24 weeks of study treatment. Improvement of median proteinuria reduction from base line was 64.2% at 12 weeks and 88% at 24 weeks. Serum vitamin D levels were inversely associated with the urinary protein creatinine UP/C ratio ($p < 0.001$) and urinary vit D binding protein DBP/C ($p < 0.001$).

Conclusions Our findings show that new strategy of adding vitamin D therapy as new treatment for 24 weeks to maintain optimal serum 25(OH) D levels and diminish proteinuria in lupus nephritis patients. We need longer duration and more studies to confirm our results from different countries.

PS7:139 EFFICACY AND SAFETY OF RITUXIMAB IN RESISTANT SLE

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Objective Rituximab is a B cell depleting monoclonal anti-CD20 antibody that has been suggested by number of research as a potential Effective agent in Resistant active SLE. however, related clinical trials; ‘Explorer’, ‘lunar’ trials both failed to show clinically significant efficacy of Rituximab compared to placebo. In this uni-centre study, we evaluated the Efficacy and safety of Rituximab in refractory SLE patients.

Abstract PS7:139 Table 1 Characteristics of the 23 SLE patients receiving rituximab

Age	
mean $\pm$ SD years	28.6 $\pm$ 15.2
mean pediatric $\pm$ SD	14.3 $\pm$ 3.1
mean adult $\pm$ SD	33.7 $\pm$ 14.4
Disease duration, mean $\pm$ SD years	4.2 $\pm$ 3.8
Main indication for rituximab % (n)	
Renal (Lupus nephritis)	26.1 (6)
Hematological	21.7 (5)
Neuropsychiatric	30.4 (7)
Cutaneous	13.0 (3)
Lupus nephritis/ hematological (pancytopenia)	8.6 (2)
Disease activity before rituximab	
SLEDAI score, mean $\pm$ SD years	15.0 $\pm$ 8.8
Anti-DNA antibody, % (n)	78.3 (18)
C3, mean $\pm$ SD g/L	0.66 $\pm$ 0.34
C4, mean $\pm$ SD g/L	0.12 $\pm$ 0.09
Previous immunosuppressive medications % (n)	
Prednisone	91.3 (21)
Methylprednisolone	52.2 (12)
Cyclophosphamide	8.7 (2)
Azathioprine	26.1 (6)
Cyclosporine	13.0 (3)
Mycophenolate mofetil	34.8 (8)
Hydroxychloroquine	91.3 (21)
Number of previous immunosuppressive agents, mean $\pm$ SD	3.4 $\pm$ 1.5
Medication concomitant with rituximab % (n)	
Prednisone	65.2 (15)
Cyclophosphamide	26.1 (6)
Hydroxychloroquine	91.3 (21)
IVIg	13.0 (3)
Methylprednisolone	17.4 (4)
Number of immunosuppressive agents concomitant with rituximab, mean $\pm$ SD	2.3 $\pm$ 1.0
Dose of prednisone, mean $\pm$ SD mg/day	
Before rituximab	26.2 $\pm$ 15.5
With rituximab	56.0 $\pm$ 21.3
After rituximab	14.8 $\pm$ 15.3
Rituximab regimen % (n)	
4 x 375 mg/m ²	43.5 (10)
2 x 1 g	30.4 (7)
Others	26.1 (6)

Method We analysed retrospectively the data of resistant SLE patients who received Rituximab

