P153 REAL WORLD EXPERIENCE WITH BELIMUMAB IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS IN A PORTUGUESE SINGLE CENTER COHORT

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Objective To describe the experience of belimumab in Systemic Lupus Erythematosus (SLE) patients between 2013 and 2023 in a Portuguese Single Center Cohort.

Methods Patients who were treated with belimumab were included. Retrospective patients' clinical charts review to demographic data, clinical manifestations, serological characteristics, previous infections and treatments, clinical and serological response to belimumab, time on belimumab, reasons to stop treatment and adverse events. SPSS was used to statical analysis.

Results Twenty-eight Caucasian patients with SLE were treated with belimumab between 2013 and 2023 in our center, 27 (96,4%) female and 1 male (3,6%). Age at diagnosis was in average 25,0+/-9,5 years old, age first treatment with belimumab was 36,4+/-9,1 and disease duration before belimumab 10,8+/-6,1 years. Seventeen patients started intravenous belimumab and 2 of them switch to subcutaneous when it became available in our country. Eleven patients started subcutaneous. When belimumab was started constitutional involvement was present in 18 (64,3%), mucocutaneous 20 (71,4%), musculoskeletal 19 (67,9%), serositis 6 (21,4%), vasculitis 6 (21,4%), neurological 3 (10,7%) and renal 12 (42,9%). In average patients had 3 active SLE organ involvement. Four patients stopped belimumab before 3 months due to severe infection (2 patients), severe alopecia (1) and lost to follow-up (1). The patients with severe infection after belimumab had higher burden of immunosuppression and previous severe infections. From the patients who did more than 3 months of treatment, 19 (82,6%) had clinical response and 15 (65,2%) had serological response. In average, patients who responded were on belimumab treatment for 28,4+/-15,24 months. Time to first flare after belimumab was on average 15,5+/-10,6 months, and just 1 patient stopped belimumab after the first flare. Eight patients who responded, stopped belimumab due to lost of efficacy (3 patients), prolonged remission (2), patient's choice (1), depressive symptoms (1) and need to treat other concomitant disease (1). Minor infections occurred but none led to belimumab suspension.

Conclusions Our cohort experience with belimumab add-on treatment led to clinical response in SLE patients in 82,6% of the patients. Early severe infections happened in patients with previous higher immunosuppression and severe infections burden.

P154 RILONACEPT USE IN LUPUS PERICARDITIS

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Objective Lupus pericarditis affects 22% of patients with systemic lupus erythematosus (SLE), is associated with worse outcomes, and often requires immunosuppression. Rilonacept is an IL-1 receptor antagonist approved for the treatment of recurrent idiopathic pericarditis, but its efficacy in lupus pericarditis is unknown. Here, we report the efficacy of rilonacept in a case series of patients with lupus pericarditis.

Methods We describe a case series of 4 patients with refractory lupus pericarditis treated with rilonacept in the Johns Hopkins Lupus Center. All patients met the 2012 SLICC criteria for SLE. Refractory lupus pericarditis was defined as recurring or persistent typical pericardial pain symptoms despite standard-of-care treatment including at least one immunosuppressant.

Results Four patients with refractory pericarditis were included (table 1). All patients were women, age ranged 26–44 years, 2 patients reported White, 1 Black, and 1 Hispanic ethnicity. Extra-pericardial SLE manifestations were heterogeneous among patients. Only 1 of 3 patient had elevated CRP (not measured in one). Two patients were previously treated with anakinra with initial response, but pericarditis redeveloped in both. Rilonacept led to complete resolution of pericardial symptoms in 3 patients, and partial resolution (40%) in 1, within 2 weeks.

