study was aimed to determine the effect of steroid therapy on the performance of IGRA in SLE.

Methods This experimental study included 50 female SLE. Data such as age, disease duration, SLE disease activity index (SLEDAI), steroid therapy doses, and Body Mass Index (BMI) were obtained. Steroid therapy doses were categorized as high-dose (≥7.50 mg) and low-dose steroids (<7.50 mg). Patients were excluded if they had history of TB or current active TB. Each group underwent IGRA testing using the QuantiFERON-TB Gold Plus kit. Data analysis was performed using SPSS.

Results There were 50 (100%) female SLE with average age 32.64 (±8.16) years old, mean disease duration 5.78 (±3.99) years, mean SLEDAI score 2.64 (±3.19), mean steroid therapy doses 8.47 (±8.35) mg/day, and median of BMI 21.7 (16.4–36.0). In high-dose steroids group (n=20), IGRA result was 5% positive, 15% indeterminate, and 80% negative. In low-dose steroids group, 3.3% was positive and 96.7% was negative. There was no significant association between steroid therapies and IGRA results (p=0.084).

Conclusions Steroid therapy causes dysregulation of immune response and has tendency to influence IGRA result in SLE. In our study steroid therapy is not associated with IGRA result. Further study to explore latent tuberculosis infection and steroid therapy in SLE is of great interest.

LP-198 CLINICAL RESPONSE BY SUBGROUPS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) RECEIVING STANDARD OF CARE TREATMENT PLUS PLACEBO (SOC +PBO): A POST HOC ANALYSIS FROM CLINICAL TRIAL DATA

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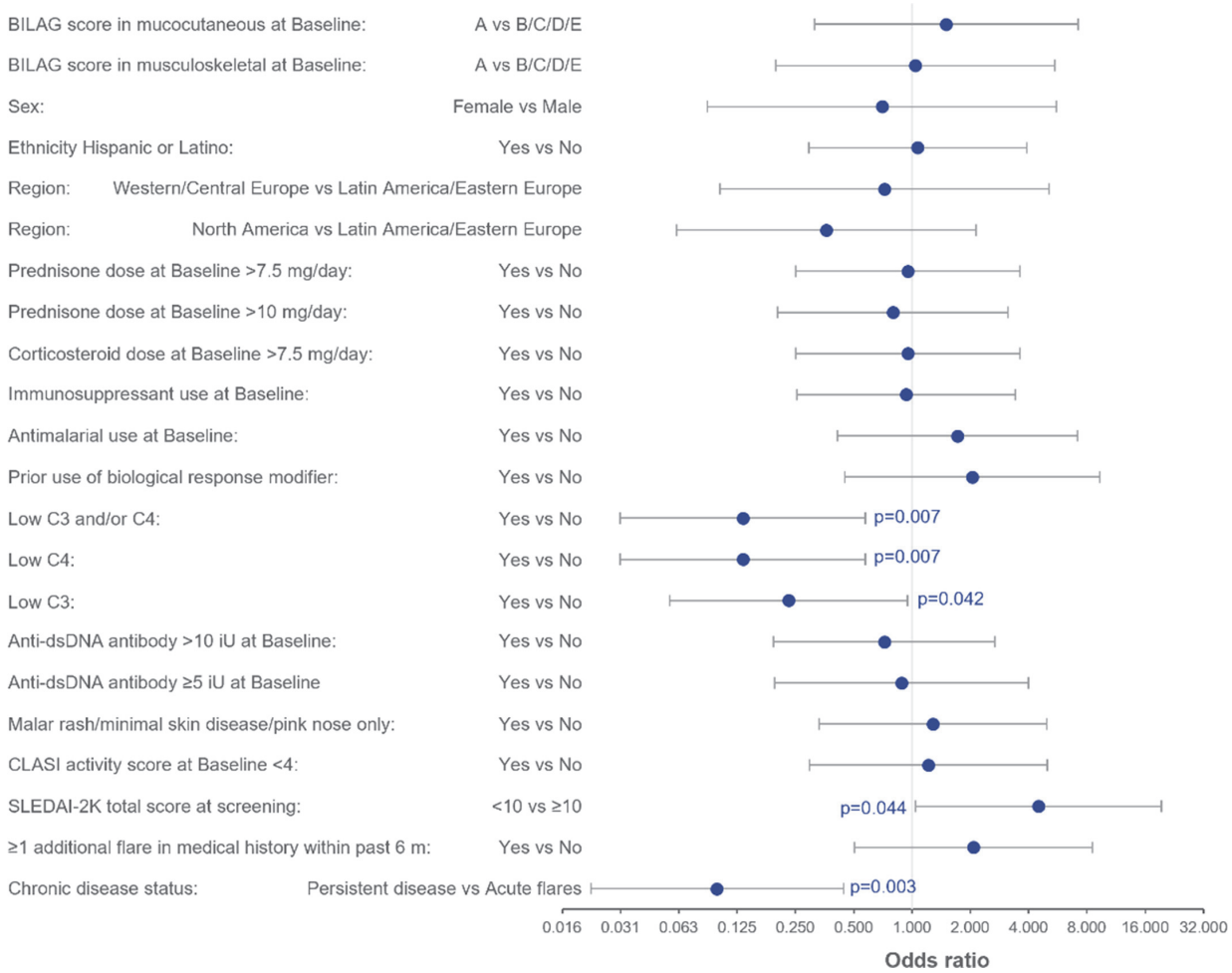
Background Wide-ranging placebo responses challenge SLE trials. This analysis aims to identify predictors of SOC+PBO response in patients with SLE.

