ORIGINAL INVESTIGATIONS

Hospital mortality in a cohort of Kyrgyz patients with systemic lupus erythematosus

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High disease activity, major organ involvement, severe exacerbations, and infections are considered the most common causes of hospitalization in patients with systemic lupus erythematosus (SLE). Hospital mortality (HM) is higher among Asian, African, and Latin American patients compared with Caucasians. In Asian cohorts, the most frequent causes of HM are infections, severe lupus nephritis (LN), and neuropsychiatric manifestations.

Objective. *To analyze the structure of mortality and prognostic factors associated with increased risk of death in hospitalized SLE patients.*

Material and methods. *The study included 800 patients with a confirmed diagnosis of SLE treated at the National Center of Cardiology and Therapy named after academician Mirsaid Mirrahimov from January 2012 to December 2024. An electronic database of fatal cases was used to assess the structure and causes of HM. For comparative analysis of clinical and laboratory manifestations of SLE and to identify predictors of HM, patients were divided into survivor and non-survivor groups. All patients underwent standard clinical, laboratory, and instrumental examinations.*

Results and discussion. *In the Kyrgyz SLE population, HM reached 3.3%. The vast majority of deceased patients were young women (88.5%) with acute disease course (53.9%), high activity (73.1%), kidney involvement (76.9%), central nervous system (CNS) involvement (50%), and lung involvement (34.6%). The main causes of HM were severe LN (30.8%) and combined kidney and CNS involvement (15.4%). Independent predictors of HM were LN with advanced chronic kidney disease (CKD), irreversible organ damage, and C3 hypocomplementemia.*

Conclusion. *The main causes of HM were severe LN (30.8%) and combined kidney and CNS involvement (15.4%). Independent predictors of HM included LN with advanced CKD, irreversible organ damage, and C3 hypocomplementemia.*

Keywords: *systemic lupus erythematosus; activity; Asians; hospital mortality; infection; lupus nephritis; predictors.*

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Systemic lupus erythematosus is a systemic autoimmune rheumatic disease of unknown etiology, characterized by hyperproduction of organ-nonspecific autoantibodies to various components of the cell nucleus and the development of immune-inflammatory damage to internal organs [1]. Advances in the treatment of patients with SLE over the past decade have led to improved survival rates, a high frequency of sustained remission, and a reduction in hospitalizations, irreversible organ damage (IOD), and mortality [2–4]. However, despite improved prognosis and survival, mortality in SLE is 1.5–3 times higher than in the general population [5] and exceeds that in patients with other immune-inflammatory rheumatic diseases (IIRD), such as systemic scleroderma, Sjцgren's syndrome, idiopathic inflammatory myopathies, and systemic vasculitis associated with antineutrophil cytoplasmic antibodies [6].

In recent years, the proportion of multisyndromic involvement in the structure of mortality in SLE has significantly decreased, while the role of infectious, oncological, and cardiovascular diseases has increased [7–12]. The most common causes of hospitalization in patients with SLE are considered to be high activity, frequent exacerbations and severe infections [13–18]. The majority of population-based and cohort studies indicate a severe course of SLE and high mortality among hospitalized Asian, African American, and Latino patients compared with patients of Caucasian ethnicity [19–22].

The aim of the study was to investigate the mortality structure and prognostic factors associated with an increased risk of fatal outcome in hospitalized patients with SLE.

Materials and methods. The prospective cohort study included 800 patients with a confirmed diagnosis of SLE who met the classification criteria of SLICC (Systemic Lupus International Collaborating Clinics, 2012) [23] and were treated at the clinic of NCCT named after Academician Mirsayid Mirrakhimov at of the Ministry of Health of the Kyrgyz Republic, from January 2012 to December 2024. Patients were included at different stages of the study as they were initially hospitalized to the NCCT. All patients signed an informed consent form. The research design and examination methods used were approved by the local ethics committee at the NCCT in 2012. Inclusion criteria: confirmed diagnosis of SLE; signed informed consent to participate in the study; age ≥18 years. *Non-inclusion criteria:* age <18 years.

The patients were divided into two groups (survivors and non-survivors) for comparative analysis of clinical and laboratory manifestations and determination of predictors of HM. Pathological autopsies were not performed because relatives declined permission, primarily for religious reasons. A detailed analysis of the structure

and causes of HM was performed based on the electronic database of fatal cases and postmortem epicrises from official medical documents. HM was calculated as the ratio of the number of deceased patients with SLE to all patients hospitalized with this disease within 1 year [24].