Abstract P154 Table 1 Clinical and demographic characteristics of the 4 patients with lupus pericarditis treated with rilonacept

Patient No	Age (years)	Sex	Lupus clinical features	Notable comorbidities	CRP	Ineffective treatments	Response
1	44	F	Serositis, arthritis, thrombocytopenia, leukopenia, alopecia, and ANA.	Inflammatory bowel disease	Low	Hydroxychloroquine, azathioprine, methotrexate, mycophenolate, colchicine, corticosteroids, ibuprofen, belimumab, rituximab, tofacitinib, certolizumab, anakinra.	Complete resolution of pleurisy and pericardial chest pain
2	42	F	Serositis, arthritis, oral ulcers, alopecia, pericarditis, pleurisy, fever, class I lupus nephritis, ANA, anti-dsDNA, anti-Smith, anti-RNP, and low C3/C4.	Stroke	High	Hydroxychloroquine, azathioprine, methotrexate, corticosteroids, ibuprofen, anakinra.	Complete symptoms resolution
3	33	F	Serositis, arthritis, oral ulcers, interstitial lung diseaseANA, anti-dsDNA, anti-RNP		Low	Hydroxychloroquine, azathioprine, methotrexate, mycophenolate, belimumab, ibuprofen, colchicine, corticosteroids.	Resolution of pleurisy, pericardial chest pain improved >40%
4	26	F	Serositis, arthritis, photosensitivity, ANA, anti-dsDNA, anti-RNP, anti-Ro, anti-La, Coombs test, and low complement	Strokes	Unknown	hydroxychloroquine, indomethacin, azathioprine, colchicine, methotrexate, and rituximab	Complete resolution of chest pain (persistent arthritis)

Conclusion Rilonacept successfully treated lupus pericarditis in this case series. Rilonacept should be considered for the treatment of lupus pericarditis.

P155 IDENTIFYING A POPULATION OF PATIENTS FOR INTENSIVE FIRST-LINE THERAPY IN SLE: A CLINICAL AND BIOMARKER MODEL TO PREDICT THE NEED FOR INTENSIVE THERAPY

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Objective To investigate the clinical and biomarkers factors predicting a requirement for intensive therapy or time to intensive therapy from diagnosis of Systemic Lupus Erythematosus (SLE).

Methods We conducted a retrospective longitudinal study of all patients with a diagnosis of SLE from two Leeds Cohort databases (CONVAS and DEFINITION) for over 30 years follow-up. Data collection included demographics, clinical characteristics, the 2019 EULAR/ACR classification criteria score, the SLEDAI-2K score, and routine immunological tests. The primary endpoint was the time from SLE diagnosis to initiation of intensive therapy (cyclophosphamide, rituximab, belimumab, or other biologic agents). Univariable analysis (UVA) and multivariable (MVA) Cox proportional hazards regression models were used to test the potential predictors of the primary endpoint. MVA was done using forward selection and backward elimination with $p<0.1$ associated with

Abstract P155 Table 1 Baseline characteristics, serology, and biomarkers in patients with Systemic Lupus Erythematosus

