Results: In TULIP-2 (anifrolumab, n=180; placebo, n=182) and TULIP-1 (anifrolumab, n=180; placebo, n=184), fewer patients experienced ≥1 BILAG-2004 flare in the anifrolumab groups (TULIP-2: 31.1%, n=56; TULIP-1: 36.1%, n=65) compared with the placebo groups (TULIP-2: 42.3%, n=77; TULIP-1: 43.5%, n=80; Figure 1). Results favoring anifrolumab were observed in time to first flare (TULIP-2: hazard ratio [HR] 0.65, 95% confidence interval [CI] 0.46–0.91 and TULIP-1: HR 0.76, 95% CI 0.55–1.06; Figure 2) and BILAG-based annualized flare rates (TULIP-2: adjusted rate ratio 0.67, 95% CI 0.48–0.94 and TULIP-1: rate ratio 0.83, 95% CI 0.60–1.14) across both trials.

Conclusion: Across 2 phase 3 trials, we observed reductions in the total number of flares and annualized flare rates, as well as prolongation of time to first flare with anifrolumab treatment compared with placebo. These results support the potential of anifrolumab to reduce disease activity and reduce flares, benefiting patients with SLE.

References:


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SAT0175 IMPACT OF ANTIMALARIAL ADHERENCE ON MORTALITY AMONG PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: A POPULATION-BASED COHORT STUDY

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Background: Evidence has consistently shown that adherence to AM is poor in systemic lupus erythematosus (SLE) patients. However, data on the impact of adherence to AM on mortality is scarce.

Objectives: To assess the effect of AM adherence on all-cause mortality in SLE patients from the general population.

Methods: This study used administrative databases from British Columbia, Canada. We created an incident SLE cohort between January 01, 1997, and March 31, 2015, using the physician billing data and a 7-year washout period. The inclusion criteria were at least two physician visits, at least two months apart, within two years, with an ICD-9 code (710.0) or ICD-10 code (M32.1, M32.8, M32.9) for SLE. Follow-up started at the first day of having both SLE and AM, i.e., at the SLE index date (second ICD code) for those whose first AM use occurred before the SLE index date, or the date of the first AM use if otherwise. Our outcome was all-cause mortality, obtained from the vital statistics registry. In the analysis, the follow-up time was divided into 30-days windows, for a total of 293,190 person-months. For each window, a measure of adherence, the proportion of days covered (PDC), was calculated and categorized as adherent (PDC≥0.90), non-adherent (0<PDC<0.90), and discontinuer (no drug or PDC = 0). We used both Cox’s proportional hazards models and marginal structural models (MSM) to estimate the effect of AM adherence on all-cause mortality. Both analysis controlled for base- line demographics (age, sex, residence, income quintile), as well as the following baseline and time-varying covariates: immunosuppressive and other medications, hospitalizations, impatient, and other visits, and Charlson comorbidity index. To account for the possibility of a few time-varying covariates being mediators in the causal pathway from AM adherence to mortality, which may cause the Cox model to yield biased estimates of the adherence effects, we conducted the MSM analysis that can produce valid estimates as it balances the distributions of time-varying confounders among the three adherence groups via inverse probability weighting.

Results: We identified 3,385 individuals with incident SLE (mean age 47.3 years, 89% were women) who had at least one filled AM prescription. Over the mean follow-up of 6.86 years, 288 (8.5%) incident SLE patients died. The incidence rate (IR) of mortality for AM adherent, non-adherent, and discontinuer patients were 4.31, 11.86, and 19.51 per 1000 person-years, respectively. Using the Cox model, the adjusted hazard ratio (HRs) obtained for AM adherent and non-adherent SLE patients were 0.20 and 0.66, respectively, compared to discontinuer SLE patients (Table 1). Using MSM, those adjusted HRs were found as 0.18 and 0.64. Also, the adjusted hazard ratio (HRs) obtained for AM adherent and non-adherent SLE patients were 0.20 and 0.66, respectively, compared to discontinuer SLE patients (Table 1). Using MSM, those adjusted HRs were found as 0.18 and 0.64. Also, the adjusted hazard ratio (HRs) obtained for AM adherent and non-adherent SLE patients were 0.20 and 0.66, respectively, compared to discontinuer SLE patients (Table 1). Using MSM, those adjusted HRs were found as 0.18 and 0.64.

Conclusion: SLE patients that adhere to AM therapy have a lower risk of death than patients who do not adhere or who discontinue AM (5 and 3 times, respectively) in both the MSM and Cox analysis. Our findings support the importance of AM adherence to prevent premature deaths in SLE patients.

Disclosure of Interests: None declared

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SAT0176 THERAPEUTIC APERHESIS DURING PREGNANCY IN RHEUMATIC DISEASES

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Methods:
Between January 2005 and April 2019, 843 TA procedures were performed during 51 pregnancies in 43 patients: 745 plasma exchange sessions and 98 immunoabsorption sessions. TA was performed in 29 (57%) pregnancies of 21 (48.8%) patients with antiphospholipid antibody syndrome (APS), in 20 (39.2%) pregnancies of 20 (46.5%) patients with congenital heart block (CHB), in 1 (1.9%) pregnancy of 1 (2.3%) patient with systemic sclerosis (SSc) and 1 (1.9%) pregnancy of 1 (2.3%) patient affected by lupus nephritis (SLE).

