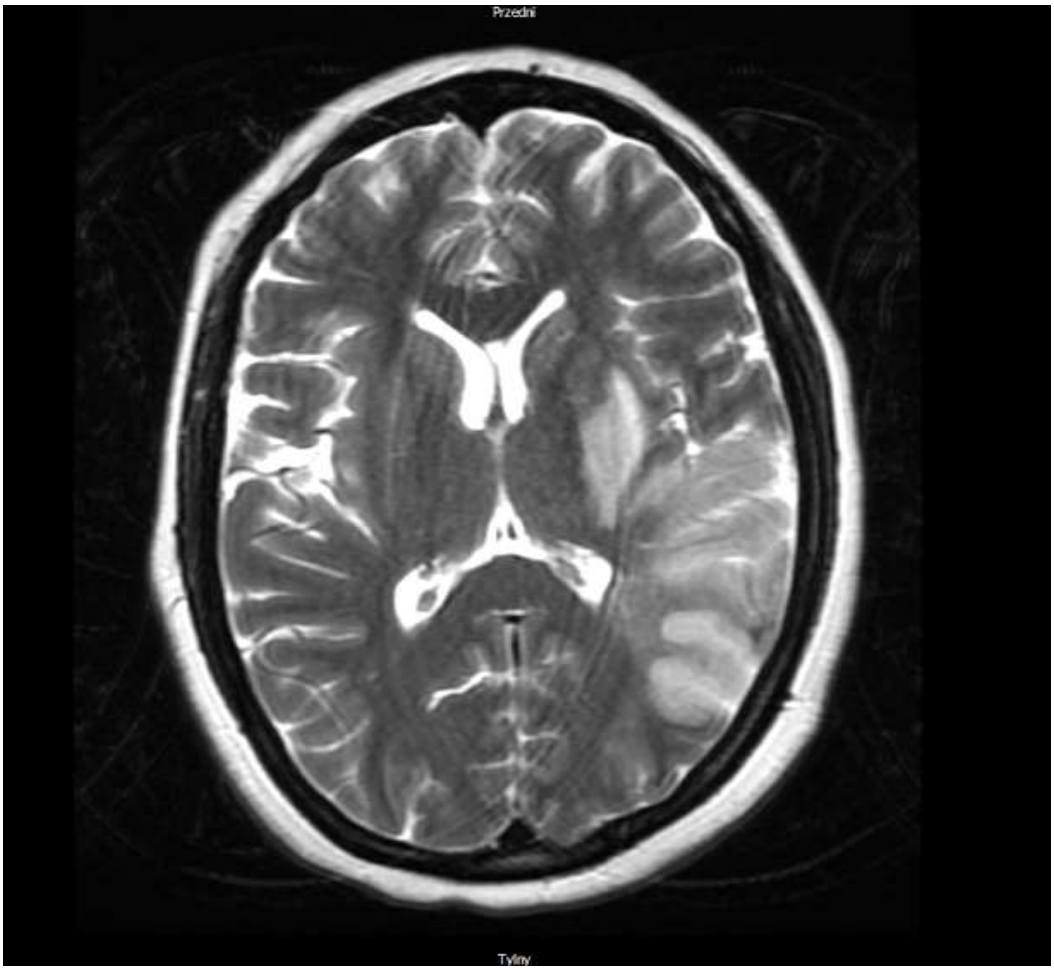

Figure 1. CT of the head (T2-weighted scan) – within the temporal lobe / basal ganglia on the left side hyperintensive areas with diffusion restriction are visible (40x75 mm) [authors' material]