Variables	Total N=229 (%)	Intensive therapy-given n=110 (%)	Intensive therapy-not given n=119 (%)	*p*-value
Female gender	209 (91.3)	100 (90.9)	109 (91.6)	0.854
Mean age at diagnosis (SD)	38.8 (14.7)	35.67 (13.97)	41.70 (14.87)	0.002
Age by category				
Q1: under 28	63 (27.5)	38 (34.5)	25 (21.0)	0.032
Q2: 29 – 38	56 (24.5)	25 (22.7)	31 (26.1)	0.667
Q3: 39 – 51	55 (24.0)	28 (25.5)	27 (22.7)	0.738
Q4: over 51	55 (24.0)	19 (17.3)	36 (30.0)	0.032
Ancestry				
European	158 (69)	76 (69.1)	82 (68.9)	
South-Asian	31 (13.5)	17 (15.5)	14 (11.8)	
Oriental	5 (2.2)	2 (1.8)	3 (2.5)	
African/Caribbean	17 (7.4)	7 (6.4)	10 (8.4)	
Mixed	11 (4.8)	7 (6.4)	4 (3.4)	
Unknown	7 (3.1)	1 (0.9)	6 (5)	
Intensive therapy given				
Cyclophosphamide	-	45 (40.9)	-	
Rituximab	-	57 (51.8)	-	
Belimumab	-	4 (3.6)	-	
Ocrelizumab	-	2 (1.8)	-	
Efalizumab	-	1 (0.9)	-	
Clinical trial drug	-	1 (0.9)	-	
Median time to censored; year (IQR)	5.9 (1.7,11.6)	2 (0.5,8)	8.6 (5.2,15.3)	< 0.001
Autoantibodies positivity				
Anti-dsDNA Ab	124 (54.4)	63 (57.8)	61 (51.3)	0.322
Anti-Ro Ab	108 (47.2)	66 (60)	42 (35.3)	< 0.001
Anti-La Ab	34 (14.8)	23 (20.9)	11 (9.2)	0.013
Anti-Sm Ab	38 (16.6)	24 (21.8)	14 (11.8)	0.041
Anti-Sm/RNP Ab	55 (24)	30 (27.3)	25 (21)	0.268
Anti-RNP Ab	36 (15.7)	24 (21.8)	12 (10.1)	0.015
Anti-chromatin Ab	81 (35.4)	44 (40)	37 (31.1)	0.159
Anti-ribosomal P Ab	8 (3.5)	5 (4.5)	3 (2.5)	0.486
Mean cumulative number of Ab positivity (SD)	2.1 (1.6)	2.6 (1.8)	1.7 (1.3)	< 0.001
Low complement (C3 and/or C4)	66 (28.8)	43 (39.1)	23 (19.3)	< 0.001
aPL positivity	50 (21.8)	29 (26.4)	21 (17.6)	0.111
Median 2019 EULAR/ACR criteria score (IQR)	16 (12,22)	20 (16,25)	14 (11.5,18)	< 0.001
2019 EULAR/ACR criteria score≥20	79 (34.5)	57 (51.8)	22 (18.5)	< 0.001
Median SLEDAI-2K score (IQR)	10 (6,14)	13 (10,19.8)	7 (6,10)	< 0.001
Median cSLEDAI-2K score (IQR)	8 (5,12)	12 (8,17)	6 (4,8)	< 0.001
SLEDAI-2K≥10	118 (51.5)	85 (77.3)	33 (27.7)	< 0.001

Abstract P155 Table 2 Univariable and Multivariable Cox regression analysis of factors predicting the intensive therapies requirement

Variables	Univariable Hazard ratio (95% CI)	Univariable *p*-value	Multivariable Hazard Ratio (95% CI)	Multivariable *p*-value
Age under 28 vs >28 years	1.1 (0.73–1.70)	0.66	Not included	Not included
European ancestry vs non-European	0.85 (0.52–1.4)	0.52	Not included	Not included
Anti-dsDNA ab positivity	1.2 (0.81–1.7)	0.48	Not included	Not included
Anti-Ro Ab positivity	1.7 (1.20–2.60)	0.005	1.47 (1.00–2.19)	0.052
Anti-La Ab positivity	1.2 (0.75–1.9)	0.45	Not included	Not included
Anti-Sm Ab positivity	1.6 (1.00–2.60)	0.036	Included in MVA but removed from final model as $p>0.1$	
Anti-RNP Ab positivity	1.5 (0.94–2.30)	0.093	Included in MVA but removed from final model as $p>0.1$	
Cumulative number of Ab positivity	1.2 (1.1–1.3)	0.003	Excluded due to collinearity	
Low complement (C3 and/or C4)	2.4 (1.60–3.50)	<0.0001	1.99 (1.32–2.97)	<0.001
2019 EULAR/ACR criteria score>20	2.1 (1.50–3.10)	<0.0001	Included in MVA but removed from final model as $p>0.1$	
cSLEDAI-2K score	1.1 (1.07–1.12)	<0.0001	1.10 (1.07–1.12)	<0.001