confirmed by x-rays. Analyses were performed with logistic regression (forward selection procedure). Outcome measures were incident fracture in general (yes/no), vertebral fracture (yes/no), and peripheral fracture (yes/no).

Results Of the 145 included patients, 131 (90%) were females and 100 (69%) Caucasian. The mean age was 41 years (SD 12) at baseline, and median follow-up was 7.2 years (IQR 6.12). A total of 42 incident fractures (vertebral and peripheral) occurred during 998 patient years. The incidence rate of vertebral and peripheral fractures was 2.0 per 100 patient years (95% CI 1.303.13), and 2.20 per 100 patient years (95% CI 1.453.35), respectively.

Any fracture (both vertebral and peripheral) was predicted by postmenopausal status and Caucasian ethnicity. Vertebral fractures were predicted by age, in which the older the SLE patient, the higher the odds of getting vertebral fractures. Peripheral fractures were predicted by history of stroke, postmenopausal status and moderate alcohol use (112 units per week). Use of higher dosages of alcohol (>13 units per week) did not reduce peripheral fracture occurrence. Table 1 shows the final prediction models.

Conclusions The results of our study suggest a twofold increased risk of both vertebral and peripheral fractures in SLE patients compared to the general population.^{1 2} Age, Caucasian ethnicity and postmenopausal status are important risk factors for incident fractures in SLE. In addition, special attention should be paid to SLE patients with a history of stroke since this subgroup of patients is at high risk of peripheral fractures.

Funding Source(s): None

REFERENCES

1. Bultink IEM, et al. *Osteoporos Int* 2014;**25**:127583.
2. Ballane G, et al. *Osteoporos Int* 2017;**28**:153142.

10

PERFORMANCE OF SLEDAI-2K RESPONDER INDEX-50 IN A RANDOMIZED PLACEBO-CONTROLLED TRIAL WITH USTEKINUMAB (UST) IN SYSTEMIC LUPUS ERYTHEMATOSUS

¹Zahi Touma, ¹Dafna D Gladman*, ²Kaiyin Fei, ²Y Irene Gregan, ²Robert Gordon, ²Kim Hung Lo, ²Shawn Rose, ³Murray B Urowitz. ¹Krembil Research Institute, University of Toronto; ²Janssen Research and Development, LLC; ³Krembil Research Institute, University of Toronto

10.1136/lupus-2019-lsm.10

Background While traditional Systemic Lupus Erythematosus (SLE) Disease Activity Index 2000 (SLEDAI-2K) scoring assesses complete SLE response for individual disease manifestations, the SLEDAI-2K Responder Index-50 (S2K RI-50) evaluates responses using partial improvement (50%) in each of the 9 organ-systems of SLEDAI-2K and generates a total score. We aimed to evaluate the performance of S2K RI-50 at 24 weeks in a randomized, placebo (PBO) controlled trial of UST in patients with moderate-to-severe SLE disease activity to ascertain a minimal threshold of partial improvement.

Methods The UST phase 2, PBO-controlled study enrolled adults with active disease (SLEDAI score 6 with 1 BILAG A and/or 2 BILAG B scores) despite standard-of-care therapy. Patients (n=102) were randomized (3:2) to receive UST IV~6 mg/kg or PBO at week (wk) 0, followed by SC injections of UST 90 mg q8w or PBO beginning at wk8, both added to standard of care. We calculated S2K RI-50 response in

Abstract 10 Table 1 S2K RI-50 response rates at Wk24 for various cut-offs to define response

Decrease from Baseline	UST (%) ^{a,b} (n=62)	PBO (%) ^{a,b} (n=40)	Difference between UST and PBO	p-value ^c
1 Point Decrease	95.3	88.6	6.7	0.13
2 Point Decrease	93.5	79.3	14.2	0.03
3 Point Decrease	84.1	70.9	13.2	0.08
4 Point Decrease	84.0	60.7	23.3	0.01
5 Point Decrease	73.5	50.7	22.8	0.02
6 Point Decrease	70.5	44.0	26.5	0.01

^a Values for subjects meeting treatment failure criteria are set to missing from point of treatment failure forward.

^b Response based upon multiple imputations for missing data from Wk16 to Wk24, where Markov chain Monte Carlo method is used to make missing pattern monotone and serial logistic regression is used to impute monotone missing. The imputation model includes treatment group and baseline SLEDAI-2K covariate.

^c Test for greater treatment effect in UST over PBO (alternative hypothesis) is based upon logistic regression with treatment group, baseline SLEDAI-2K, baseline medication use for SLE and race as covariates.

patients receiving UST (n=62) vs PBO (n=40) at wk24 using increasing S2K RI-50 reductions of 1, 2, 3, 4, 5, or 6 points from baseline to determine 50% improvement. In order to determine a minimal cut-off to discriminate a treatment effect reflecting partial improvement, nominal level was set at 0.05 for this post hoc analysis.

Results The performance of S2K RI-50 in detecting the treatment difference between UST and PBO with various cut-offs is presented in table 1. A 2-point reduction in S2K RI-50 was the lowest threshold to demonstrate a p-value<0.05. As an example, this could reflect partial improvement in one of the following: renal, arthritis, or myositis, or partial improvement in two of the following disease manifestations: rash, alopecia, mucosal ulcers, pleurisy, pericarditis, low complement or increased anti-dsDNA.

Conclusions S2K RI-50 captures partial improvement of 50% in SLE disease activity in the most common disease manifestations in SLE. S2K RI-50 could be used as an outcome in clinical trials as a clinically meaningful measure of partial improvement.

Funding Source(s): Janssen Research and Development, LLC, supported this study.

11

INDUCTION OF VASCULITIC LESIONS IN LUPUS-PRONE MICE: A MODEL FOR LUPUS-ASSOCIATED PERIPHERAL VASCULAR DISEASE

Tatsuya Ishikawa*, Meiyang Wang, Isela Valeram, George Sarantopoulos, Ram Raj Singh. University of California, Los Angeles

10.1136/lupus-2019-lsm.11

Background Systemic lupus erythematosus is an autoimmune disorder that affects many organs. Many patients develop vasculitis in skin and internal organs. There is no effective treatment for lupus-associated vasculitis. Research on the pathogenesis of lupus vasculitis has been hampered due to lack of appropriate model systems. Many patients with lupus develop chronic changes in skin, kidney and other organs, characterized by fibrosis. To understand the induction and mechanisms of fibrosis in lupus, we injected lupus-prone mice