All patients underwent standard clinical, laboratory, and instrumental examinations. The onset of the disease was classified according to the classification by V.A. Nasonova (1972) [25] as acute, subacute, or chronic. SLE activity was determined using SLEDAI-2K (Systemic Lupus Erythematosus Disease Activity Index,

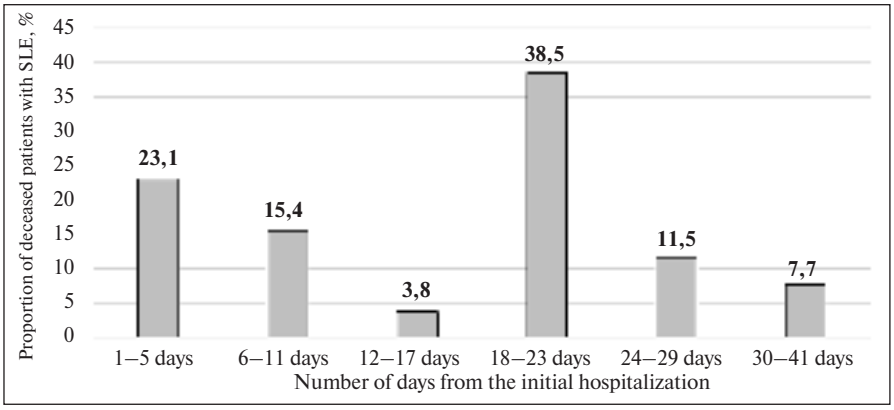

Fig. 1. *Distribution of deceased patients by timing of HM in 2012–2024 (n=26)*

Table 1. Comparative characteristics of survivors and non-survivors with SLE

Parameters	surviving patients (n=774)	deceased patients (n=26)	p
Female/male, n (%)	712 (92)/62 (8)	23 (88,5)/3 (11,5)	0,51
Ethnicity, n (%):			
Kyrgyz/ Asians/ Slavic people	685 (88,5)/58 (7,5)/31 (4)	22 (84,6)/3 (11,5)/1 (3,9)	0,87
Age of patients during hospitalization, years, M±SD:	35,4±12,1	33,7±14,4	0,26
female	35,5±12,2	35,2±14,5	0,91
male	34,0±10,4	22,0±4,3	<0,001
Duration of SLE at the time of hospitalization, months, Me [25th; 75th percentiles]	25 [7; 72]	24,5 [5; 96]	0,84
Duration of SLE at the time of diagnosis, months, Me [25th; 75th percentiles]	12 [4; 48]	8,5 [3; 96]	0,82
SLE diagnosis, n (%):			
early	255 (33)	10 (38,4)	0,68
late	519 (67)	16 (61,6)	0,79
Course of SLE, n (%):			
acute	253 (32,7)	14 (53,9)	0,02
subacute	278 (35,9)	3 (11,5)	0,01
chronic	243 (31,4)	9 (34,6)	0,67
SLEDAI-2K, n (%):			
remission	5 (0,7)	0	0,68
low	55 (7,1)	0	0,17
moderate	227 (29,3)	2 (7,7)	0,05
high	285 (36,8)	5 (19,2)	0,18
very high	202 (26,1)	19 (73,1)	<0,001
SLICC:			
n (%)	135 (17,4)	20 (76,9)	<0,001
M±SD	0,27±0,68	1,77±1,31	

modified in 2000): 0 – no activity, 1–5 – low, 6–10 – moderate, 11–19 – high, and >20 points – very high activity [26]. IOD was assessed using the SLICC/ACR (American College of Rheumatology) damage index [27]: no damage – 0 points, low damage index – 1-point, moderate damage index – 2–4 points, high damage index – >4 points. Patients were stratified into groups of early (<6 months) and and late (≥6 months) SLE diagnosis after the onset of the disease [28, 29].

The diagnosis of neuropsychiatric manifestations of SLE (NPSLE) was made in accordance with the modified ACR 2001 classification criteria [30]. LN was diagnosed according to the ACR (2004) criteria [31]. Clinical and laboratory manifestations of LN were assessed using the clinical classification of I.E. Tareeva (1976) [32]. To identify acute kidney injury (AKI), the KDIGO (kidney disease: Improving Global Outcomes) criteria were used [33]. To determine the degree of reduction in glomerular filtration rate (GFR) and severity of proteinuria, the KDIGO (2012) classification of chronic kidney disease (CKD) was used [34].

Statistical data processing was performed using Statistica 10.0 (Stat Soft Inc., USA) and SPSS, version 23 (IBM, USA). Quantitative variables with normal distribution are presented as M±SD, variables with distribution other than normal are presented as median with interquartile range (Me [25th; 75th percentiles]). In the comparative analysis of normally distributed data, an

unpaired t-test was used. In the absence of normal distribution of quantitative parameters, the Mann–Whitney criterion was used. Qualitative variables were compared using the χ^2 test, Fisher's and Pearson's two-tailed criteria with Yates's correction. Independent predictors of HM were determined using logistic regression analysis with calculation of the odds ratio (OR) and 95% confidence interval (CI) and presentation of data on a forest plot graph. Differences were considered significant at $p<0.05$.

Results. The number of patients hospitalized with SLE was significantly higher than with other IIRD, ranging from 13.8% to 21% per year. During the analyzed period, 26 (3.3%) patients died during hospitalization. The median duration of SLE before death was 20.5 [5.0; 96.0] months, and the average length of hospitalization before death was 13.31±8.93 days. The analysis of the HM by the length of stay of the deceased patients in the hospital is presented in Fig. 1. Thus, 6 (23.1%) patients died in the first 5 days of hospitalization, and 4 (15.4%) patients died on the 6th–11th day. The highest number of fatalities occurred on days 8–23 (38.5%).

