every 4 weeks, with the primary endpoints assessed at Week 52, in patients with moderate to severe SLE despite standard-of-care treatment.\textsuperscript{1,2} BICLA responses were compared between anifrolumab 300 mg and placebo groups, and robustness of BICLA responses was assessed across protocol-defined subgroups.

**Results** TULIP-2 (anifrolumab, n=180; placebo, n=182) and TULIP-1 (anifrolumab, n=180; placebo, n=184) had comparable BICLA responses (figure 1). Across multiple subgroups, higher percentages of patients achieved BICLA responses at Week 52 in the anifrolumab vs placebo arms. There was concordance of BICLA responses favoring anifrolumab across the protocol-defined subgroups of baseline disease severity (SLEDAI-2K \(\leq 10\) points [difference 15.3%, TULIP-2; 16.9%, TULIP-1] vs \(> 10\) points [difference 16.7%, TULIP-2; 17.1%, TULIP-1]) and baseline oral corticosteroid use (prednisone or equivalent \(\leq 10\) mg/d [difference 20.1%, TULIP-2; 16.2%, TULIP-1]) vs \(> 10\) mg/d [difference 12.0%, TULIP-2; 17.7%, TULIP-1]). Other subgroups including age, sex, age at onset, race, and anti-drug antibody status showed similar uniformity of response.

**Conclusions** The uniformity of robust BICLA response rates across prespecified subgroups in both phase 3 trials shows consistent clinical benefit of anifrolumab irrespective of patient baseline characteristics.

### Atherosclerotic Vascular Events in a Multicentre Inception SLE Cohort

**Background/Purpose** The prevalence of atherosclerotic vascular events (AVE) in published literature of an inception cohort with SLE is 10%. We aimed to investigate the accrual and the associated factors of AVE in a multinational multiethnic inception cohort of patients with SLE.

**Methods** A large 33-centre multinational inception cohort of SLE patients was followed yearly according to a standardized protocol between 1999–2017. AVEs are attributed to atherosclerosis on the basis of SLE being inactive at the time of the event, and the presence of typical atherosclerotic changes on imaging or pathology and/or evidence of atherosclerosis elsewhere. Analysis included descriptive statistics, rate of AVE from November 2006 to January 2019. Death was the primary outcome. The model was developed using Cox proportional hazards regression modeling. We developed a prognostic index (PI), summing the number of risk points corresponding to weighted covariates, which were used to configure the nomogram. Internal validation of the nomogram was assessed by discrimination and calibration using bootstrapping.

**Results** Of the 310 patients included in the study, 68 deaths (22.2%) occurred at a median follow-up of 4.9 (interquartile range [IQR] 3.2–6.3) years. The final prognostic model included 6 variables: N terminal-pro brain natriuretic peptide (NT-proBNP), Lactic Dehydrogenase (LDH), Direct Bilirubin table 1. The inclusion of aCL/LA led to the exclusion of 405 patients. Prior other nonatherosclerotic vascular events and high BMI were predictive of first AVE while only antimalarial therapy demonstrated a highly significant protective effect, [HR (95%CI): 0.54 (0.32, 0.91)], after adjustment for the other factors in the model.

**Conclusion** More effective control of classic atherosclerotic risk factors and more frequent use of antimalarial may have both contributed to controlling AVEs in this inception cohort.

### Multivariate Models

**Abstract O26 Table 1 Multivariate Models. Outcome: first AVE**

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Hazard Ratio (95% CI)</th>
<th>p-value</th>
<th>Hazard Ratio (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prior other VEs</td>
<td>4.00 (1.55, 10.3)</td>
<td>0.004</td>
<td>4.76 (1.80, 12.6)</td>
<td>0.002</td>
</tr>
<tr>
<td>Female</td>
<td>0.60 (0.32, 1.24)</td>
<td>0.12</td>
<td>0.57 (0.28, 1.13)</td>
<td>0.11</td>
</tr>
<tr>
<td>Never vs ever smoker</td>
<td>0.51 (0.24, 1.06)</td>
<td>0.07</td>
<td>0.48 (0.23, 1.03)</td>
<td>0.06</td>
</tr>
<tr>
<td>Ex-smoker vs current smoker</td>
<td>0.67 (0.30, 1.48)</td>
<td>0.33</td>
<td>0.53 (0.23, 1.21)</td>
<td>0.13</td>
</tr>
<tr>
<td>Anti-malarials</td>
<td>0.54 (0.32, 0.92)</td>
<td>0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI 30–40</td>
<td>0.95 (0.48, 1.78)</td>
<td>0.89</td>
<td>0.88 (0.42, 1.87)</td>
<td>0.74</td>
</tr>
<tr>
<td>BMI &gt;40</td>
<td>2.74 (1.04, 1.18)</td>
<td>0.04</td>
<td>3.10 (1.78, 2.3)</td>
<td>0.02</td>
</tr>
<tr>
<td>aCL/LA ever</td>
<td>1.73 (0.95, 3.12)</td>
<td>0.07</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

