

compared to 39.4% of those diagnosed within a year. Respondents diagnosed after a year were more likely to report insurance not covering costs, long wait times for appointments with primary care physicians (PCP), and lack of nearby doctors treating lupus.

Conclusion Multiple factors associated with lupus diagnosis delays, including delayed access to specialists, onerous health care costs, and health insurance obstacles. An ongoing shortage of rheumatologists may further magnify the delay in diagnosis and explain the apparent long wait times for specialists, misdiagnosis, and increasing costs. Further practitioner education, rheumatology provider recruitment, and studies examining reasons for lupus diagnosis delays may help us better understand and reduce inequities in reaching a prompt lupus diagnosis.

It is important to recognize the limitations of the study; the convenience sample study design lacks the rigor of random sampling, some questions may have introduced recall bias, and socioeconomic status and disease burden at time of diagnosis weren't addressed in this survey.

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1400 – Lupus-targeted therapeutics

1401

EFFECTS OF ANIFROLUMAB ON RENAL DISEASE IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Background The type I interferon (IFN) receptor antibody, anifrolumab, has shown efficacy in patients with systemic lupus erythematosus (SLE) in the phase 3 TULIP-1 and TULIP-2 trials. Type I IFN dysregulation is associated with lupus nephritis (LN) pathogenesis. Pooled TULIP data were analyzed to assess baseline characteristics of patients with and without renal involvement and to evaluate the effects of anifrolumab on renal disease.

Methods TULIP-1 and TULIP-2 were randomized, placebo-controlled trials of intravenous anifrolumab in patients with moderate to severe SLE despite standard therapy, which excluded patients with severe active LN. Renal involvement at baseline was defined as any of the following: British Isles Lupus Assessment Group (BILAG)-2004 renal score A–C; SLE Disease Activity Index 2000 (SLEDAI-2K) renal score >0; urine protein–creatinine ratio (UPCR) >0.5 mg/mg. Baseline characteristics were evaluated in patients with and without renal involvement, and the following endpoints were compared for the anifrolumab 300 mg and placebo groups: cumulative UPCR (area under the curve, AUC) through Week (W)52; percentage of patients with UPCR >0.5 mg/mg at baseline who improved to UPCR ≤0.5 mg/mg at W52; cumulative glucocorticoid (GC) use (AUC) through W52; and percentage changes in complement C3/C4 from baseline to W52.

Results Of the 726 patients in TULIP-1/2 (anifrolumab, n=360; placebo, n=366), 99 had renal involvement at baseline (anifrolumab, n=45; placebo, n=54), 57 of whom had

Abstract 1401 Table 1 Renal endpoints in TULIP-1 and TULIP-2

Endpoint (baseline to Week 52)	Placebo	Anifrolumab 300 mg
UPCR AUC^a		
n	54	45
LS mean (SE)	271.8 (54.8)	217.7 (60.0)
LS mean difference (SE), 95% CI	–54.1 (54.3), –161.9, 53.6	
Improvement from >0.5 to ≤0.5 mg/mg UPCR^b		
n	33	24
Patients with improvement (%)	36.3	41.2
Difference, % (SE), 95% CI	4.9 (13.3), –21.1, 30.9	
Glucocorticoid AUC^c		
n	54	45
LS mean (SE)	3524.5 (339.0)	3314.2 (365.2)
LS mean difference (SE), 95% CI	–210.3 (332.6), –870.7, 450.1	
Change in C3/C4 (%)^c		
C3		
n	31	21
Mean (SE)	20.3 (6.2)	26.6 (5.0)
C4		
n	19	14
Mean (SE)	29.1 (12.0)	38.7 (13.8)

AUC, area under the curve; CI, confidence interval; LS, least squares; UPCR, urine protein–creatinine ratio; SE, standard error; n, number satisfying baseline inclusion criteria for subgroup.

^aPatients with baseline renal involvement; analysis of covariance.

^bStratified Cochran–Mantel–Haenszel.

^cPatients with renal involvement and abnormal C3/C4 at baseline.

UPCR >0.5 mg/mg (anifrolumab, n=24; placebo, n=33). Patients with vs without renal involvement had a lower mean age (37.8 vs 42.4 years) and were more likely to be male (14.1% vs 6.1%), Asian (16.2% vs 9.6%), IFN gene signature high (89.9% vs 81.5%), and anti-dsDNA positive (69.7% vs 40.4%); have a SLEDAI-2K score ≥10 (91.9% vs 68.4%); and be receiving GC ≥10 mg/day (67.7% vs 49.1%) or mycophenolate (26.3% vs 11.5%) at baseline. Among patients with baseline renal involvement, anifrolumab treatment was associated with a greater improvement vs placebo in cumulative UPCR (AUC) through W52 (table 1). More patients improved from UPCR >0.5 mg/mg at baseline to ≤0.5 mg/mg at W52 with anifrolumab vs placebo (table 1). Among patients with renal involvement, cumulative GC use (AUC) through W52 was lower with anifrolumab vs placebo and there were greater improvements in C3/C4 from baseline to W52 (table 1).

Conclusions TULIP data suggest renal benefit with anifrolumab in patients with SLE with mild/stable renal disease, supporting further investigation into anifrolumab's efficacy in patients with active LN.

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AUGMENTED GLUCOSE DEPENDENCY OF AUTOREACTIVE B CELLS PROVIDES A TREATMENT TARGET FOR LUPUS

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