LN was noted in any of the 7 pts. 5 pts are followed by Rheumatology for extrarenal SLE; no extrarenal manifestations are documented in the other 2. Data on SLE Disease Activity pre and post bela were available and scored in 3/5 pts using the SLEDAI-2KG which accounts for clinical and laboratory manifestations, as well as steroid use (Fig. 2). Mean ds-DNA pre and post bela was 133 ± 178 UI/mL and 58 ± 72 UI/mL; C3 89 ± 31 mg/dL pre and 91 ± 21 mg/dL post; C4 35 ± 15 mg/dL pre and 38 ± 7 mg/dL post.

Conclusion: Bela in LN KT recipients may decrease extrarenal manifestations, attenuate CNI toxicity and stabilize allograft function, providing a better alternative to CNI regimens. Furthermore, these data suggest that bela may be a therapeutic option in SLE.

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AB0462 IMMUNIZATION TO RITUXIMAB IS MORE FREQUENT IN AUTOIMMUNE SYSTEMIC AUTOIMMUNE DISEASES THAN IN RHEUMATOID ARTHRITIS AND MAY BE MANAGED BY SWITCHING FROM RITUXIMAB TO OFATUMUMAB

Alice Combier, Gaetane Nocturne, Julien Henry, Rakiba Belkhir, Stephane Pavy, Xavier Mariette, Raphaële Seror. *Bicetre Hospital AP-HP, Rheumatology, Le Kremlin-Bicêtre, France*

Background: The most widely used B cell targeted therapies in autoimmune diseases (AID) is Rituximab (RTX), a murine chimeric monoclonal antibody. Among RTX's side effects, immunization and anti-drug antibodies to RTX (RTX-ADA) production have been reported but their consequences are poorly described. The immunization rate against RTX in rheumatoid arthritis (RA) is 2.7–9.2% and it seems to be higher in other sAID although data is lacking.

Objectives: We aimed to evaluate the frequency, consequences and predictive factors of RTX-ADA in RA and sAID, as well as the use of an alternative B-cell targeted therapy, ofatumumab (OFA) in case of RTX-ADA.

Methods: All patients with RA or sAID treated with RTX from 2012 to 2017 in our tertiary reference centre for sAID were retrospectively studied. Patients who were tested for RTX-ADA were identified. Clinical and biological characteristics of RTX immunized patients were compared to those of non-immunized patients. For patients treated with OFA, clinical and biological efficacy was obtained before and after treatment.

Results: 199 patients were treated with RTX (RA: 124, sAID: 75 including 38 primary Sjögren's Syndrome (pSS), 15 systemic lupus erythematosus, 7 myositis, 6 overlap syndrome, 5 ANCA-associated vasculitides and 4 other sAID). Among the 62/199 (31.1%) tested for RTX-ADA, 14 were positive: 3/35 RA (8.6%) and 11/27 (40.7%) other sAID, ($p=0.005$). Among the whole RTX-treated patients, the frequency of RTX-ADA was 2.4% and 14.7% ($p=0.003$) in RA and in other sAID, respectively. Most of the immunized patients experienced delayed infusion reactions (11/14 [78.5%]). Delayed reactions were observed within the first 15 days after the infusion, and after a median 2 cycles [range; 1–2]. They were mainly rash (72.7%), fever (54.5) and/or abdominal pain (36.3%). Predictive factors of immunization were a sAID compared to RA (40.7% vs 8.6%,

$p=0.005$), and young age (50.5 vs 61.5 years, $p=0.003$). Neither hypergammaglobulinemia, rheumatoid factor, disease activity, nor associated immunosuppressive therapy were associated with RTX-ADA. Among 23 tested patients with SLE or pSS, anti-SSA antibodies tended to be more frequent in immunized patients than non immunized (9/10 (90.0%) vs. 8/13 (61.5%), $p=0.18$). Three pSS patients immunized against RTX were treated with OFA because of associated cryoglobulinemic vasculitis or MALT lymphoma. All 3 experienced complete remission of their disease. Relapse occurred in 2 patients, one with MALT lymphoma after 14 months, one with cryoglobulinemic vasculitis after 10 months and were retreated successfully with OFA. No adverse events were reported.