The main initial clinical characteristics of the patients are presented in Table 1. In both groups, young women predominated. The mortality rate among women (88.5%) was higher than among men (11.5%; p=0.0009). The deceased men were significantly younger than the surviving male patients (mean age – 22.0±4.3 and 34.0±10.4 years, respectively; p<0.001). The highest HM was observed in the 18–23 age group (30.8%).

Table 2. Comparative characteristics of clinical and laboratory manifestations of SLE in survivors and non-survivors, n (%)

Indicator	Survivors (n=774)	Non-survivors (n=26)	p
Clinical symptoms			
Constitutional manifestations	437 (56,5)	18 (69,2)	0,19
Alopecia	544 (70,3)	21 (80,8)	0,25
Cutaneous-mucosal syndrome	724 (93,5)	24 (92,3)	0,96
Vasculitis	194 (25,1)	7 (26,9)	0,87
Arthritis	354 (45,7)	7 (26,9)	0,06
Serositis (pericarditis/pleuritis)	347 (44,8)	22 (84,6)	<0,001
Heart involvement	25 (3,2)	2 (7,7)	0,21
GIT involvement	37 (4,8)	4 (15,4)	0,02
Neurological involvement (n=183)			
NPSLE	170 (22)	13 (50)	0,01
The central nervous system	86 (11,1)	13 (50)	<0,001
including:			
aseptic meningitis	1 (0,1)	0 (0)	0,85
CVD	8 (1,0)	3 (11,5)	<0,001
myelopathy	11 (1,4)	0 (0)	0,54
seizure disorder	16 (2,1)	5 (19,2)	<0,001
acute delirium	4 (0,5)	0 (0)	0,71
cognitive dysfunction	1 (0,1)	0 (0)	0,85
psychosis	45 (5,8)	5 (19,2)	0,01
PNS	84 (10,8)	0 (0)	0,09
Kidney involvement (n=339)			
LN	319 (41,2)	20 (76,9)	0,03
Proteinuria >500 mg/day	212 (27,4)	19 (73,1)	<0,001
Nephrotic syndrome	55 (7,1)	2 (7,7)	0,91
ESRD	34 (4,4)	15 (57,7)	<0,001
AKI	11 (1,4)	13 (50)	<0,001
Severe CKD (4–5th stage)	44 (5,6)	15 (57,7)	<0,001
Respiratory system involvement (n=78)			
Lung involvement	69 (8,9)	9 (34,6)	0,002
Pneumonitis	34 (4,4)	4 (15,4)	0,03
Interstitial lung disease	17 (2,2)	0 (0)	0,94
PAH	14 (1,8)	1 (3,9)	0,39
pulmonary embolism	3 (0,4)	3 (11,5)	<0,001
acute hemorrhagic alveolitis	0 (0)	1 (3,9)	0,03
ULN	1 (0,13)	0 (0)	0,99
Hematological disorders (n=276)			
Hematological disorders,	259 (93,8)	17 (6,2)	0,03
including:			
isolated	183 (70,7)	10 (58,8)	0,19
combined	76 (29,3)	7 (41,2)	0,01
Immunological abnormalities (n=800)			
Anti-dsDNA antibodies	534 (69)	17 (65,4)	0,68
Low C3 levels	449 (58)	22 (84,6)	0,69

Note: AKI – acute kidney injury, anti-dsDNA – antibodies to double-stranded DNA, GIT – the gastrointestinal tract, ESRD – end-stage renal of the diseases, CVD – the cerebrovascular disease, CKD – chronic kidney disease, PNS – peripheral nervous system, PAH – pulmonary arterial hypertension, NPSLE – the neuropsychiatric manifestations of the systemic lupus erythematosus, RPGN – rapidly progressive glomerulonephritis, ULN – ulcerative necrotizing laryngotracheitis.

Deceased patients were more likely than survivors to have an acute variant of SLE (in 53.9% and 32.7% of cases, respectively). p=0,02). At the time of initial hospitalization, remission and low activity according to SLEDAI-2K were recorded only in the survivor group (in 0.7% vs 7.1% of cases). Moderate and high activity in survivors was observed slightly more often than in deceased patients (in 29.3% and 7.7%; 36.8% and 19.2% of cases, respectively), but these differences were not statistically significant. At the same time, very high disease activity in the deceased group was significantly more common than in the survivor group (in 73.1% and 26.1% of cases, respectively; p<0.001).