Results Data included 23 refractory SLE patients that received Rituximab which was indicated for lupus nephritis 26.1%, haematological involvement 21.7%, neuropsychiatric complications 30.4%, cutaneous involvement 13.0%, and combination of lupus nephritis and haematological involvement 8.6%. Mean $\pm$ SD of SELENA Modified version – SLEDAI score at baseline was 15.0 $\pm$ 8.8 and 9.2 $\pm$ 9.0 at 6 months after treatment (p value. 002). Among patients with lupus nephritis Complete renal response was noted in 2 (8.7%) out of 8 patients. Partial response was documented in 3 (13.0%). 3 of 7 patients with haematological involvement responded completely, 2 have responded partially the other 2 did not respond to Rituximab. 5 (21.7%) patients of 7 neuropsychiatric patients showed complete response, and no response was noted in 2 (8.7%) patients. 4 out of 5, who showed complete response undergone remission. Two non-responders eventually died. Only 3 (13.0%) patients showed cutaneous involvement. 2 of them showed partial response and remaining 1 showed complete response. Adverse effects were noted in 8 (34.8%) patients. 2 (8.7%) of them reported acute infusion reaction, 4 (17.4%) showed features of severe infection and 2 (8.7%) patients died due to septic shock and multi organ failure (table 3).

Conclusion Rituximab is an effective and relatively safe agent for refractory SLE, additional well-structured controlled studies are needed to prove efficacy in those patients compared to other conventional therapy.

PS7:140 BELIMUMAB IN SYSTEMIC LUPUS ERYTHEMATOSUS. 1 YEAR OF FOLLOW UP

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Objectives To describe patients with Systemic Lupus Erythematosus (SLE) treated with Belimumab (BLM).

Material and methods Review of patients with SLE treated with BLM from 2012 to 2017.

Results We enrolled 18 patients, 16 women and 2 men. The median age at of SLE diagnose was 34.3 (IQR 27–45.7) years and the median age at BLM start was 2.2 (IQR 38.9–47) years. The received sDMARDs median number was 2.5 (IQR 2–4). Before starting BLM 7 patients had received rituximab, 4 cyclophosphamide, 1 abatacept and 1 one efalizumab. At BLM begin 17 patients were taking prednisone, 13 hydroxychloroquine, 7 methotrexate, 1 azathioprine, 1 mycophenolate and 1 leflunomide (table 1).

The median BLM bolus received was 8.5 (IQR 2–32.7) and it was stopped in 4 patients due to lack of efficacy; this 4 patients had persistence of arthritis and non of them had received bDMARD before.

13 patients had at least one antiphospholipid autoantibody positive and 4 developed antiphospholipid syndrome.

The median titer of antinuclear antibodies (ANA) was 1/360 (IQR 1/160–1/1280). All patients but one had ANA positive; among them 9 patients were antiRo positive, 4 antiLa, 4 antiSm and 4 antiRNP positive antibodies. 9 patients were antiDNA positive and 5 were positive for rheumatoid factor (RF) able 1 shows SLEDAI, C3 and C4, antiDNA levels.

Abstract PS7:140 Table 1 Baseline characteristics

Age (years) #	42.26 (38.67-48.03)
Sex (female)	16 (89%)
Age at SLE diagnosis #	34.33 (26.56-45.91)
Disease duration (years) #	8.05 (3.54-14.02)
Previous sDMARD #	2.50 (2.00-4.00)
- Hydroxychloroquine	16
- Methotrexate	12
- Mycophenolate mofetil	9
- Leflunomide	7
- Azathioprine	7
Previous Cyclophosphamide	4
Previous bDMARD	
- Rituximab	7
- Efavizumab	1
- Abatacept	1
Concomitant sDMARD	
- Hydroxychloroquine	13
- Methotrexate	7
- Azathioprine	1
- Mycophenolate mofetil	1
- Leflunomide	1
ANA #	1/360 (1/160-1/280)
- Anti-Ro	9
- Anti-La	4
- Anti-Sm	4
- Anti-RNP	4
Rheumatoid factor	5 (27.8%)
Anti-CCP	1 (5.6%)
Clinical APS	4 (22.2%)
Anti-DNA	9 (50%)

Median (IQR)

