ORIGINAL ARTICLE

Clinical picture of late-onset systemic lupus erythematosus in a group of Polish patients

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KEY WORDS

clinical manifestations, late onset, old age, systemic lupus erythematosus

ABSTRACT

INTRODUCTION The prevalence of late-onset systemic lupus erythematosus (SLE) diagnosed in patients over the age of 50 years is estimated at 10% to 20%. SLE in elderly patients has specific features with misleading signs and symptoms, but its clinical course seems milder compared with that in younger patients.

OBJECTIVES The aim of the study was to assess clinical manifestations of late-onset SLE in a group of patients treated in Poland.

PATIENTS AND METHODS From a group of 230 consecutive patients with SLE, individuals with late-onset disease were selected. We retrospectively analyzed the incidence of clinical features of SLE, concomitant diseases, and treatment. The incidence of clinical features in late-onset patients was compared with that in a group of 108 patients with early-onset SLE.

RESULTS Late-onset SLE was confirmed in 20 patients (8.7%) including 16 women (80%) and 4 men (20%). A delay in diagnosis was 31.7 months (0–144). The most common SLE features were arthritis (50%), rash (40%), nephropathy (40%), photosensitivity (30%), mouth ulcerations (30%), interstitial lung disease (30%), fever (25%), leukopenia (65%), and thrombocytopenia (35%). Kidney involvement was present in all men and in 25% of women. Thrombotic complications were noted in 38.8% of the patients. Concomitant diseases were common in our study group.

CONCLUSIONS The clinical picture of late-onset SLE differs from that of early-onset SLE. Arthritis, leukopenia, and thrombotic complications are frequent, while skin manifestations, photosensitivity, nephropathy, vasculitis, and central nervous system involvement are less common in late-onset SLE. The diagnosis of late-onset SLE is often delayed, and treatment is determined by the presence of concomitant diseases.

INTRODUCTION Systemic lupus erythematosus (SLE) is a chronic autoimmune disease resulting from interactions between genetic and environmental factors and characterized by multiorgan involvement. In the majority of patients, it develops at a young age. The predominance of women at a childbearing age is also typical for SLE.[1-4] However, there is also a subpopulation of patients with late-onset SLE. According to the available literature, late-onset SLE is defined as SLE affecting patients older than 50 years and is observed in about 10% to 20% of patients with SLE. The clinical course of SLE in elderly patients has a number of characteristic features; however, the first signs and symptoms can be misleading and the onset of the disease—insidious. This may be caused by comorbidities related to aging, which are frequently observed in this group of patients, and a delay in the diagnosis of SLE is thus highly probable. In late-onset SLE, the involvement of major organs and systems, such as the kidneys or the central nervous system, is rarely observed. The course of late-onset SLE seems to be milder, although certain contradictory data in the literature can be found.[5-7]

The aim of our study was to assess clinical manifestations of late-onset SLE in a group of patients diagnosed and treated at the University Medical Center, Lublin, Poland, between 2004 and 2014.

PATIENTS AND METHODS Of a group of 230 consecutive Caucasian patients with SLE, we identified 20 patients (8.7%) with late-onset disease, defined as the onset of symptoms at the age of

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TABLE 1 General characteristics of patients with late-onset systemic lupus erythematosus

Variable	Women (n = 16)	Men (n = 4)	Total (n = 20)
age at the onset of first symptoms, y	55.8 (46–69)	58.2 (50–64)	56.3 (46–69)
age at diagnosis, y	56.1 (50–71)	59.0 (50–64)	56.7 (50–71)
delay in diagnosis, months	38.7 (6–144)	4 (0–12)	31.7 (0–144)
body mass index, kg/m^2	26.3 (21–34)	28.9 (25.7–33)	27.6 (21–34)

Data are presented as a mean value (range).

50 years or older. All patients fulfilled at least 4 of SLE classification criteria according to the American College of Rheumatology (ACR, 1997).[8]

In the late-onset group, there were 16 women (80%) and 4 men (20%). We retrospectively analyzed the prevalence of clinical features of SLE in patients with late-onset SLE. This included the analysis of comorbidities and the treatment of SLE.

Descriptive data were presented as a mean value and range; the incidence of clinical features and comorbidities was also reported.

The characteristics of patients with late-onset SLE are presented in **TABLE 1**.