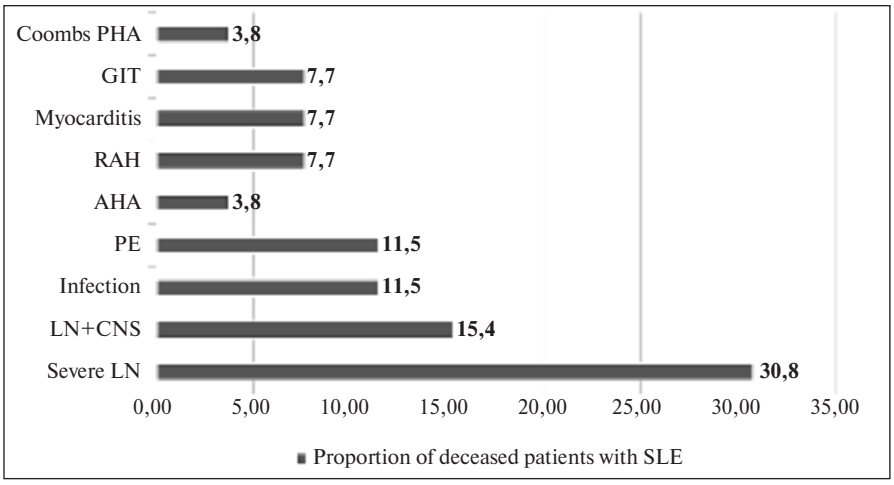

Fig. 2. *Structure of HM in SLE patients (n=26), %*

Statistically significant differences (p<0.05) were found in the frequency of LN, NPSLE, and damage to the respiratory and gastrointestinal tract (GIT), which were observed predominantly in the group of deceased patients (Table 2). The frequency of NPSLE associated with severe damage to the central CNS was significantly higher in the group of deceased patients than in the group of survivors (50% and 11.1%, respectively; p<0.001). These disorders manifested as psychosis (19.2%) and convulsive syndrome (19.2%), and somewhat less frequently as cerebrovascular disease (CVD, 11.5%). Peripheral nervous system (PNS) damage was mainly found in surviving patients (10.8%).

LN was diagnosed in 319 (41.2%) survivors and 20 (76.9%) deceased patients (p=0.037). Active LN without signs of the end-stage renal disease (ESRD) was detected mainly in surviving patients (97.2%). In the group of deceased patients, rapidly progressive glomerulonephritis (RPGN), severe stages (4–5) of CKD, low GFR, and AKI were observed significantly more often than in the group of survivors (p<0.001). In 13 (65%) of 20 deceased patients with LN, AKI developed at severe stages of CKD (stage 3b in 3 patients and stage 4 in 10 patients), and 5 (25%) patients had terminal stage (5) of CKD.

Severe pulmonary pathology was found predominantly in the group of deceased patients (34.6%; p=0.002). They had significantly more cases of lupus pneumonitis and PE than survivors: 15.4% and 4.4% of cases (p=0.031) and 11.5% and 0.4% (p<0.001), respectively. PAH was rarely detected in both groups (in 1.8% of survivors and 3.9% of deceased patients; p>0.05).

The group of surviving patients had hematological disorders more often than the group of deceased patients, but combined forms were less common (in 93.8% and 6.2% of cases, respectively; p<0.05; 29.3% and 41.2% of cases, respectively; p<0.05).

Immunological activity in surviving and deceased patients did not differ significantly (p>0.05), and the frequency of detection of anti-dsDNA and C3 and C4 hypocomplementemia was comparable (69% and 65.4%; 58% and 84.6%; respectively p>0.05).

The most common cause of HM was severe LN (n=8, 30.8%; Fig. 2). In 5 (62.5%) patients, death occurred due to AKI in the setting of stage 4 of CKD, while the remaining 3 (37.5%) died as a result of end-stage renal disease (ESRD). The second most common cause of HM was severe combined damage to the kidneys and CNS (n=4, 15.4%). In 3 (75%) of these patients with long-term LN, cerebral coma developed due to acute cerebral circulation disorders (ACCD) of ischemic and hemorrhagic types, and in 1 patient cerebral edema progressing to cerebral coma developed after a series of generalized epileptic seizures. The third leading causes of HM were infections (11.5%) and PE (11.5%). One patient had disseminated tuberculosis of the lungs with meningoencephalitis, and 2 more patients - septic pneumonia. PE was the cause of death in 2 patients with severe CKD and in 1 patient with combined damage to the kidneys (RPGN) and lungs (pneu-

