

and 0.69 per 1000 person-years, respectively. Patients with SLE had a higher risk of HP ($p < 0.001$, figure 1). The age- and sex-adjusted hazard ratio (HR) was 2.04 (95%CI;1.53–2.73). After adjusting for age, sex and baseline covariates, the HR was 1.85 (95% CI;1.37–2.52). After adjusting for age, sex, baseline covariates, and weighted time-dependent covariates, the HR was 1.63 (95% CI;1.07–2.50).

Conclusions Patients with a new diagnosis of SLE have 1.6 fold increased risk of HP than the general population. Given the impact of HP, this has important implications for mortality, functional status, and quality of life of people with SLE.

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77 SULFAMETHOXAZOLE AND TRIMETHOPRIM CAUSES TRUE LUPUS EXACERBATIONS RATHER THAN DRUG REACTION

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Background Sulfamethoxazole and trimethoprim (TMP-SMX) is frequently used for urinary tract infections and Pneumocystis prophylaxis in patients on high dose systemic steroids or cyclophosphamide. Recommendations on avoiding TMP-SMX in systemic lupus erythematosus (SLE) are based on anecdotal evidence. Many authors describe adverse effects of TMP-SMX to be a drug reaction or allergy rather than a true SLE exacerbation.

Methods We performed chart review in an urban community clinic setting from 2013 to 2018.

Results Three patients were identified as having a lupus exacerbation within one week of exposure to TMP-SMX, and one patient within two months. Exacerbations consisted of fever and arthralgia, lupus enteritis, lupus enteritis with pericarditis, and inflammatory arthritis. Three cases occurred in the summer (two in June and one in September) and one case in the winter (December). All patients required hospitalization. Two of four patients had stable SLE prior to exacerbation. Symptoms in all patients resolved after treatment with high dose systemic glucocorticoids. There were no recurrent manifestations after TMP-SMX was stopped. All patients continued baseline medications and did not need additional long-term immunosuppression.

Conclusions TMP-SMX can cause severe exacerbations of SLE and should be avoided in these patients. To the best of our knowledge, this is the first report of two instances of TMP-

SMX induced lupus enteritis. Serologic associations may identify those with greater risk, as a positive RNP, Smith and chromatin antibodies were found in three patients and SSA was positive in only one patient. Increased photosensitivity secondary to TMP-SMX may lead to exacerbation, as three cases occurred during summer months. More studies are needed to clarify guidelines for TMP-SMX use in patients with SLE and promote awareness of exacerbation risk within the primary care community.

Funding Source(s): None

78 DISCOVERY OF A NOVEL ANTI-TACI ANTIBODY WITH SLE TREATMENT EFFECT AND LIMITED SIDE-EFFECT

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Background All clinical trials involving the BAFF axis to date have targeted the ligand, BAFF. Remarkably, there have been no reports of targeting any of the BAFF receptors, BCMA, TACI, or BR3. TACI is critical for T cell-independent type antibody production and memory B cell survival. Also IgG autoantibody production in SLE patient is mainly a T cell-independent. Thus, TACI is a promising target for SLE treatment.

Methods In this study, we discovered a novel anti-TACI antibody, GenSci-X002, by hybridoma and humanization approach to investigate its function on SLE treatment.

Results GenSci-X002 specifically binds to TACI receptor with sub-nanomolar affinity and blocks the binding of TACI and BAFF ligand. Also, GenSci-X002 neutralized BAFF-TACI downstream signal in a luciferase reporter assay. In SLE mouse model, GenSci-X002 exhibited equivalent SLE treatment effects with Belimumab, evidenced by the significant decrease of pathogenic autoantibody production, kidney Ig deposition and the development of nephritis. Remarkably, GenSci-X002 didn't alter normal B cell subsets and B cell numbers with minimal side effect.

Conclusions Here, we show that anti-TACI antibody protected against BAFF-mediated autoimmune manifestations while preserving B cells, suggesting that loss of BAFF signaling through TACI rather than loss of B cells may underpin the effect of Belimumab in the clinic. Therefore, B cell blockade of TACI by GenSci-X002 may offer a more specific and safer therapeutic alternative to broad B cell depletion in SLE. The candidate therapeutics, GenSci-X002, will be further studied in preclinical evaluation to enter early-stage clinical trials of SLE in future.

Funding Source(s): None

79 CLINICAL CHARACTERISTICS OF PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS COMPLICATED WITH PULMONARY THROMBOEMBOLISM

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Background There is strong evidence for an association between SLE and an increased risk of pulmonary thromboembolism (PTE). PTE occurs with a higher frequency in SLE patients compared to the general population.

Abstract 77 Table 1

Demographics	Serology/APS	Baseline Medications	Reaction to TMP-SMX
47 F	Chromatin, Smith, RNP	Hydroxychloroquine	Fever, Arthralgia
37 F	Chromatin, Smith, RNP	Belimumab	Lupus Enteritis
56 M	Chromatin, Cardiolipin IgM, Beta 2 glycoprotein 1 IgM	Hydroxychloroquine, Belimumab	Inflammatory Arthritis
42 F	Chromatin, Smith, RNP, dsDNA, Ribosomal P, SSA	Hydroxychloroquine, Prednisone	Lupus Enteritis, Ascites, Pericarditis

Demographics, serologies, and manifestations of patients with lupus exacerbation

Methods A retrospective analysis of 29 SLE patients with PTE admitted to our hospital from January 2009 to August 2018 was conducted.