Results:
During the period considered, apheresis sessions applied to pregnant women were 71% of the total (n = 13,251). The average age at the first treatment was 33 years (range 24-43). The mean management age at the first apheresic treatment was 21 weeks (range 4-32). Twelve (14%) apheresis sessions were complicated by adverse events, none required or prolonged hospitalization. There were four (4.8%) live births, 3 (5.9%) spontaneous abortions and 2 (3.9%) voluntary terminations of pregnancy. Among the pregnancies that followed the first apheresis session, 2 (3.9%) lost to follow-up. The average gestational age at birth was 35 weeks (range 24-37) and cesarean section was performed in 41 (80.4%) cases. TA was added to conventional therapy in 24/29 (82.7%) patients with APS, to the detection of fetal cardiac activity, while in 5/26 (17.3%) it was introduced when the first signs of pregnancy complications such as mild preclampsia, HELLP and IUGR were detected. TA was started within 24 hours of atroventricular block (AVB) detection; 10/20 (50%) mothers with CHB were diagnosed with 2nd degree AVB, 9/20 (45%) with 3rd degree AVB and one (5%) with sinus bradycardia and endocardial fibroelastosis. The patient with SSc was treated with TA twice a week from the 32nd SG until delivery, which occurred at the 36th SG, due to severe IUGR and oligohydramnios. The patient with SLE complicated by lupic nephritis was treated with TA twice a week, from the 26th SG until the birth, which took place at the 31st SG.

Conclusion: Our data have shown that TA in pregnancy is well tolerated. Close collaboration between rheumatologist, obstetrician and specialist in TA is essential to ensure a successful outcome of high-risk pregnancies.

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SAT0177 MULTITARGET THERAPY WITH TACROLIMUS AND MYCOPHENOLATE MOFETIL FOR TREATMENT OF LUPUS NEPHRITIS PRESENTED WITH RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS

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Background: Although, most lupus nephritis patients present with chronic glomerulonephritis or nephrotic syndrome, some patients develop rapidly progressive glomerulonephritis (RPGN) which is a clinical syndrome characterized by rapid loss of renal function over a short period of time (days to months). Multitarget therapy using tacrolimus and mycophenolate mofetil (MMF) has been reported to be effective as induction therapy of Class III to Class V lupus nephritis. However, its efficacy on lupus nephritis presented with RPGN has not been well reported.

Objectives: We aimed to examine the efficacy of multitarget therapy on lupus nephritis presented with RPGN.

Methods: We retrospectively analyzed patients with biopsy-proven lupus nephritis, who clinically showed RPGN, and were treated by multitarget therapy with tacrolimus and MMF in our department. Data were expressed as mean±SD.

Results: Five lupus nephritis patients (3 female) with RPGN were treated by multitarget therapy with tacrolimus and MMF in our department. Data were expressed as mean±SD.

SAT0178 HYDROXYCHLOROQUINE CONTROLS DISEASE ACTIVITY IN SLE AND MULTIMODAL IMAGING TECHNIQUES SHOULD BE USED TO DETECT OCULAR TOXICITY

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Background: Hydroxychloroquine (HCQ) is an immunomodulatory drug that has been shown to improve disease activity in systemic lupus erythematosus (SLE). However, retinal toxicity is an important concern.

Objectives: In this study we sought to evaluate the effect of HCQ on disease activity and damage in patients with SLE in whom HCQ was discontinued due to retinal toxicity and whether it could be restarted by a detailed ophthalmologic examination.

Methods: Patients who met SLE SLICC classification criteria and were on HCQ for at least 3 years after reaching Lupus Low Disease Activity State (LLDAS) following remission induction and were followed up for at least 3 years after termination of HCQ treatment due to retinal toxicity diagnosed with visual field test were analyzed. Disease activity (LLDAS and SLEDAI-2K) and both the number and severity of flares were recorded for each patient whilst on HCQ and after cessation of treatment. All patients were examined by two experienced ophthalmologists and were assessed by computerized visual field, optical coherence tomography (OCT), fundus autofluorescence (FAF) and fundus florescence angiography (FFA) to further analyze toxicity.

Results: Out of 88 patients with recorded HCQ retinal toxicity in a cohort of 1500 patients with SLE, 64 patients (mean age at diagnosis 33.4 ± 10.5 (10-57); 88.5% female) with complete data and ophthalmologic re-examination results were included in the analyses. The average duration on HCQ was 122 ±85 (39-336) months, and the mean follow-up time was 74.6 ± 48.3 (36-239) months after the drug was discontinued. Comparison of mean disease activity in the 3-year period when patients were on HCQ to 3 years post-cessation revealed a significantly lower mean SLEDAI and SLEDAI-2K from 8.128 vs 12.136, p<0.02. The % of visits maintaining LLDAS was higher during HCQ treatment (89.7 ± 176 vs. 80.1 ± 23.5, p=0.001). There was significantly a higher frequency of flares with a dominance of mild-moderate types after HCQ was ceased (475 vs. 26.2%, p=0.03; 39.3 vs. 22.9%, p=0.024 respectively). There were more patients with serious flares in the post-discontinuation period but without statistical significance (13.1% vs. 4.9% p=0.08). Thirty-seven (60%) of patients restarted the treatment after ophthalmologic examination. Although 38 (62.3%) patients had visual field defects in the latest examination, multimodal imaging with OCT, FAF and FFA revealed that only 19 (31%) patient had typical retinal toxicity. Five patients were found to have macular atrophy due to other causes (4 age related macular degeneration and 1 vitreomacular adhesion). Since the discrimination of macular pathology would not be possible with imaging in these patients, HCQ was not prescribed. Comparison of patients with and without retinal toxicity showed that duration of HCQ use and HCQ-free time was not significantly different between patients.

Conclusion: HCQ is effective in controlling disease activity in patients with SLE and an opportunity for re-medication with HCQ is valuable. More than half of the