Disease duration: years from diagnosis to BLM start

sDMARD: synthetic Disease Modifying Anti-Rheumatic Drugs

bDMARD: biologic Disease Modifying Anti-Rheumatic Drugs

APS: Antiphospholipid Syndrome

Abstract PS7:140 Table 2 One year follow up after BLM treatment

	T0	T3	T6	T12
N° of patients	18	16	14	7
Prednisone dose (mg/day)	6.9 (4.4-12.5)	7.5 (3.1-10.0)	5.0 (5.0-7.5)**	7.5 (1.5-10.0)
SLEDAI	10.0 (8.0-13.7)	4.5 (2.7-10.7)*	5.0 (2.0-10.2)**	5.0 (2.0-10.4)***
C3	73.5 (67.2-101.7)	92.5 (71.0-105.0)*	91.0 (72.7-110.0)**	89 (58.0-102.0)
C4	11.0 (7.75-19.75)	11.0 (8.0-21.2)	15.5 (10.0-24.7)**	11 (2.0-14.0)
Anti-DNA levels	11.7 (1.7-97.5)	17.0 (3.3-115.7)	31 (4.55-107)	29 (5.3-486.0)

All values are Median (IQR)

*p < 0.05 (T0-T3); **p < 0.05 (T0-T6); ***p < 0.05 (T0-T12)

T0: Baseline before BLM begin;

T3: 3 months after BLM;

T6: 6 months after BLM;

T12: 12 months after BLM.

At BLMin 5 patients had renal involvement; 3 of them improved after 3 months of treatment. Among 14 patients with joint involvement, 3 improved by month 3, 2 by month 6 and 1 by month 7 after treatment beginning. 3 patients had heart and lung disease; no one of them improved with BLM. Among 9 patients with skin disease only one showed

improvement after 12 months of treatment. 9 patients had haematological disorders: 1 improved by month one, 1 by month 3, one by month 5 and 2 by month 6 of treatment.

Conclusion In this series BLM improved SLEDAI and complement levels allowing sparing of maintenance corticosteroid in SLE patients.

PS7:141 SAFETY AND TOLERABILITY OF OMALIZUMAB IN SUBJECTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: PRELIMINARY OUTCOMES

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Background Studies performed in Lyn-/- mice indicate that IgE autoantibodies induce a lupus nephritis-like disease. In SLE, these autoantibodies are associated with increased disease activity. We propose that omalizumab, a monoclonal antibody that binds IgE, will reduce SLE activity by reducing circulating IgE autoantibodies, subsequently blocking basophil activation and immune dysregulation. The primary aim of this study was to determine the safety and tolerability of omalizumab in mild to moderate SLE.

Methods SLE participants (n=16) with SLEDAI >4 were randomised to receive omalizumab or placebo (2:1) added to their baseline SLE therapy for 16 weeks, followed by a 16 week open label and 4 week washout period. The regimen was a loading dose of omalizumab 600 mg dose followed by 300 mg every 4 weeks. Type I Interferon (IFN) gene signature was determined using a previously validated four gene interferon score (IFI27, IFI44, IFI44L, RSAD2) using quantitative PCR.

Demographics Variables n=16

Age (Years)

Mean 43.4

STD 13.2

Range 22–69

Disease Duration (years)

Mean 16

STD 13.3

Range 4–50

Race/Ethnicity, N (%)

African American 3 (18.8%)

Asian 2 (12.5%)

Caucasian 4 (25.0%)

Hispanic 7 (43.8%)

Sex, N (%)

Female 16

Male 0

Safety: There were a total of 52 adverse events, with the majority (49) being classified as mild. Three events met the criteria for SAE (primary varicella, pulmonary embolism, and bronchitis). No local or systemic allergic reactions were observed.

Clinical: There was a trend during the first 16 weeks of therapy toward reduction in the SLEDAI 2K score (p=0.077).

Biological: There was a trend in the reduction in the type I IFN signature in subjects treated in the first 16 weeks (p=0.11), especially in those subjects with high baseline IFN scores (p=0.052).

Conclusion The use of omalizumab in subjects with SLE appears to be well tolerated. There was a trend showing efficacy of omalizumab. The type I IFN signature also trended down, particularly in subjects with high signatures at baseline. The data analysis of other clinical parameters and mechanistic studies is still ongoing.