The diagnosis of late-onset SLE was established at disease onset in 8 patients (40%). The average delay in diagnosis was 31.7 months (range, 0–144 months). Before the diagnosis of late-onset SLE, other diseases were considered, including rheumatoid arthritis in 4 patients (20%), Sjögren syndrome in 4 patients (20%), mixed connective tissue disease in 1 patient (5%), undifferentiated arthritis in 1 patient (5%), systemic sclerosis in 1 patient (5%), autoimmune thrombocytopenia in 1 patient (5%), and antiphospholipid syndrome (APS) in 1 patient (5%).

In addition, we selected 108 patients with SLE who fulfilled at least 4 of the 1997 ACR classification criteria,[8] were younger than 50 years old at the time of diagnosis (early-onset SLE), and were treated at our center. We compared the incidence of clinical features and immune disorders between the 2 groups of patients. A statistical analysis was performed using a 2-proportion test (STASTISTICA, version 12.0, StatSoft, United States). A *P* value of less than 0.05 was considered statistically significant.

RESULTS Of 230 consecutive patients with SLE included in this study, 20 patients (8.7%) were diagnosed with SLE at an age of 50 years or older (including 16 women [80%] and 4 men [20%]). The period between the onset and diagnosis of SLE in patients with late-onset disease varied from 0 to 144 months (mean, 31.7 months) and was longer in women (38.7 months; range, 6–144 months) than in men (4 months; range, 0–12 months). This finding might be explained by a more severe course of the disease in men in our study group.

TABLE 2 Clinical manifestations at diagnosis in patients with late-onset and early-onset systemic lupus erythematosus

Manifestation	Systemic lupus erythematosus				*P* value
	late-onset (n = 20)			early-onset (n = 108; 89 women, 19 men)	
	women (n = 16)	men (n = 4)	total (n =20)		
fever	3 (18.75)	2 (50)	5 (25)	31 (40.2)	0.198
weight loss	2 (12.5)	2 (50)	4 (20)	17 (18.7)	0.892
fatigue	–	2 (50)	2 (10)	29 (26.8)	0.107
lymphadenopathy	1 (6.25)	–	1 (5)	2 (1.85)	0.392
photosensitivity	6 (37.5)	–	6 (30)	59 (54.6)	0.043
malar rash	5 (31.25)	1 (25)	6 (30)	62 (57.4)	0.024
discoid lesions	2 (12.5)	–	2 (10)	8 (7.4)	0.691
rash	7 (43.75)	1 (25)	8 (40)	79 (73.1)	0.004
vasculitis	1 (6.25)	–	1 (5)	34 (31.5)	0.015
oral ulcers	5 (31.25)	1 (25)	6 (30)	31 (28.7)	0.906
alopecia	4 (25)	–	4 (20)	44 (40.7)	0.079
Raynaud phenomenon	2 (12.5)	–	2 (10)	16 (14.8)	0.57
myalgia	1 (6.25)	–	1 (5)	19 (17.6)	0.154
arthritis	9 (56.25)	1 (25)	10 (50)	57 (52.7)	0.824
nephropathy	4 (25)	4 (100)	8 (40)	78 (72.2)	0.005
central nervous system involvement	1 (6.25)	1 (25)	2 (10)	36 (33.3)	0.036
peripheral nervous system involvement	1 (6.25)	–	1 (5)	2 (1.85)	0.392
serositis	–	1 (25)	1 (5)	25 (23.1)	0.064
leukopenia	10 (62.5)	3 (75)	13 (65)	70 (64.8)	0.986
thrombocytopenia	5 (31.25)	2 (50)	7 (35)	31 (28.7)	0.571
hemolytic anemia	–	1 (25)	1 (5)	6 (5.5)	0.928

Data are presented as number (percentage) of patients.