Results Among the 29 patients, 26 (89.66%) were female and 3 (10.34%) were male. The age ranged from 23 to 66 years. The SLEDAI scores of 29 cases ranged from 0 to 18, with scores 15 points in 1 case (3.45%), 10–14 points in 9 cases (31.03%), 5–9 points in 11 cases (37.93%), 0–4 points in 8 cases (27.59%). Among the 29 patients, 13 patients (44.83%) were admitted to the hospital with chest pain or dyspnea as the first symptom. Only one case (3.45%) was admitted to the hospital with hemoptysis as the first symptom. Of the 29 patients, 21 (72.41%) had chest pain or difficulty breathing during the course of the disease. Among the 29 patients, 13 cases (44.83%) had SLE at the initial diagnosis, and the remaining 16 cases (55.17%) had SLE duration ranging from 1 month to 20 years, 3 (18.75%) in one year and 7 (43.75%) in 1 to 10 years. The course of disease was more than 10 years in 6 cases (37.50%). In this group of patients, 1 case (3.45%) with cerebral infarction, 2 cases (6.90%) with renal vein thromboembolism, and 14 cases (48.28%) with lower extremity venous thrombosis. Of the 29 patients, 3 were normal D-D dimers (normal value 0–0.55 mg/L), and the remaining 26 (89.66%) were elevated, with an average of 4.50 mg/L. All patients underwent echocardiography, 14 of whom (48.28%) indicated pulmonary artery widening. Of the 29 patients, 3 patients did not receive anticardiolipin antibodies, and of the remaining 26, 13 were positive and 13 were negative. In terms of prognosis, 3 of 10 patients (10.34%) died.

Conclusions SLE combined with PTE is easily missed and misdiagnosed. The clinical manifestations are not typical. In the active stage of SLE, or patients have chest pain and dyspnea symptoms, or test indicates that D-D dimer is elevated, or echocardiography indicates pulmonary artery widening, clinicians should think that it might be SLE merge PTE. For patients with anticardiolipin antibody negative, systemic lupus erythematosus should not be relaxed. During the treatment of patients with SLE combined with PTE, they should be alert to the occurrence of thrombosis in other sites.

Funding Source(s): NO

Methods Retrospective study including 9 women diagnosed with primary APS. Clinical records were reviewed to obtain demographic and clinical data.

Results Two women had a history of thrombosis, while the remaining 7 had merely obstetric manifestations. All had suffered from early pregnancy losses and 1 of them also had a history of fetal death. We studied 42 pregnancies in these women. The mean number of pregnancies was 4.67 ± 0.71 per woman. Maternal age was 35.8 ± 4.6 years. Overall there were 30 abortions (71.4%) and 1 fetal death (2.4%). Regarding treatments used during pregnancy, 25 (59.5%) pregnancies were on some treatment: LDA (25), LMWH (24), corticosteroids (13), and intravenous immunoglobulins (IVIG) (4). As expected, in all pregnancies treated with corticosteroids, these drugs were combined with LDA and LMWH. When analyzing the effect of therapies, we found a tendency to decrease pregnancy loss in pregnancies treated with LDA (64 vs 82.4%; $p=0.3$) and LMWH (62.5 vs 83.3%; $p=0.18$). Treatment with corticosteroids significantly increased the rate of successful pregnancy (the rate of pregnancy loss was 38.5% in treated vs 86.2% in non treated pregnancies; $p=0.003$). The results of the global pregnancy analysis were confirmed by bivariate and multivariate GEE analysis. In the bivariate analysis, LDA tended to be protective (OR=0.38, CI 0.11–1.33; $p=0.129$) and LMWH and corticosteroids significantly protected against pregnancy loss (OR=0.34, CI 0.13–0.85; $p=0.021$; and OR=0.17, CI 0.06–0.51; $p=0.002$, respectively). After multivariate analysis, only corticosteroids remained inversely associated with pregnancy loss (OR=0.09, CI 0.012–0.683; $p=0.020$). However, the independent effects of LDA/LMWH could not be adequately tested because all patients on corticosteroids were also treated with LDA and LMWH. As adverse events, 2 cases of gestational diabetes and 1 of preeclampsia were observed.

Conclusions The addition of corticosteroids to standard therapy with LDA and LMWH seems to be safe and effective for improving the pregnancy outcome in women with obstetric APS.

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80 CORTICOSTEROIDS IMPROVE PREGNANCY OUTCOME IN PATIENTS WITH OBSTETRIC ANTIPHOSPHOLIPID SYNDROME

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Background Recurrent pregnancy losses are the most common obstetric manifestation of antiphospholipid syndrome (APS). Despite standard therapy with low dose aspirin (LDA) and low molecular weight heparin (LMWH), the rate of successful pregnancies is not greater than 70%. Corticosteroids have been suggested as a potential therapy in these patients. Our aim is to describe a cohort of patients with obstetric APS treated with low dose corticosteroids.

81 ANTIPHOSPHOLIPID SYNDROME IN SYSTEMIC LUPUS ERYTHEMATOSUS LEADS TO A MORE SEVERE DISEASE

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Background Antiphospholipid antibodies (aPL) have been associated with organ damage and certain features in systemic lupus erythematosus (SLE) patients. Our aim was to