2014 to Sep 2019. Patients complicated with pulmonary artery hypertension, deep venous thrombosis or pulmonary embolism and underwent cardiac surgery during the follow-up period were excluded. Left ventricular(LV) systolic dysfunction was defined as ejection fraction (EF) < 50%. LV diastolic dysfunction was defined according to ASE/EACVI guideline⁴. LV dysfunction (LVD) includes one or both of LV systolic dysfunction and LV diastolic function. Monocyte to HDL ratio (MHR) was calculated by dividing monocyte count with HDL-C level.

Prevalence of left ventricular abnormalities was analysed at baseline and follow-up examination. Clinical characteristics and laboratory data were compared among patient groups as follows; patients with LV dysfunction (Group A) and without LV dysfunction (Group B) at the follow-up echocardiography, patients with LV asynergy at any point of examination (Group C) and patients free of LV abnormalities during the follow-up period (Group D).

Results: At the baseline examination, LV dysfunction (5/29 cases, 13.8%), LV asynergy (6/29 cases, 21.7%) were detected. Pericarditis was detected in 7 patients (24.1%, LVD in 3 patients, LV asynergy in 2 patients) and 2 of them with subacute onset had progressive LV dysfunction, while 5 patients were normal in echocardiography after remission induction therapy for SLE. At the follow-up examination, LV dysfunction (9/29 cases, 31.0%, 5 new-onset and 1 improved case), LV asynergy (6/29 cases, 21.7%, 2 new-onset and 2 improved cases) were detected. Though any significant differences were observed between Group A and Group B at the baseline, platelet count (156.0 vs 207.0, $p=0.049$) were significantly lower in LV dysfunction group (Group A) at the follow-up examination. Group C patients had significantly higher uric acid ($p=0.004$), monocyte count ($p=0.009$), and MHR ($p=0.003$) than Group D(results in table).

Conclusion: LV dysfunction is progressive in most of patients and requires regular follow-up once they developed. Uric acid, monocyte count and MHR are elevated in SLE patients with LV asynergy. Since MHR elevation was reported as useful marker of endothelial dysfunction⁵, our future goal is to analyse involvement of monocyte activation and endothelial dysfunction in LV asynergy of SLE patients.

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	Group C(LV asynergy +, n=8)	Group D(no abnormality, n=10)	P
Age, (y)	63.0(51.3-71.0)	49.0(45.8-61.8)	ns
SLE duration, (y)	22.9(13.4-33.9)	7.0(4.9-25.6)	ns
SLEDAI	1.0(0.0-2.0)	2.0(0.0-6.3)	ns
White blood cell count, ($\times 10^9/L$)	7.0(5.4-11.2)	6.1(4.7-7.9)	ns
Lymphocyte count, ($\times 10^9/L$)	1.2(0.88-2.1)	0.89(0.67-1.2)	ns
Monocyte count, ($\times 10^9/L$)	0.48(0.29-0.57)	0.24(0.17-0.29)	0.009
Hb, (g/dL)	11.7(10.5-14.2)	13.1(12.3-14.2)	ns
Platelet, ($\times 10^9/L$)	195.0(157.3-270.8)	225.0(183.3-284.8)	ns
Uric acid, (mg/dL)	7.2(5.8-8.1)	4.5(3.8-6.0)	0.004
IgG, (mg/dL)	1201(807.0-1347)	1260(1034-1786)	ns
Anti-DNA antibodies, (mg/dL)	0.0(0.0-7.3)	3.0(0.0-26.0)	ns
CH ₂ O, (mg/dL)	42.4(38.6-44.4)	39.7(28.9-46.9)	ns
Triglyceride, (mg/dL)	127.0(80.0-156.0)	109.0(75.5-155.0)	ns
HDL-Cholesterol, (mg/dL)	53.0(5.1-25-62.0)	54.5(42.8-82.7)	ns
LDL-Cholesterol, (mg/dL)	102.0(89.0-103.0)	118.5(82.8-134.3)	ns
MHR	0.0087(0.0063-0.012)	0.0039(0.0026-0.0051)	0.003

Numbers are median (interquartile range), Mann-Whitney u test were performed, p value less than 0.05 was considered statistically significant.

Disclosure of Interests: : None declared

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AB0423

NEUROPSYCHIATRIC OUTCOME OF CHILDREN BORN TO WOMEN WITH SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) WOMEN AND EXPOSED IN UTERO TO AZATHIOPRINE: A CASE-CONTROL STUDY.

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Background: A possible increase in neurodevelopmental (ND) and learning disorders (LD) in the offspring of mothers affected by SLE have been suggested in some studies, along with the identification of different possible risk factors. Azathioprine (AZA) is commonly used during pregnancy, based on its non-teratogenicity and extended experience in women with different diseases. However, a few small studies suggested an association between in utero exposure to AZA and possible increased frequency of ND/LD in children, indirectly derived from increased request of supportive educational services.