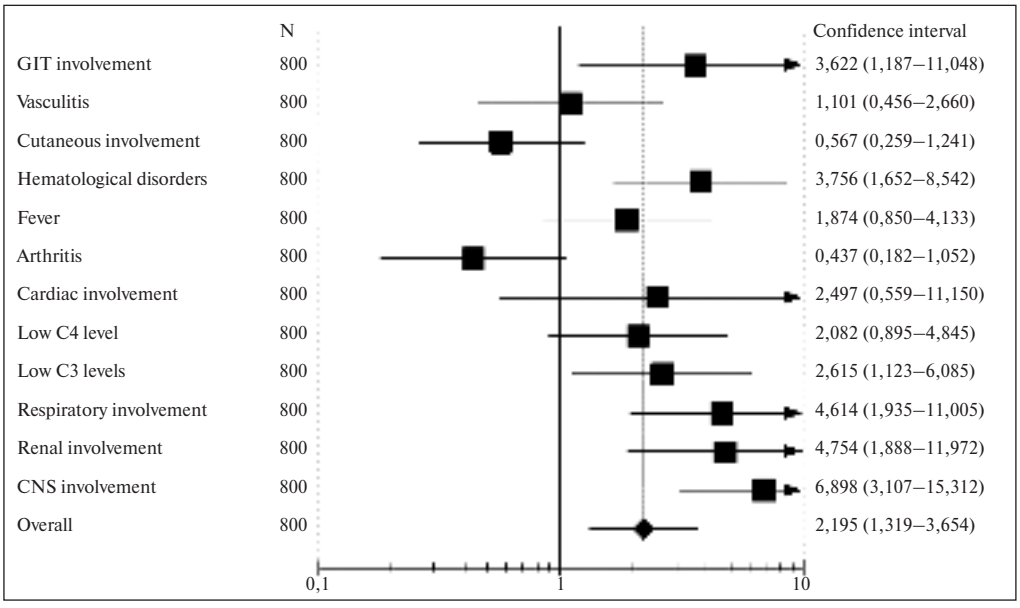

Fig. 3. *Clinical and laboratory indicators of SLE activity as risk factors for HM*

Table 3. Independent predictors of HM in SLE patients (n=26)

Parameters	OR	95% CI	p
Low GFR	0,98	0,97–0,99	0,01
Low C3 levels	4,12	1,41–12,1	0,01
SLICC damage index	2,67	1,51–4,7	<0,001
Sensitivity – 50% Specificity – 99.5%			

monitis). One patient with CKD had AHA with pulmonary hemorrhage. Two patients died as a result of PAH with decompensated chronic heart failure (CHF), and two more patients - as a result of diffuse myocarditis with signs of severe heart failure and acute pulmonary edema. In two cases, HM was caused by gastrointestinal pathology: one patient developed acute pancreonecrosis with multiple organ failure syndrome (MOFS), and the other had acute hepatic coma caused by decompensated biliary cirrhosis of the liver.

One more patient with severe Coombs-positive hemolytic anemia (Coombs-PHA) died of MOFS with acute pulmonary edema. To determine the likely predictors of HM, a one-dimensional logistic regression analysis was used with calculation of OR and 95% CI and presentation of data in the form of a forest plot graph (Fig. 3).

The highest risk of HM in our patients was observed in the presence of severe CNS lesions (OR 6.898; 95% CI 2.78–11.78; p=0.0001), kidney damage (OR 4.754; 95% CI 1.888–11.972; p<0.001), and respiratory organ damage (OR 4.614; 95% CI 1.935–11.005; p<0.001). A less significant increase in the risk of HM was associated with gastrointestinal pathology (OR 3.662; 95% CI 1.187 – 11.048; p=0.024), hematological disorders (OR 3.756; 95% CI 1.652 – 8.542; p=0.002) and C3 hypocomplementemia (OR 2.615; 95% CI 1.123–6.085; p=0.026).

Logistic regression analysis showed that statistically significant independent predictors of the risk of developing HM in the Kyrgyz cohort of patients with SLE (p <0.05) are low GFR, the presence of IOD, and C3 hypocomplementemia (Table 3).

Discussion. In different cohorts, the frequency of hospitalization for SLE varies from 8.6% to 50% and depends on a number of factors, including the severity of the patient's condition, the presence of complications, ethnicity, social status, and access to highly specialized medical care [35–37]. There are few studies devoted to the causes and structure of HM in SLE, which can be explained by insufficient information from tertiary care facilities or a small sample of hospitalized patients [9, 10]. According to the results of early population studies conducted in the 1970s and 1980s, the incidence of HM in Asians was 3–5 times higher than in white patients [38–42]. More recent cohort studies covering two observation periods (from 1998 to 2002 and from 2003 to 2011) provide more detailed data on the causes and structure of HM in patients with SLE living in the United States. Thus, the frequency of HM in SLE in the late 1990s and early 2000s did not exceed 3.1% [11]. More than 50% of deaths occurred in the first 7 days of hospitalization, with a second peak on day 33 and a third on day 57. According to L.B. Goss et al. [12], Asians and African Americans were hospitalized almost three times more often than Caucasian patients. The mortality rate among men was higher than among women (2.6% and 1.8%, respectively).

The average age of those who died was 51.5 years (95% CI 50.6– 52.3) in 2003 and 51.3 years (95% CI 50.6–52.0) in 2011. According to data from other studies, from the mid-1990s until 2017, the causes of frequent hospitalization for SLE in Western Europe, the US, and Canada were infections and severe exacerbation of SLE.

Data on SLE in Asian cohorts differ somewhat from those for Caucasians. For example, in a Malaysian cohort, the ratio of women to men with SLE was 10:1, the average age of patients was 30.5±12.2 years, and the average duration of the disease was 36.5±51.6 months [43]. Among clinical manifestations in this population, hematological disorders (73.3%), LN (70.9%), and specific skin lesions (67.3%) predominated. The frequency of HM in Malaysian patients with SLE was high (10.4%) due to severe exacerbations (19%) and fatal infections (19%). Prognostically unfavorable factors increasing the risk of HM in Malaysian patients included frequent SLE exacerbations (OR 5.56) and high IOD rate (OR 1.91). In the Jordanian cohort, the frequency of SLE-related hospitalizations from 2002 to 2017 reached 28.6%, and HM – 14.1% [44]. The average age of onset was 34±12.5 years, with a female-to-male ratio of 8.4:1. The high frequency of HM in this cohort was due to serious infectious complications and progression of IOD (42.5% and 40%, respectively).