TABLE 3 Immunological laboratory test findings in patients with late-onset and early-onset systemic lupus erythematosus

Parameter	Systemic lupus erythematosus				*P* value
	late-onset (n = 20)			early-onset (n = 108; 89 women, 19 men)	
	women (n = 16)	men (n = 4)	total (n =20)		
ANA	16 (100)	4 (100)	20 (100)	108 (100)	–
anti-ds-DNA	10 (62.5)	3 (75)	13 (65)	85 (78.7)	0.184
anti-SS-A	11 (68.75)	1 (25)	12 (60)	48 (44.4)	0.199
anti-SS-B	5 (31.25)	1 (25)	6 (30)	22 (20.4)	0.340
anti-Sm	–	–	–	23 (21.3)	0.023
anti-RNP	–	–	–	28 (25.9)	0.01
anti-Rib P	–	–	–	5 (4.6)	0.328
anti-Scl-70	1 (6.25)	1 (25)	2 (10)	4 (3.7)	0.22
antinucleosomes	6 (37.5)	1 (25)	7 (35)	32 (29.6)	0.63
antihistones	3 (18.75)	1 (25)	4 (20)	26 (24.1)	0.691
ACPA	3 (18.75)	–	3 (15)	5/45 (11.1)	0.659
RF-IgM	5 (31.25)	2 (50)	7 (35)	19/36 (52.8)	0.201
aCl	5 (31.25)	4 (100)	9 (45)	39 (36.1)	0.450
LAC	2 (12.5)	3 (75)	5 (25)	30 (27.7)	0.8
anti-β_2-GPI	–	3 (75)	3 (15)	17/38 (44.7)	0.024
aPL positivity in total	5 (31.25)	4 (100)	9 (45)	50 (46.3)	0.915
hypocomplementemia	6 (37.5)	3 (75)	9 (45)	84 (77.7)	0.026

Data are presented as number (percentage) of patients.

In the early-onset group: ACPA were measured in 45 patients; RF-IgM, in 36 patients; and anti-β_2-GPI, in 38 patients.

Abbreviations: aCl, anticardiolipin antibodies; ACPA, antibodies against cyclic citrullinated peptides; ANA, antinuclear antibodies; anti-β_2-GPI, antibodies against β_2 glycoprotein I; anti-ds-DNA, anti-double-stranded DNA antibodies; aPL, antiphospholipid antibodies; anti-Rib P, antibodies against ribosomal P proteins; anti-RNP, antibodies against ribonucleoprotein; RF-IgM, class IgM rheumatoid factor

Clinical manifestations of SLE are presented in TABLE 2. The most common signs and symptoms in all patients with late-onset SLE were arthritis (50%), rash (40%), nephropathy (40%), photosensitivity (30%), ulcerations in the mouth (30%), fever (25%), leukopenia (65%), and thrombocytopenia (35%). Interstitial lung disease was diagnosed in 30% of the patients. Kidney involvement was present in all men and in 25% of women. Pleuritis was not observed, while pericarditis was present only in 1 male patient (5%). Involvement of the central nervous system was diagnosed in 2 patients (10%) and of the peripheral nervous system—in 1 patient (5%).

All men had an acute onset of SLE, with more common general symptoms at onset and a more severe course of the disease with more common kidney and nervous system involvement, as compared with women. Cardiovascular disease and APS were also more common in men than in women.

Compared with the late-onset group, patients with early-onset SLE more often showed the following manifestations: photosensitivity (54.6% vs 30%, $P = 0.043$), malar rash (57.4% vs 30%, $P = 0.024$), other skin involvement (73.1% vs 40%, $P = 0.004$), vasculitis (31.5% vs 5%, $P = 0.015$), nephropathy (72.2% vs 40%, $P = 0.005$), and central nervous system involvement (33.3% vs 10%, $P = 0.036$).

Abnormalities in immunological laboratory test results are shown in TABLE 3. All patients with late-onset SLE were positive for antinuclear antibodies; anti-double-stranded DNA antibodies (anti-ds-DNA) were positive in 75% of men and 62.5% of women (65% of the patients in total); anti-SS-A were positive in 60% of the patients (68.75% of women; 25% of men). Antinucleosomes were present in 35%; anti-SS-B, in 30%; rheumatoid factor in IgM class (RF-IgM), in 35%; anticardiolipin antibodies, in 45%; and lowered complement concentration, in 45% of the patients. None of the patients were positive for anti-Sm, antiribosomal P protein, or antiribonucleoprotein (anti-RNP) antibodies. All men and 31.25% of women were positive for antiphospholipid antibodies (aPL).