Conclusion: Our results show that immunization against RTX is more frequent in other sAID than in RA. Testing for ADA must be performed in patients with infusion reactions. Patients immunized to RTX might be treated efficiently with OFA, which should be further evaluated in sAID.

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AB0463 BELIMUMAB IV EFFECT IN GLUCOCORTICOID TREATMENT IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS

Marta De la Rubia Navarro¹, Susana Cerdá Checa², Jose Ivorra Cortés^{1,2}, Elena Grau García¹, Cristina Alcañiz Escandell¹, Inmaculada Chalmeta Verdejo¹, Jorge Juan Frago-Gil¹, Roxana Gonzalez Mazario¹, Luis Gonzalez Puig¹, Isabel Martínez Cordellat¹, Rosa Neguerolles Albuixech¹, Jose Eloy Oller Rodríguez¹, Francisco Miguel Ortiz Sanjuan¹, Cristóbal Páez Perales¹, Elvira Vicens Bernabeu¹, Carmen Nájera Herranz¹, Inés Cánovas Olmos¹, Jose Andrés Román Ivorra^{1,2}. ¹Rheumatology Department. HUP La Fe, Valencia, Spain; ²Medical School. UCV, Valencia, Spain

Background: Systemic lupus erythematosus (SLE) is an autoimmune chronic disease produced by aberrant immunological response that consequently, causes a widespread organic damage. Treatment leads to regulating this disrupted immunological response. As a result, in 2011 Belimumab was approved for adults patients diagnosed with SLE, what is a monoclonal human antibody whose target is a B-lymphocyte stimulator (BLyS), a protein involved in the disease pathogeny.

Objectives: To evaluate the glucocorticosteroid dose variation in adult patients with systemic lupus erythematosus during Belimumab IV treatment.

Methods: Retrospective, observational study that includes patients diagnosed with SLE according to SLICC 2012 criteria, who are treated with Belimumab IV. Treatment has been administrated as an initial dose: 10 mg/kg every 14 days and then, a maintenance dose: 10 mg/kg every 28 days. Data from serological profile, clinical manifestations at diagnoses and at the onset of treatment, existence of comorbidities or other diseases, concomitant therapy directed to the main disease or to the complications that came from the disease, non administration causes and definitive treatment discontinuation were collected from July 2012 until December 2018.

Results: A total of 19 patients (94.74% women) with 28 years old (Q1 14, Q3 31.82) as a median age at diagnosis and 11 (Q1 6.5, Q3 20) years since disease diagnosis were included. Follow up mean was 29 (Q1 7.5, Q3 37) months.

Regards to concomitant therapies, 5 patients (26.31%) were treated with Azathioprine and 3 patients with Methotrexate (15.79%). At the onset of biological therapy, 17 patients were treated with glucocorticoids, at a dose of 10 mg (Q1 9.38, Q3 10.63). At the end of follow up, 18 patients (94.73%) were treated with corticoid and the mean dose was 8.75 mg (Q1 8.75, Q3 8.75).

Table 1. Characteristics of patients on bela.

ID	Age at SLE dx	Race	LN class	Time to KT (yrs)	Type KT	Induction	Reason for conversion	Immunosuppressive regimen before conversion
A	16	Black	IV, VI	13	DDKT	ATG	Non-adherence	TAC, MMF
B	30	Asian		2	LRKT	ATG	Mod IFTA & Arteriosclerosis	TAC, MPA, PRED
C	18	White	IV, V	23	DDKT	ATG	Cortical necrosis	CYC, MMF, PRED
D	19	Black	V	9	LUKT	ATG	CNI side effects	TAC, MPA, PRED
E	22	Asian		5	LRKT	ATG	CNI side effects	TAC, MMF, PRED
F	18	Black	V	17	DDKT	Alentuzumab	CNI side effects	TAC, MMF, PRED
G	25	White (Hispanic)	III, IV	23	DDKT	Basiliximab	TMA (biopsy)	TAC, MPA, PRED