In our study, the incidence of SLE in the general population was 11 times higher in women than in men (91.9% and 8.1%, respectively). In the Kyrgyz cohort, statistically significant differences were found in the frequency of LN, NPSLE, respiratory and gastrointestinal involvement, observed mainly in the group of deceased patients, with the exception of polyserositis, which was diagnosed predominantly in survivors. Over the 12-year period analyzed since the time of initial hospitalization, the overall HM rate was 3.3%. The average age of deceased patients was 33.7± 14.4 years. The highest mortality rate (30.8%) was observed in the age group of 18-23 years. The HM in women was higher than in men (88.5% and 11.5%, respectively; p=0.0009). In our cohort, the highest number of cases of HM (38.5%) occurred on the 8th–23rd days of hospitalization. In the group of deceased patients, acute SLE, high activity according to SLEDAI-2K and IOD were more common than in the group of survivors.

In a Chinese cohort, the mean age of deceased SLE patients was practically comparable to the indicators we have obtained (37.8±14.7 years) [45]. The median duration of the disease was 2.6 [0.5; 7.0] years, and the duration of observation was 3.0 [1.4; 5.1] years. The mortality rate was quite high – 4.9%. In Chinese patients, infections were the main cause of mortality, accounting for one-third of cases (31.1%), followed by ESRD as a result of severe LN, then isolated PAH and ACCD. Independent risk factors for mortality in Chinese patients were older age at disease onset, infections, Coombs-PHA, thrombocytopenia, and PAH. In our cohort, all patients who died at the time of hospitalization were in extremely serious condition. HM in most cases was caused by severe HF (30.8%). Thus, 62.5% of patients with RPGN, had AKI with a clinical picture of acute uremic pulmonary edema. In 37.5% of patients with LN, death was due to decompensated CHF as a result of ESRD. The second most common direct cause of HM was severe combined kidney and CNS damage (15.4%). In 75% of patients with LN from this group, cerebral coma developed as a result of ischemic and hemorrhagic types of ACCD. The predictor of HM in our patients with SLE was damage to vital organs (CNS, kidneys, lungs, and gastrointestinal tract), and

independent predictors of HM were severe LN with pronounced CRF, the presence of IOD, and decreased C3 levels.

Thus, the results of this study demonstrate that Asian patients with SLE are potentially at increased risk of adverse outcomes and, therefore, require careful monitoring using modern methods of therapy both in hospital and in outpatient settings.

Conclusion. The hospital mortality in the Kyrgyz cohort of SLE patients reached 3.3%. The main causes were severe form of LN (30.8%), combined kidney and central nervous system damage (15.4%). Independent predictors of hospital mortality were Lupus nephritis with severe ESRD, presence of irreversible organ damage and C3 hypocomplementemia.