Compared with the late-onset group, patients with early-onset SLE were significantly more often positive for anti-RNP ($P = 0.01$) and anti-Sm ($P = 0.023$) antibodies as well as for antibodies against β_2 glycoprotein I (anti-β_2-GPI; $P = 0.023$). Hypocomplementemia was also more common in younger patients ($P = 0.026$). The statistical analysis of data on the presence of RF-IgM, anti-β_2-GPI, and antibodies against cyclic citrullinated peptides (ACPA) in the early-onset group was performed in a smaller number of patients owing to the lack of relevant data in all patients.

Concomitant diseases were common in the study group (TABLE 4). The most frequent

TABLE 4 Concomitant diseases in patients with late-onset systemic lupus erythematosus

Disease	Women (n = 16)	Men (n = 4)	Total (n = 20)
antiphospholipid syndrome	2 (12.5)	3 (75)	5 (25)
Sjögren syndrome	6 (37.5)	1 (25)	7 (35)
Hashimoto thyroiditis	3 (18.75)	1(25)	4 (20)
osteoporosis/osteoporotic fractures	3 (18.75)	1 (25)	4 (20)
recurrent infections	5 (31.25)	2 (50)	7 (35)
type 2 diabetes / glucose intolerance	2 (12.5)	1 (25)	3 (15)
dyslipidemia	6 (37.5)	3 (75)	9 (45)
metabolic syndrome	3 (18.75)	2 (50)	5 (25)
struma nodosa	3 (18.75)	1 (25)	4 (20)
hypothyroidism	1 (6.25)	–	1 (5)
cholelithiasis	5 (31.25)	–	5 (25)
gastric ulcers	1 (6.25)	–	1 (5)
arterial hypertension	11 (68.75)	4 (100)	15 (75)
myocardial infarction	1 (6.25)	3 (75)	4 (20)
paroxysmal atrial fibrillation	1 (6.25)	1 (25)	2 (10)
heart valve defect	1 (6.25)	–	1 (5)
abdominal aortic aneurysm	–	1 (25)	1 (5)
stroke	1 (6.25)	1 (25)	2 (10)
transient ischemic attack	1 (6.25)	–	1 (5)
chronic kidney disease	3 (18.75)	2 (50)	5 (25)
chronic obstructive pulmonary disease	2 (12.5)	–	2 (10)
interstitial lung disease	6 (37.5)	–	6 (30)
glaucoma/cataract	3 (18.75)	–	3 (15)
osteoarthritis	9 (56.25)	2 (50)	11 (55)

Data are presented as number (percentage) of patients.

TABLE 5 Treatment of patients with late-onset systemic lupus erythematosus

Treatment	Total, n (%)
steroids <15 mg/d	20 (100)
steroid IV pulses	6 (30)
chloroquine/hydroxychloroquine	20 (100)
methotrexate	9 (45)
azathioprine	4 (20)
mofetil mycophenolate	2 (10)
cyclosporine	1 (5)
cyclophosphamide IV	5 (25)

comorbidities were arterial hypertension (75% of the patients; 100% of men, 68.75% of women), dyslipidemia (45% of the patients; 75% of men, 37.5% of women) and osteoarthritis (55% of the patients; 50% of men, 56.25% of women). Recurrent infections were observed in 35% of the patients (50% of men, 31.25% of women), the most common being urinary and respiratory tract infections. One male patient had a tumor in the neck region, which was diagnosed as an abscessed lateral neck cyst.

Thrombotic complications were observed in 38.8% of the patients. Pulmonary embolism and stroke were both diagnosed in 1 man and 1 woman, while myocardial infarction was diagnosed in 3 men (75%) and 1 woman. Carotid artery thrombosis was confirmed in 1 woman and deep vein thrombosis in the lower limbs was diagnosed in 2 men and 1 woman. The diagnosis of APS was established in 25% of the patients (3 men [75%], 2 women [12.5%]). In 1 man, catastrophic APS was diagnosed. Sjögren syndrome was diagnosed in 35% of the late-onset group (1 man [25%] and 6 women [37.5%]).

Tumors were observed in 38.9% of the patients; however, they were predominantly benign (angiomyolipoma in 1 woman, liver angioma in 1 woman, uterine myoma in 1 woman, and prostate adenoma in 1 man). Skin cancer (carcinoma basocellulare) was diagnosed in 1 woman at the age of 68 years, before the first manifestations of SLE were observed. Vitamin D deficiency was observed in 72.2% of the patients (71.4% of women and 100% of men) at diagnosis of SLE.

