	HR	95% CI	P
Age, y	0.9336	0.8947 to 0.9742	0.0016
Disease duration, y	0.9712	0.9257 to 1.0191	0.2340
SLEDAI	1.1193	0.8084 to 1.5498	0.4971
SLICC	1.5583	0.7785 to 3.1191	0.2102
[HCQm] ng/mL	0.9997	0.9987 to 1.0007	0.3983
[DCQ] ng/mL	0.9973	0.9922 to 1.0024	0.3048
Clinical remission off/on therapy	1.3702	0.6001 to 3.1282	0.4546
Complete remission off/on therapy	0.7298	0.3197 to 1.6663	0.4546
Time remission	0.7984	0.6287 to 1.0138	0.0647
Previous renal involvement	1.1207	0.4942 to 2.5416	0.7850
Time HCQ	0.9735	0.9128 to 1.0383	0.4140
Glucocorticoids	2.1489	0.8966 to 5.1502	0.0863
Immunosuppressive therapy	3.6639	1.5924 to 8.4304	0.0023
HCQ dosage	0.4995	0.2113 to 1.1805	0.1137

Figure 1. Predictors of flares analyzed by Cox regression.*Serena Fasano and Valentina Messinini contributed equally to this abstract

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AB0280

THE INFLUENCE OF TARGET THERAPY AS WELL AS GLUCOCORTICOID TAPERING ON DISEASE FLARE IN SYSTEMIC LUPUS ERYTHEMATOSUS: RESULTS FROM A PROSPECTIVE CHINESE COHORT

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Background: Treat-to-target in systemic lupus erythematosus (SLE) has been proposed for 7 years and several recommendations were developed [1]. In these recommendations, prevention of flares should be a realistic target. Meanwhile, 'remission' or 'low disease activity' was recommended as the treatment target and minimizing glucocorticoids (GC) dose or withdrawal if possible was suggested in the maintenance treatment. However, would target therapy and GC tapering/withdrawal influence disease flare?

Objectives: To investigate the frequency and determinants of disease flare, especially the influence of target therapy as well as GC tapering on flare in Chinese lupus patients.

Methods: The baseline and follow-up data of all consecutive patients in a prospective longitudinal lupus cohort from January 2017 to June 2020 were collected. The lupus low disease activity state (LLDAS) was defined as in Golder et al., 2019[2]. The criteria for remission were from DORIS definitions [3]. Flare was assessed using the SELENA-SLEDAI flare index [4].

Results: We enrolled 185 patients with disease duration at recruitment of 2.3 (0.8–7.7) years. During the 26.2 (12.5–34.5) months of follow-up, 73 (39.5%) patients experienced 95 flares, including 70 mild/moderate and 25 severe flares. The incidence of flare per patient-year was 0.27. Kaplan-Meier analyses showed that compared with those who never achieved LLDAS or DORIS, the patients who achieved the target at least once had a higher flare free survival rate; meanwhile, the patients with prednisone withdrawn had significantly lower flare free rate compared with those with small dose of GC maintained (≤ 7.5 mg/d) (Figure 1A), but among the patients with different prednisone maintain doses (7.5–5mg, 5–2.5mg, and ≤ 2.5 mg) there was not significant difference (Figure 1B). Cox regression analysis showed that younger age at disease onset and lower Complement 3 (C3) level at recruitment were independent risk factors for flare and achieving LLDAS or DORIS $\geq 50\%$ of visits was independent protective factor (Table 1).

Conclusion: In this Chinese prospective SLE cohort, age at disease onset, C3 level at recruitment and therapeutic target achieving influenced disease flare independently and significantly. GC tapering in appropriate patients and with appropriate pace did not increase the flare rate, but prednisone withdrawal may induce more disease exacerbation, which needs to be confirmed by large prospective studies.

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Table 1. Determinants of disease flare by multivariate Cox regression analyses

	Model 1 (LLDAS) ^Δ			Model 2 (RONT) ^Δ			Model 3 (Complete RONT) ^Δ		
	HR	95%CI	P value	HR	95%CI	P value	HR	95%CI	P value
Age at disease onset (years) [†]	0.97	0.95-0.99	0.004	0.97	0.95-0.99	0.003	0.97	0.95-0.99	0.003
Anti-dsDNA positive at recruitment	1.34	0.82-2.18	0.217	1.12	0.68-1.85	0.649	1.19	0.73-1.96	0.486
C3 (mg/L) at recruitment	0.998	0.997-0.999	0.004	0.998	0.997-0.999	0.007	0.998	0.997-1.000	0.010
Minimum prednisone dose during follow-up (mg/d)	0.98	0.90-1.08	0.716	1.01	0.94-1.08	0.747	1.04	0.97-1.11	0.243
Therapeutic target achieved $\geq 50\%$ of observations ^Δ	0.60	0.39-0.94	0.001	0.54	0.34-0.87	0.011	0.68	0.51-0.92	0.011

^Δ In the three hazard models, the different target achievement status were included respectively. RONT: Clinical remission on treatment; Complete RONT: Complete remission on treatment.

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AB0281

MORTALITY PREDICTORS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: A SINGLE TERTIARY CENTER REFERRAL EXPERIENCE

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Background: Systemic Lupus Erythematosus (SLE) is a heterogeneous chronic multisystemic disease that has an increased risk of requiring inpatient management with higher morbidity and mortality rates.

The most frequent causes of mortality are a high disease activity, infections, and cardiovascular diseases.

Objectives: To determine the predictors of mortality in patients diagnosed with SLE.

Methods: A longitudinal, descriptive, observational study was performed from March 2017-March 2020 at the University Hospital of the Autonomous University of Nuevo León, México, including patients with a diagnosis of SLE according to the ACR-EULAR 2012/2019 criteria.

Descriptive statistics were used for demographic variables. Associations between mortality and clinical variables were determined using binary logistic regression analysis; a p < 0.05 was considered statistically significant. The SPSS program version 20 was used.

Results: One-hundred and eighty-six patients with SLE diagnosis who fulfilled ACR-EULAR 2012/2019 criteria were included, 161 (86.6%) were women; the mean age was 35.8 (SD 15.41) years. The main chief complaints were dyspnea 31 (16.7%), fever in 28 (15.1%), renal involvement in 21 (11.29%), and arthritis in 19 (10.22%) patients.

Of the 186 patients, 34 (18.3%) had a readmission, 13 (7%) required intensive care unit management, 68 (36.6%) had some type of infection and 22 (11.8%) died. Factors associated with mortality are shown in table 1.

Patients receiving steroids and immunosuppression previous to hospitalization were 73 (39.2%) and 92 (49.5%) respectively.

Conclusion: Infections remain the leading cause of death in SLE patients. Factors that predispose to infections are a chronic use of steroids and immunosuppression as well as high activity of disease.