REFERENCES

1. Насонов ЕЛ, Соловьев СК, Аршинов АВ. Системная красная волчанка: история и современность. Научно-практическая ревматология. 2022;60(4):397-412.
Nasonov EL, Solov'ev SK, Arshinov AV. Systemic lupus erythematosus: history and modernity. *Nauchno-prakticheskaya revmatologiya*. 2022;60(4):397-412. (In Russ.).
2. Alarcyn GS, McGwin G Jr, Bastian HM, et al. Systemic lupus erythematosus in three ethnic groups. VIII. Predictors of early mortality in the LUMINA cohort. *Arthritis Rheum*. 2001 Apr;45(2):191-202. doi: 10.1002/1529-0131(200104)45:2<191: AID-ANR173>3.0.CO;2-2.
3. Yazdany J, Marafino BJ, Dean ML, et al. Thirty-day Hospital Readmissions in Systemic Lupus Erythematosus: Predictors and Hospital and State-level Variation. *Arthritis Rheumatol*. 2014 Oct;66(10):2828-36. doi: 10.1002/art.38768.
4. Tektonidou MG, Lewandowski LB, Hu J, et al. Survival in adults and children with systemic lupus erythematosus: a systematic review and Bayesian meta-analysis of studies from 1950 to 2016. *Ann Rheum Dis*. 2017 Dec; 76(12):2009-2016. doi: 10.1136/annrheumdis-2017-211663.
5. Barber M, Drenkard C, Falasinnu T, et al. Global epidemiology of systemic lupus erythematosus. *Nat Rev Rheumatol*. 2021 Sep; 17(9):515-532. doi: 10.1038/s41584-021-00668-1.
6. Scherlinger M, Mertz P, Sagez F, et al. Worldwide trends in all-cause mortality of auto-immune systemic diseases between 2001 and 2014. *Autoimmun Rev*. 2020 Jun;19(6): 102531. doi: 10.1016/j.autrev.2020.102531.
7. Lee J, Dhillon N, Pope J. All-cause hospitalizations in systemic lupus erythematosus from a large Canadian referral Centre. *Rheumatology (Oxford)*. 2013 May;52(5): 905-9. doi: 10.1093/rheumatology/kes391.
8. Chan K, Dekis A, Clarke AE, et al. Hospitalizations in patients with systemic lupus erythematosus: updated analyses from 2006 to 2011. *Arthritis Res Ther*. 2012;14 Suppl 3:A59.
9. Pires da Rosa G, Fontecha Ortega M, Teixeira A, et al. Causes and factors related to hospitalizations in patients with systemic lupus erythematosus: analysis of a 20-year period (1995–2015) from a single referral Centre in Catalonia. *Lupus*. 2019 Aug;28(9):1158-1166. doi: 10.1177/0961203319861685.
10. Gu K, Gladman DD, Su J, Urowitz MB. Hospitalizations in patients with systemic lupus erythematosus in an academic health science center. *J Rheumatol*. 2017 Aug;44(8): 1173-1178. doi: 10.3899/jrheum.170072.
11. Krishnan E. Hospitalization and mortality of patients with systemic lupus erythematosus. *J Rheumatol*. 2006 Sep;33(9):1770-4.
12. Goss LB, Ortiz JR, Okamura DM, et al. Significant Reductions in Mortality in Hospitalized Patients with Systemic Lupus Erythematosus in Washington State from 2003 to 2011. *PLoS One*. 2015 Jun 18;10(6): e0128920. doi: 10.1371/journal.pone.0128920.
13. Yen EY, Shaheen M, Woo JMP, et al. 46-Year Trends in Systemic Lupus Erythematosus Mortality in the United States, 1968 to 2013: A Nationwide Population-Based Study. *Ann Intern Med*. 2017 Dec 5;167(11): 777-785. doi: 10.7326/M17-0102.
14. Bernatsky S, Boivin JF, Joseph L, et al. Mortality in Systemic Lupus Erythematosus. *Arthritis Rheum* 2006; 54 (8): 2550–2557. PubMed: 16868977.
15. Kasitanon N, Magder LS, Petri M. Predictors of survival in systemic lupus erythematosus. *Medicine (Baltimore)*. 2006 May; 85(3):147-156. doi: 10.1097/01.md.0000224709. 70133.f7.
16. Lim SS, Helmick CG, Bao G, et al. Racial Disparities in Mortality Associated with Systemic Lupus Erythematosus – Fulton and DeKalb Counties, Georgia, 2002–2016. *MMWR Morb Mortal Wkly Rep*. 2019 May 10; 68(18):419-422. doi: 10.15585/mmwr.mm6818a4.
17. Alarcon GS, Friedman AW, Straaton KV, et al. Systemic lupus erythematosus in three ethnic groups: III. A comparison of characteristics early in the natural history of the LUMINA cohort. Lupus in minority populations: Nature vs. Nurture. *Lupus*. 1999;8(3): 197-209. doi: 10.1191/096120399678847704.
18. Walsh SJ, DeChello LM. Geographical variation in mortality from systemic lupus erythematosus in the United States. *Lupus*. 2001; 10(9):637-46. doi: 10.1191/096120301682430230.
19. Gomez-Puerta JA, Barbhaiya M, Guan H, et al. Racial Ethnic Variation in All-Cause Mortality Among United States Medicaid Recipients with Systemic Lupus Erythematosus: A Hispanic and Asian Paradox. *Arthritis Rheum*. 2015 Mar;67(3):752-60. doi: 10.1002/art.38981.
20. Kaslow RA. High rate of death caused by systemic lupus erythematosus among U.S. residents of Asian descent. *Arthritis Rheum*. 1982 Apr;25(4):414-8. doi: 10.1002/art.1780250409.
21. Serdula MK, Rhoads GG. Frequency of systemic lupus erythematosus in different ethnic groups in Hawaii. *Arthritis Rheum*. 1979 Apr;22(4):328-33. doi: 10.1002/art.1780220403.
22. Ward MM. Hospital Experience and Mortality in Patients with Systemic Lupus Erythematosus. *Arthritis Rheum*. 1999 May;42(5): 891-8. doi: 10.1002/1529-0131(199905)42:5<891: AID-ANR7>3.0.CO;2-B.
23. Petri M, Orbai A, Alarson G, et al. Derivation and validation of the Systemic Lupus International Collaborating Clinics Classification Criteria for Systemic lupus erythematosus. *Arthritis Rheum*. 2012 Aug;64(8):2677-86. doi: 10.1002/art.34473.
24. Драпкина ОМ, Самородская ИВ, Какорина ЕП, Чернявская ТК. Причины госпитальной смертности взрослых по данным медицинских свидетельств о смерти. Профилактическая медицина 2024;27(3):7-13.
Drapkina OM, Samorodskaya IV, Kakorina EP, Chernyavskaya TK. Causes of adult hospital mortality according to medical death certificates. *Profilakticheskaya meditsina* 2024;27(3): 7-13. (In Russ.).
25. Насонова ВА. Системная красная волчанка. Москва: Медицина; 1972.
Nasonova VA. Systemic lupus erythematosus. Moscow: Meditsina; 1972.
26. Gladman DD, Ibanez D, Urowitz MB. Systemic lupus erythematosus disease activity index 2000. *J Rheumatol*. 2002 Feb;29(2):288-91.
27. Gladman DD, Ginzler E, Goldsmith C, et al. The Development and initial validation of the Systemic lupus International Collaborating Clinics/American College of Rheumatology (SLICC/ACR) Damage Index for Systemic lupus erythematosus. *Arthritis Rheum*. 1996 Mar;39(3):363-9. doi: 10.1002/art.1780390303.
28. Oglesby A, Korves C, Laliberte F, et al. Impact of Early Versus Late Systemic Lupus Erythematosus Diagnosis on Clinical and Economic Outcomes. *Appl Health Econ Health Policy*. 2014 Apr;12(2):179-90. doi: 10.1007/s40258-014-0085-x.
29. Faurschou M, Starklint H, Halberg P, Jacobsen S. Prognostic factors in lupus nephritis: diagnostic and therapeutic delay increases the risk of terminal renal failure. *J Rheumatol*. 2006 Aug;33(8):1563-9.
30. Ainiala H, Hietaharju A, Loukkola J, et al.