Objectives: To evaluate the medium-long term outcome in terms of ND/LD in children of school age (≥ 6 years) born to SLE women treated with AZA during pregnancy, as compared to that of children born to SLE mothers not treated with AZA during pregnancy.

Methods: Data from our Pregnancy Clinic registry were collected for prospectively followed pregnancies of SLE women treated with AZA (cases) and compared to pregnancies of SLE women not treated with AZA (controls), that were matched for age at pregnancy, presence of renal involvement and aPL positivity. SLE patients (cases and controls) were interviewed by phone to collect data about their children, focusing on the presence of ND/LD certified by Neuropsychiatrists.

Results: Data were collected for 14 SLE mothers in the AZA group and 31 in the control group, with similar age at pregnancy (30.3 ± 5.21 vs 31.4 ± 4.70 years, $p:0.45$) and frequency of renal involvement (50.0% vs 44.1% , $p:0.77$), aPL positivity (33.3% vs 29.4% , $p:0.76$) and anti-Ro/SSA positivity (27.8% vs 26.5% , $p:0.55$). A SLE flare during pregnancy was more frequently recorded in the AZA group (27.8% vs 2.94% , $p:0.02$). Other medications included HCQ (55.6% vs 70.6% , $p:0.36$) and corticosteroids (100% vs 79.4% , $p:0.08$).

We collected data for 18 children in the AZA group and 34 children in the control group, that had a similar mean age at the time of the interview (12.7 ± 4.80 vs 12.9 ± 5.61 years, $p:0.91$). The two groups had also similar gestational age (37.4 ± 2.20 weeks vs 38.0 ± 1.29 weeks, $p:0.23$), birth weight (3003 ± 433 g vs 3011 ± 453 g, $p:0.95$) and rate of male sex (61.1% vs 44.1% , $p:0.38$).

We recorded similar frequency of ND/LD in the two groups. In particular, a ND was present in 2/18 (11.1%) of children exposed to AZA vs. 2/34 (5.88%) in the control group ($p:0.60$). A LD was present in 1/18 cases (5.56%) and 6/34 controls (17.6%) ($p:0.40$).

Conclusion: The medium-long term outcome of children born to SLE mothers in the whole cohort was characterized by the presence of ND in 4/54 (7.69%) and LD in 7/52 (13.5%). ND/LD do not seem to be related to in utero exposure to AZA.

Disclosure of Interests: None declared

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AB0424

PREDICTORS OF SLE FLARE-UP AND PREMATURE DELIVERY IN PREGNANCY

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Background: Systemic lupus erythematosus (SLE) is a life-threatening autoimmune disease that affects many women of child-bearing age, with potentially severe consequences on pregnancy outcome. SLE flare-ups may occur during pregnancy and the post-partum period. Previous studies documenting the incidence and risk factors of flare-up of SLE during pregnancy and puerperium had partially discordant results.

Objectives: We aimed to delineate the pregnancy complications of women with SLE, as well as neonatal outcomes of their offspring, and hoped to clarify the incidence and risk factors of SLE flare-ups during pregnancy and puerperium.

Methods: We retrospectively reviewed the medical records of SLE patients with previous records of pregnancies in our institution. Flare events during pregnancy and puerperium were documented. The pregnancy outcomes recorded include live births, intra-uterine fetal death (IUFD), premature delivery (< 36 weeks of gestational age), NICU admission, and small for gestational age (SGA, <10th percentile). Univariate logistic regression was performed to determine the factors associated with disease relapse and pregnancy outcomes.

Results: From January, 2000 to December, 2019, a total of 94 SLE patients with 139 pregnancies were identified. The overall live birth rate was 92.4% (134/145). Forty-six (34.3%) of the neonates were delivered prematurely. Forty-six (34.3%) of them were SGA. The admission rate to the neonatal intensive care unit was 25% (30/120). Nine (6.4%) were diagnosed to have SLE during pregnancy. The flare rate during pregnancy was 20% while post-partum 9.4%. The majority of the relapses during pregnancy occurred in the second trimester (46.2%), followed by the first trimester (30.8%), and the third trimester (23.1%). Low complement C3 (C3 < 80mg/dl), thrombocytopenia (PLT < $100 \times 10^3/uL$) at conception, and low serum albumin level at the first trimester were associated with antepartum flare. Presence of disease flare and pre-eclampsia in pregnancy, and low serum albumin level at conception were significantly associated with premature delivery.

Conclusion: Low complement C3 and thrombocytopenia at conception, and low serum albumin level at the first trimester were associated with disease flare-up during pregnancy. Patients with relative low serum albumin level at conception, or presence of eclampsia or disease flare-up during pregnancy had a higher risk of premature delivery.

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