Methods Analyses used the SOC+PBO arm of the phase 2b trial of dapirolizumab pegol (NCT02804763), a polyethylene glycol conjugated antigen-binding fragment lacking a functional Fc domain, which inhibits CD40-CD40L interaction.[1]



Abstract LP-198 Figure 1 Univariate analysis of predictors of BICLA response at Week 24 in patients who received SOC+PBO. Anti-dsDNA: anti-double-stranded deoxyribonucleic acid; BICLA: BILAG-based Composite Lupus Assessment; BILAG: British Isles Lupus Assessment Group; CLASI: Cutaneous Lupus Erythematosus Disease Area and Severity Index; m: months; PBO: placebo; SLEDAI-2K: Systemic Lupus Erythematosus Disease Activity Index 2000; SOC: standard of care.

Response was assessed by BILAG-based Composite Lupus Assessment (BICLA) response at Week 24.[2] Univariate/multivariate analyses were performed, with 22 previously determined potential predictors. Stepwise multivariate analysis included univariate predictors with $p<0.25$, using $p\leq0.10$ as entry/stay criteria. Disease activity at screening was defined using BILAG 2004 item level scores as acute flare (worsening/new symptoms) or persistent (symptoms the same) based on the past 4 weeks compared with the prior 4 weeks; low C3/C4 was defined as below the lower limit of normal at screening. Results were supported by additional biologic clinical trial datasets.

Results Univariate analysis found the following significant predictors of BICLA non-response at Week 24 ($p<0.05$): persistent disease (vs acute flare; $p=0.003$), low C3 and/or C4 (vs normal; $p=0.007$), low C4 (vs normal; $p=0.007$), low C3 (vs normal; $p=0.042$), and SLEDAI-2K score ≥10 (vs <10; $p=0.044$) (figure 1). In the multivariate stepwise analysis, significant predictors of BICLA non-response (odds ratio [OR]; [95% confidence interval]; $p<0.05$) were persistent disease (vs acute flare; 0.047 [0.004, 0.491]; $p=0.011$) and low C4 (vs normal; 0.094 [0.010, 0.857]; $p=0.036$).

To explore the impact of identified predictors, subgroup analyses showed a higher proportion of patients with acute flare without low C3/C4 ($n=10$) achieved BICLA response at Week 24 vs patients with acute flare and low C3/C4 or persistent disease activity ($n=33$) (80.0% vs 24.2%; $p=0.005$ for OR vs acute flare without low C3/C4).

Conclusions Acute flare with normal complement predicted high placebo response in this analysis. Recent medical history should be considered when defining SLE study populations.