Regarding the frequency of concomitant autoimmune diseases, no significant differences between the late-onset and early-onset groups in the prevalence of Hashimoto thyroiditis (20% vs 7.4%, $P = 0.076$) and APS (25% vs 34.25%, $P = 0.418$) were observed. Sjögren syndrome was more frequent in the late-onset group compared with the early-onset group (35% vs 11.1%, $P = 0.006$).

The treatment of patients with late-onset SLE is presented in TABLE 5. All patients were treated with chloroquine or hydroxychloroquine and low-dose steroids. Methotrexate was used in 45%, intravenous steroid pulses in 30%, and IV cyclophosphamide in 25% of the patients.

DISCUSSION According to epidemiological data, SLE is most commonly diagnosed in young adults; however, it may also develop in children and elderly patients.[1-7] The prevalence of late-onset SLE, defined as the onset of disease at the minimum age of 50 years, is estimated at 10% to 20% of SLE patients, according to different published studies.[5,7,9-11] In our study, late-onset SLE was confirmed in 20 of 230 patients (8.7%). The lower incidence of SLE in our study might be explained by epidemiological and ethnic differences, even though according to the literature, the onset of SLE is more common in elderly Caucasian patients.[1,5,7] The course of SLE in Caucasian patients is regarded as milder.[1]

We established the diagnosis of late-onset SLE at the time of symptom manifestation only in 40% of our patients. In the remaining patients (60%), different autoimmune diseases were diagnosed before the diagnosis of SLE, including rheumatoid arthritis in 20%, Sjögren syndrome in 20%, mixed connective tissue disease in 5%, undifferentiated arthritis in 5%, systemic sclerosis in 5%, autoimmune thrombocytopenia in 5%, and APS in 5% of the patients. A delay in the diagnosis of SLE was between 0 and 144 months (mean, 31.7 months). This illustrates difficulties in the diagnosis of late-onset SLE. According to

the available data, the course of SLE in older people is generally considered to be milder, insidious, and misleading, resulting in diagnostic challenge because of nonspecific manifestations and multiple comorbidities.[5,7,10] This could also explain the low percentage of patients with late-onset SLE in our study group. At the same time, due to physiological process of aging, organ damage might be greater.

The age at onset of SLE might affect the clinical manifestations of the disease but the available data are inconclusive. In late-onset SLE, photosensitivity, skin manifestations, and nephritis are less common[6,9-11]; in general, major organ involvement is less frequent.[5] A number of authors claimed that the prevalence of arthritis, serositis, fever, Sjögren syndrome, Raynaud phenomenon, lung disease, and neuropsychiatric manifestations in elderly patients with SLE is more frequent.[11,12] Older patients frequently show positivity for rheumatoid factor, anti-Ro, and anti-La antibodies, while hypocomplementemia and the presence of anti-ds-DNA antibodies are uncommon in this group.[6,10,11] Cytopenias are more common in late-onset SLE.[13] The course of SLE in elderly people is considered to be milder and have a better prognosis.[7,11,12] Nevertheless, owing to a higher incidence of comorbidities and greater organ damage in older people, the mortality rate tends to be higher.[5,11,13] Although the multiple organ and system damage might be provoked by SLE itself, it could be also triggered by treatment.[14] Differences in the prevalence of symptoms and the course of late-onset SLE in the literature might be caused by a small number of patients in previous studies.[5,13]

The most common clinical manifestations of SLE in our late-onset group were arthritis (50%), nephropathy (40%), skin manifestations (40%), photosensitivity (30%), and interstitial lung disease (30%). In laboratory findings, 65% of the patients had leukopenia and 35% had thrombocytopenia. All patients were positive for antinuclear antibodies, while 65% were positive for anti-ds-DNA antibodies. Anti-SS-A were present in 60% of the patients; anti-SS-B, in 30%; and RF-IgM, in 35% of the patients. Hypocomplementemia was observed in 45% of the patients. Anti-P antibodies, considered to be characteristic for early-onset SLE,[15] were not present in the study group.