Validity of the new American College of Rheumatology criteria for neuropsychiatric lupus syndromes: a population-based evaluation. *Arthritis Rheum*. 2001 Oct;45(5):419-23. doi:10.1002/1529-0131(200110)45:5<419.
31. Dooley M, Aranow C, Ginzler E. Review of ACR renal criteria in systemic lupus erythematosus. *Lupus*. 2004;13(11):857-60. doi: 10.1191/0961203304lu2023oa.
32. Тареева ИЕ. Волчаночный нефрит. Москва: Медицина; 1976.
Tareeva IE. Lupus nephritis. Moscow: Meditsina; 1976.
33. Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. *Kidney International Supplements*. 2012;2, 1. doi:10.1038/kisup.2012.1
34. Inker LA, Astor BC, Fox CH, et al. KDOQI US commentary on the 2012 KDIGO clinical practice guideline for the evaluation and management of CKD. *Am J Kidney Dis*. 2014 May;63(5):713-35. doi: 10.1053/j.ajkd.2014.01.416.
35. Dorgham DA, Anwar S, Khaled A. Infection in systemic lupus erythematosus patients. *Egypt Rheumatol*. 2021;43(2):115-118.
36. Momtaz OM, Senara SH, Zaky SH, Mohammed ES. Critically ill systemic lupus erythematosus patients referred to the intensive care unit of Fayoum University Hospital: frequency, complications, and outcome. *Egypt Rheumatol*. 2019;41(2):129-133.
37. Lee JW, Park DJ, Kang JH, et al. The rate of and risk factors for frequent hospitalization in systemic lupus erythematosus: results from the Korean lupus network registry. *Lupus*. 2016 Nov;25(13):1412-1419. doi: 10.1177/0961203316640916. Epub 2016 Jul 11.
38. Jiang J, May P. Proportion of deaths in hospital in European countries: trends and associations from panel data (2005-2017). *Eur J Public Health*. 2021 Dec 1;31(6):1176-1183. doi: 10.1093/eurpub/ckab169.
39. Broad JB, Gott M, Kim H, et al. Where do people die? An international comparison of the percentage of deaths occurring in hospital and residential aged care settings in 45 populations, using published and available statistics. *Int J Public Health*. 2013 Apr;58(2):257-67. doi: 10.1007/s00038-012-0394-5.
40. Clarke AE, Esdaile JM, Bloch DA, et al. A Canadian study of the total medical costs for patients with systemic lupus erythematosus and the predictors of costs. *Arthritis Rheum*. 1993 Nov;36(11):1548-59. doi: 10.1002/art.1780361109.
41. Edwards CJ, Lian TY, Badsha H, et al. Hospitalization of individuals with systemic lupus erythematosus: characteristics and predictors of outcome. *Lupus*. 2003;12(9):672-6. doi: 10.1191/0961203303lu452oa.
42. Petri M, Genovese M. Incidence of and risk factors for hospitalizations in systemic lupus erythematosus: a prospective study of the Hopkins Lupus Cohort. *J Rheumatol*. 1992 Oct;19(10):1559-65.
43. Teh CL, Ling GR. Causes and predictors of mortality in hospitalized lupus patient in Sarawak General Hospital, Malaysia. *Lupus*. 2013 Jan;22(1):106-11. doi: 10.1177/0961203312465780. Epub 2012 Oct 30.
44. Adwan MH, Qasem U, Mustafa KN. In-hospital mortality in patients with systemic lupus erythematosus: a study from Jordan 2002–2017. *Rheumatol Int*. 2020 May;40(5):711-717. doi: 10.1007/s00296-020-04538-z. Epub 2020 Mar 7.
45. Mu L, Hao Y., Fan Y, et al. Mortality and prognostic factors in Chinese patients with systemic lupus erythematosus. *Lupus*. 2018 Sep;27(10):1742-1752. doi: 10.1177/0961203318789788.

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