^ Missing values in the predictors led to fewer events in some analyses. BMI – body mass index; aCL/LA – anticardiolipin/lupus anticoagulant; VEs – vascular events

### Development and Validation of a Multivariable Model for 5-Year Survival in Systemic Lupus Erythematosus-Associated Pulmonary Arterial Hypertension: CSTAR-PAH Cohort Study

**Background** Pulmonary arterial hypertension (PAH) is the major mode of death in systemic lupus erythematosus (SLE), but there is no validated algorithm to identify those at highest risk.

**Methods** A prognostic model was developed from a multicenter, longitudinal cohort study of 310 consecutively evaluated patients with SLE-associated PAH. The study was conducted from November 2006 to January 2019. Death was the primary outcome. The model was developed using Cox proportional hazards regression modeling. We developed a prognostic index (PI), summing the number of risk points corresponding to weighted covariates, which were used to configure the nomogram. Internal validation of the nomogram was assessed by discrimination and calibration using bootstrapping.

**Results** Of the 310 patients included in the study, 68 deaths (22.2%) occurred at a median follow-up of 4.9 (interquartile range [IQR] 3.2–6.3) years. The final prognostic model included 6 variables: N terminal-pro brain natriuretic peptide (NT-proBNP), Lactic Dehydrogenase (LDH), Direct Bilirubin...
(DBIL), 6-minute walking distance (6MWD), serositis and alopecic. 5-year survival probability-predictive nomogram with PI in the main analysis was established (figure 1). The model's ability to predict risk was validated with C statistic (0.82 [95% CI, 0.73–0.91]) and calibration curve. Risk stratification was made based on PI to improve the primary prevention and management of SLE-associated PAH.

Conclusions This new, validated risk stratification model for SLE-PAH may provide individualized estimates of risk at 5 years using readily obtained clinical risk factors. External validation studies are required to demonstrate the accuracy of this model’s predictions in diverse patient populations.

Acknowledgements This work was supported by the Chinese National Key Technology R&D Program, Ministry of Science and Technology (2017YFC0907601, 2017YFC0907602) and the Chinese National High Technology Research and Development Program, Ministry of Science and Technology (2012AA02A513).

Abstract 027 Figure 1 The nomogram based on the six significant predictors for survival

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CHARACTERISTICS AND RISK FACTORS OF PULMONARY EMBOLISM IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: A SINGLE-CENTER COHORT STUDY

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Background Pulmonary embolism (PE) is life threatening but evidence assessing risk factors of PE in systemic lupus erythematosus (SLE) is scarce. This study was conducted to explore the characteristics and risk factors of PE in SLE patients.

Methods Using the Hospital Information System of Peking Union Medical College Hospital, we conducted a case-control study in SLE patients complicated with PE (SLE-PE) and age-, sex-, and entry-time-matched control group (SLE-non-PE). Clinical and laboratory data were collected. We explored the risk factors of SLE-PE using multivariate logistic regression analyses.

Results A total of 90 patients were confirmed with PE from 6994 SLE patients. The annual incidence was 1.29% (95% CI: 1.15% to 1.42%), higher than that in general in-patients (0.347% and 95% CI: 0.34% to 0.354%). The overall incidence of PE in male SLE patients (1.86% and 95% CI: 1.40% to 2.32%) was higher than that in female SLE patients (1.21% and 95% CI: 1.07% to 1.35%). 257 contemporaneous SLE patients without PE were enrolled as control cohorts. In the SLE-PE group, the majority were female (74/90; 82.2%), with a mean duration of SLE before PE 3.04±2.16 years, and a high mortality rate of 8.9%. Multivariate analysis revealed that duration of SLE course <1.5 years (OR 3.501(1.801–6.804), p<0.001), lupus nephritis (OR 2.692(1.328–5.457), p =0.006), hypoalbuminemia (OR 2.819(1.272–6.244), p=0.011), high hsCRP (OR 3.163(1.499–6.675), p=0.003), aPL positive (OR 10.262(4.691–22.447), p <0.001) and glucocorticoids, highest dose (OR 1.001(1–1.002), p=0.068) were significant independent risk factors of PE in SLE patients. Use of hydroxychloroquine (OR 0.291(0.139–0.608), p =0.001) was a protective factor of PE in SLE patients.

Conclusions This study provides general population-based evidence that SLE patients have an increased risk of PE. Increased vigilance in preventing this serious, but preventable complication, especially within months after SLE diagnosis is recommended.