Our comparison of patients with late-onset and early-onset SLE yielded results that were largely in accordance with the cited literature data. The clinical manifestations such as skin involvement, photosensitivity, and nephropathy were less common in elderly patients. However, no differences were found in the incidence of arthritis, serositis, general symptoms, Raynaud phenomenon, and cytopenias between the late-onset and early-onset groups. Central nervous system involvement was significantly less common in the late-onset group. As for immunological findings, hypocomplementemia was significantly less frequent in the late-onset group, which is in line with the literature data. Anti-Sm and anti-RNP antibodies were also less frequent in the late-onset group. Anti-β_2-GPI antibodies were significantly more frequent in the early-onset group, while no significant difference was noted in the presence of anticardiolipin antibodies, lupus anticoagulant, and aPL between the study groups. In addition, we did not observe significant differences between the groups in terms of RF-IgM and ACPA positivity.

Women with late-onset SLE had a milder course of the disease. All 4 male patients had a severe course of the disease, with acute onset and the presence of aPL. The presence of aPL, possible in SLE without APS, increases the risk of thrombosis.[16] In 75% of men in our group, a diagnosis of APS was established.

The majority of our patients had concomitant diseases including autoimmune diseases such as Sjögren syndrome (35% of the patients), Hashimoto thyroiditis (20%), and antiphospholipid syndrome (25%). Cardiovascular diseases and complications were the most common in our patients. Vitamin D deficiency was confirmed in most patients (72.2%).

In conclusion, the clinical picture of late-onset SLE differs from that in early-onset SLE. Arthritis, leukopenia, and thrombotic complications are common. Skin manifestations, photosensitivity, nephropathy, vasculitis, and central nervous system involvement are less common in late-onset SLE. The diagnosis of late-onset SLE is often delayed, and the type of treatment is determined by common concomitant diseases.

Contribution statement MM conceived the idea for the study. RJ and DS contributed to the design of the research and collected the data. All authors were involved in data analysis and edited and approved the final version of the manuscript. RJ and DS prepared the manuscript for submission.

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ARTYKUŁ ORYGINALNY

Obraz kliniczny tocznia rumieniowatego układowego o późnym początku w grupie polskich pacjentów

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SŁOWA KLUCZOWE

objawy kliniczne, późny początek, starszy wiek, toczeń rumieniowaty układowy

STRESZCZENIE

WPROWADZENIE Częstość występowania tocznia rumieniowatego układowego (TRU) o późnym początku, rozpoznawanego u osób po 50. rż., ocenia się na 10–20%. U osób w wieku starszym TRU ma specyficzne cechy, z mylącymi objawami, ale jego przebieg kliniczny wydaje się łagodniejszy niż u młodszych pacjentów.

CELE Celem pracy była ocena klinicznego przebiegu TRU o późnym początku w grupie chorych leczonych w Polsce.

PACJENCI I METODY Z grupy 230 kolejnych chorych na TRU wybrano osoby, u których choroba miała późny początek. Retrospektywnie analizowano częstość występowania cech klinicznych TRU i chorób współistniejących oraz leczenie. Częstość występowania cech klinicznych TRU u pacjentów z TRU o późnym początku porównano z grupą 108 chorych z TRU o wczesnym początku.

WYNIKI TRU o późnym początku stwierdzono u 20 chorych (8,7%), w tym 16 kobiet (80%) i 4 mężczyzn (20%). Opóźnienie rozpoznania wyniosło 31,7 miesiąca (0–144). Najczęstszymi objawami TRU były: zapalenie stawów (50%), wysypka (40%), nefropatia (40%), nadwrażliwość na promienie słoneczne (30%), owrzodzenia w jamie ustnej (30%), choroba śródmiąższowa płuc (30%), gorączka (25%), leukopenia (65%) i trombocytopenia (35%). Zajęcie nerek było obecne u wszystkich mężczyzn i u 25% kobiet. Powikłania zakrzepowe stwierdzono u 38,8% pacjentów. Choroby współistniejące były częste w naszej grupie badanej.

WNIOSKI Obraz kliniczny TRU o późnym początku różni się od TRU o wczesnym początku. Zapalenie stawów, leukopenia i powikłania zakrzepowe są częste, podczas gdy objawy skórne, nadwrażliwość na światło, zapalenie naczyń, zajęcie nerek i ośrodkowego układu nerwowego są rzadsze u chorych z TRU o późnym początku. Rozpoznanie TRU o późnym początku jest nierzadko opóźnione, a leczenie – zdeterminowane występującymi chorobami współistniejącymi.

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