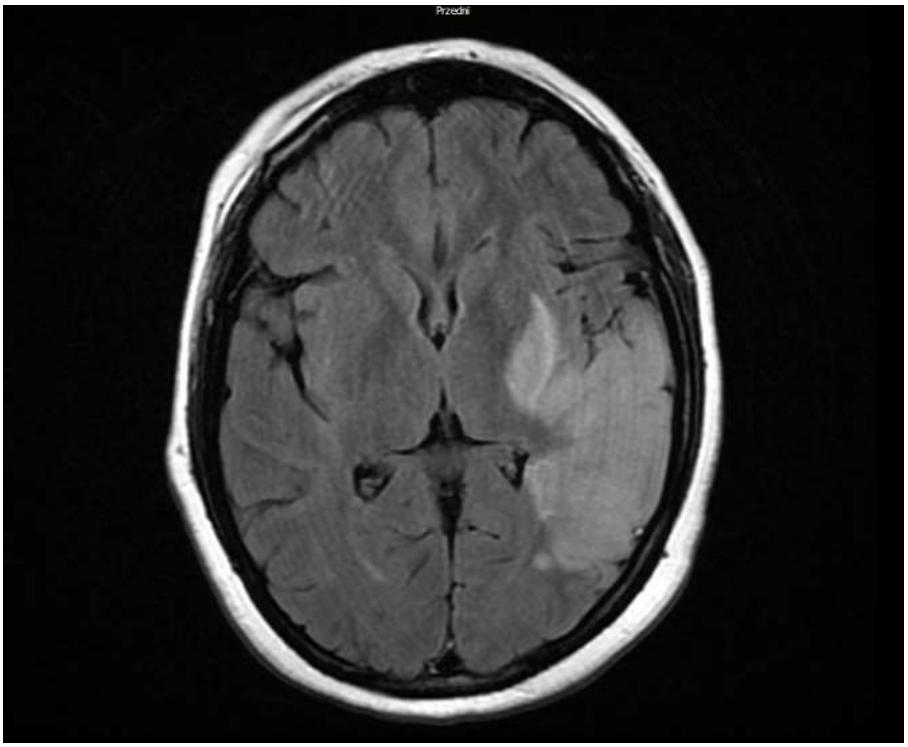

Figure 2. CT of the head (T2 FLAIR; fluid-attenuated inversion recovery) – within the temporal lobe / basal ganglia on the left side hyperintensive areas with diffusion restriction are visible (40x75 mm) [authors' material]

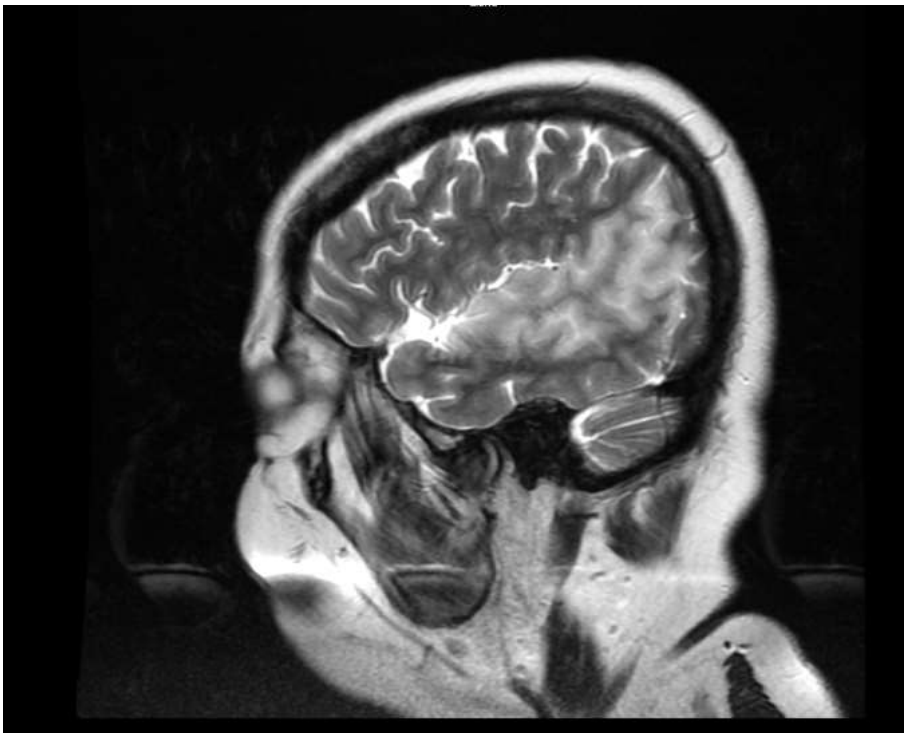

Figure 3. CT of the head (T2 FRFSE; fast-recovery fast spin-echo) – within the temporal lobe hyperintensive areas with diffusion restriction are visible [authors' material]

Table 2. Comparison of the patient's general condition on admission to the Department of Neurology and during discharge home

Type of scale	**On admission to the Department of Neurology**	**During discharge home**
NIH Stroke Scale (NIHSS)	19 points	6 points
Modified Rankin Scale (mRS)	3 points	2 points

DISCUSSION

In SLE patients, the differential diagnosis of stroke is complex. SLE itself rarely causes stroke due to vasculopathy or vasculitis [23]. The most common causes are atherosclerosis, hypertension and antiphospholipid syndrome. Rare causes are reversible posterior encephalopathy syndrome, malignancy and infection. There are also conditions that may mimic stroke in SLE patients – complicated migraines, toxic-metabolic conditions or mental disorders [24].

CLE occurs 2-3 times more often than SLE and affects men more than women, with peak incidence in the seventh decade of life [25]. It is still unknown whether CLE is associated with risk of CVD [26].

In the study by Singh et al. [26], the risk of cerebrovascular accidents in CLE was assessed in a population-based CLE cohort, and compared the risk with a matched non-CLE cohort. 155 patients with CLE were included in the study and during median follow-up of 14.6 years, 41 CLE patients developed vascular events. It turned out, that patients with CLE are not at an increased risk of ischemic heart disease and heart failure, but have an almost 3-fold higher risk of cerebrovascular accidents and a 2.1-fold higher risk of peripheral arterial disease, as compared to matched controls.

CONCLUSIONS

Patients with cutaneous lupus erythematosus are at increased risk of ischemic stroke, which is an important cause of death in this group of patients. In the described case report, a good improvement of the patient's neurological and general condition was obtained.

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