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LP-199 MEDICATIONS GIVEN TO PATIENTS WITH INCOMPLETE SYSTEMIC LUPUS ERYTHEMATOSUS

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Background Often, patients present with clinical symptoms and immunologic abnormalities suggestive of systemic lupus erythematosus(SLE), while not meeting classification criteria yet. This is referred to as incomplete SLE(iSLE). Timely treatment, however, is important to limit disease progression, and prevent organ damage and mortality. The aim of the study was to evaluate the therapy administered to patients diagnosed with iSLE.

Methods iSLE($n=60$) was defined by rheumatologists as clinical diagnosis, not fulfilling ACR or SLICCcriteria and had no classification or specific symptoms of other rheumatic diseases. The majority of the iSLE patients were female (97%), aged 38[26–47]years. The median age of iSLE diagnosis was 33[25–42]years, disease duration was 12[2–39]months, 12 (20%)pts had a disease duration of ≥5 years. The median SLEDAI-2K was 2[1–5] score, SDI – 0[0–0] score.

Results A large proportion of iSLE patients (55%) were prescribed hydroxychloroquine at a dose of 200 mg/day and oral corticosteroids (42%), the maximum dose of prednisolone was 15[6–40]mg/day. Five (8%) patients with iSLE were taking immunosuppressants: sulfasalazine-2(4%), methotrexate-1(2%), azathioprine-1(2%), and cyclophosphamide -1(2%). Six (10%) iSLE patients were taking biology: rituximab -1(2%), IL-6 inhibitor – 1(2%), intravenous human immunoglobulin – 4 (7%). Other medicines: NSAIDs – 28%, vitamin D – 27%, course of antibiotics – 18%, low dose aspirin – 17%, anticoagulants – 10%, antipsychotics – 5%, eltrombopag and antihistamines – 2% each of patients.

Conclusions Although iSLE is sometimes considered a mild form of lupus, the clinical manifestations of iSLE can be significant. This may explain why many iSLE patients are treated with immunomodulatory medications.

LP-212 SLE OVERLAP WITH PSORIATIC ARTHRITIS SUCCESSFULLY TREATED WITH SECUKINUMAB

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Description Background: Psoriasis is a chronic disease of the skin that often affects joints and IL17 has a pathogenetic role. It has been reported that interleukin-17 (IL-17) and Th17 cells play important roles in the pathogenesis of SLE: amplifies the immune response by inducing the local production of chemokines and cytokines, recruiting neutrophils and monocytes, augmenting the production of autoantibodies, and aggravating the inflammation. Secukinumab is an anti-IL17 monoclonal antibody with approval for use in AS and Psoriatic arthritis. There are few reports on interleukin-17-targeted therapy in SLE. Objectives: We report a case SLE overlapped with psoriatic arthritis, successfully treated with Secukinumab.

Methods 32 y.o.man diagnosed with psoriasis on 2016, started having arthralgia on 2018 simultaneously with a new facial rash. Skin biopsy revealed lupus erythematosus tumidus. Gradually he developed fatigue, photosensitivity, low back pain and arthritis of hand, feet hip joints. Laboratory profile: ANA 1/160 homogeneous, antidsDNA neg, C3, C4 normal, lupus anticoagulant positive, all the other autoantibodies negative.

X-rays showed MCP MTP hand DIP stenosis, and sacroiliitis. PASI score 3

First he was treated with Plaquenil and Medrol with improvement in lupus rash and failure both in arthritis and psoriasis. We then added methotrexate and subsequently cyclosporine but all combinations failed to control arthritis.

Results After one year of treatment failures we offered him a combination of Plaquenil, sc Methotrexate and Secukinumab 300 mg/4 weeks. After six months with Secukinumab treatment SLE is inactive and psoriatic arthritis has low disease activity. PASI score 1. No adverse events were observed in terms of SLE.

Conclusions Although recent studies have begun to shed light on the role of IL-17 in the pathogenesis of SLE, there is no experience in real world yet. This case report adds on to our experience on results and safety of Secukinumab in Lupus.