

Results The study group included 295 patients, 125 patients (42%) with SLE, 50 patients (17%) with SSc, 80 patients (27%) with Sjogren's, 40 patients (14%) with UCTD. A total of 465 pregnancies were registered. The maternal and fetal outcomes are detailed in table 1 and figure 1. The mean age at delivery was 31.5 ± 8.5 years and the mean duration of disease was 7.2 ± 5.6 years. Treatment with HCQ was received in 115 pregnancies in SLE (59%), 21 pregnancies in SSc (24%) 62 pregnancies in pSS (52%) and 32 pregnancies in UCTD (49%). Exposure to corticosteroids and biologics during pregnancy was 23 (18.4%), 6 (12%), 15 (19%) and 3 (7.5%), respectively. Patients with SLE had a higher risk of fetal morbidity, including abortion ($p=0.03$), mean abortion rate ($p=0.03$), preeclampsia ($p=0.04$), ectopic pregnancy ($p=0.03$), preterm delivery ($p=0.02$) and postpartum haemorrhage ($p=0.01$) than patients without SLE. The multivariate model adjusted for age, nulliparity, active disease activity during pregnancy, smoking and exposure to biologics, HCQ and corticosteroids found an association between unfavourable pregnancy outcomes and disease activity (OR 2.4 95% CI (1.3–7.2), $p=0.003$), whilst HCQ during pregnancy (OR 0.23 95% CI (0.03–0.82)) had a protective effect.

Abstract 144 Table 1 Fetal and maternal morbidity outcomes in SLE, SSc, pSS and UCTD

	SLE	SSc	pSS	UCTD	P value
Total of pregnancies	192	88	120	65	
Age at pregnancy	32.4 ± 4.5	29.5 ± 7.2	30.4 ± 3.5	33.5 ± 2.7	0.45
Smokers	32 (26%)	17 (34%)	25 (31%)	12 (30%)	0.27
Birth	103 (67%)	68 (77%)	90 (75%)	47 (72%)	0.28
Abortion	57 (37%)	12 (14%)	18 (15%)	10 (15%)	0.03
Mean abortion number	2.7 ± 0.7	1.1 ± 0.6	2.4 ± 0.3	0.9 ± 0.5	0.03
Preeclampsia	15 (9.8%)	3 (3%)	2 (2%)	2 (3%)	0.04
Ectopic pregnancy	10 (6.5%)	0	1 (1%)	1 (2%)	0.03
Placental abnormalities	8 (5%)	5 (5.7%)	2 (2%)	3 (5%)	0.21
Premature rupture of membranes (PROM)	5 (3.3%)	0	2 (2%)	1 (2%)	0.24
Preterm delivery	20 (13%)	0	8 (6%)	5 (8%)	0.02
Postpartum haemorrhage	21 (14%)	0	4 (4%)	1 (2%)	0.01

Conclusions 66% of pregnancies in patients with autoimmune diseases resulted in live births. Patients with SLE had higher rates of fetal and maternal morbidity than SSc, pSS and UCTD. Disease activity was associated with unfavourable pregnancy outcomes. Exposure to HCQ had a protective effect during pregnancy. Pregnancy planning and counselling prior to conception of patients with connective tissue diseases leads to a reduction in maternal and perinatal complications.

Acknowledgements None.

P145

CIRCULATING INTERFERON- α LEVELS ARE ELEVATED DURING PREGNANCY IN WOMEN WITH SLE WHO DELIVER INFANTS THAT ARE SMALL FOR GESTATIONAL AGE, PRETERM AND/OR HAVE LOW BIRTH WEIGHT

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10.1136/lupus-2024-el.199

Objectives Adverse pregnancy outcomes (APOs) are more common among women with SLE compared to the general population and the underlying immunopathological mechanisms are largely unknown. The type I interferon (IFN) signature persists in complicated SLE pregnancies, while it is downregulated in uncomplicated SLE pregnancies. Moreover, IFN α protein concentrations are higher in SLE compared to healthy pregnancies, but whether IFN α protein levels are associated with APOs in SLE is unknown. The aim of this study was to evaluate whether APOs are more common in Swedish women with SLE compared to healthy controls, and if this associates with circulating IFN α protein or autoantibodies.

Methods We included 83 births from 77 women with SLE and 58 births from 58 healthy controls (HC). Repeated peripheral blood samples were collected and IFN α protein levels were quantified with Simoa. Anti-nuclear antibody (ANA) specificities and anti-phospholipid antibodies (aPL) during pregnancy was analysed using multiplexed bead technology. APOs were defined as an infant small for gestational age (SGA), preterm birth, low birth weight (LBW) and/or preeclampsia. Multivariate orthogonal partial least squares analysis (OPLS) was used to examine SGA, LBW and/or preterm (combined outcomes, Y-variable) in relation to mean IFN α protein level, IFN α positivity, ANA specificity and aPL positivity during pregnancy.

Results APOs were more common in women with SLE compared to healthy women (33% compared to 12%, $p=0.005$). The most common outcome was SGA, which was present in

17% of women with SLE compared to 3% of HC ($p=0.01$). In OPLS, SGA, LWB and/or preterm birth was most positively associated to mean IFN α protein level and IFN α positivity in plasma during pregnancy. Preeclampsia was unrelated to IFN α and autoantibody positivity in women with SLE. In univariate analysis, the mean IFN α protein level was significantly higher in women with SLE who had an infant who was SGA, LWB and/or preterm compared to women without these APOs.

Conclusion IFN α protein level in plasma is a potential risk factor for giving birth to an infant who is small for gestational age, has low birth weight and/or is delivered preterm in SLE.

P146

LOW CD4+ T CELL COUNT IS RELATED TO SPECIFIC ANTI-NUCLEAR ANTIBODIES, IFN α PROTEIN POSITIVITY AND DISEASE ACTIVITY IN SYSTEMIC LUPUS ERYTHEMATOSUS PREGNANCY

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10.1136/lupus-2024-el.200

Objective Adverse pregnancy outcomes are more common among women with systemic lupus erythematosus (SLE) compared to healthy women, but there is limited understanding on how pregnancy affects the immune system and what specific immunopathological processes that precede these complications. Lymphopenia, autoantibodies and activation of the type I interferon (IFN) system are common features SLE. We aimed to investigate the impact of pregnancy on lymphocyte subset counts in SLE and their associations with autoantibody profiles and IFN α concentrations.

Methods Repeated blood samples were collected from 80 pregnant women with SLE and 51 healthy controls (HC), with additional samples from 19 women with SLE postpartum. Flow cytometry was used to measure CD4+ and CD8 + T cells, B cells, and NK cells. Positivity for anti-nuclear antibodies (ANA) fine specificities and anti-phospholipid antibodies was assessed using multiplexed bead assay. IFN α concentration was quantified with Single molecule array (Simoa) immune assay. Clinical data were retrieved from medical records.

Results Women with SLE had lower counts of all lymphocyte subsets compared to HC throughout pregnancy, but counts did not differ during pregnancy compared to postpartum. Principle component analysis revealed that low lymphocyte subset counts differentially related to autoantibody profiles, cluster one (anti-dsDNA/anti-Sm/anti-RNP/anti-Sm/RNP/anti-chormatin), cluster two (anti-SSA/anti-SSB) and cluster three (anti-CL/anti- β 2GPI), IFN α protein levels and disease activity. CD4+ T cell counts were lower in women positive to all ANA fine specificities in cluster one compared to those who were negative, and B cell numbers were lower in women positive for anti-dsDNA and anti-Sm compared to negative women. Moreover, CD4+ T cell and B cell counts were lower in women with moderate/high compared to no/low disease activity, and CD4+ T cell count was lower in IFN α protein positive relative to negative women. Finally, CD4+ T cell count was unrelated to treatment.

Conclusion Lymphocyte subset counts are lower in SLE compared to healthy pregnancies, which seems to be a feature of the disease *per se* and not affected by pregnancy. Our results also indicate that low lymphocyte subset counts relate differentially to autoantibody profiles, IFN α protein levels and disease activity, which could be due to divergent disease pathways.