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Placental Abnormalities in Systemic Lupus Erythematosus (SLE): Novel Markers for Adverse Pregnancy Outcomes.

Placenta-mediated adverse pregnancy outcomes (APO) are a huge concern in SLE. Recent efforts to understand APO include the establishment of the 2016 Amsterdam classification criteria, developed to standardize placental pathology evaluation. No study to date evaluated the Amsterdam criteria in SLE. Using the prospective “Lupus prEGnAnCY (LEGACY)” biobank, we assessed the relationship between placental abnormalities, lupus anticoagulant (LAC), and APO, applying the Amsterdam criteria.

LEGACY is a prospective cohort enrolling SLE pregnancies (before the 17th gestational week). Relevant information is collected at each trimester and/or end-of-pregnancy visits. We evaluated pregnancies delivered beyond 17 weeks at the Montreal site. Placental pathology was defined as abnormal if fulfilling at least one of the 4 main Amsterdam classification subtypes: 1) maternal vascular malperfusion, 2) fetal vascular malperfusion, 3) acute chorioamnionitis, and/or 4) villitis of unknown etiology. Pregnancies with and without abnormal pathology were further characterized based on presence of LAC and APO (i.e. stillbirth, placental insufficiency, gestational hypertension, preeclampsia, small-for-gestational age neonate <5%).

Of 44 LEGACY pregnancies delivered (beyond 17 weeks), 32 (73%) had placental pathology available. Among these 32, 15 (47%) had abnormal pathology. Of those with abnormal pathology, 6/15 (40%) had maternal vascular malperfusion, 5/15 (33%) acute chorioamnionitis, 4/15 (27%) villitis of unknown etiology, and 1/15 (7%) fetal vascular malperfusion. Mean gestational age at delivery was substantially lower in pregnancies with abnormal pathology [mean 33.7 weeks, standard deviation (SD) 6.8] versus those with normal pathology (mean 37.8 weeks, SD 1.7), with a difference in mean gestational age of -4.1 weeks (95% CI -0.6, -7.6). LAC was more frequent in pregnancies with abnormal pathology (4/15; 27%) as opposed to pregnancies with normal pathology (2/17; 12%). APO occurred in 8/15 (53%) pregnancies with abnormal pathology (including 3 with early preterm preeclampsia <34 weeks) as opposed to 7/17 (41%) pregnancies with normal pathology (none with early preterm preeclampsia). Maternal vascular malperfusion was strongly associated with APO (odds ratio 8.1; 95% CI 0.8, 83.7), although the CI included the null.

In this cross-sectional analysis, SLE pregnancies with abnormal placenta pathology, particularly maternal vascular malperfusion, experienced shorter gestation and more severe placenta-mediated APO, including early preterm preeclampsia. Future studies will aim to expand the sample and to investigate if placental abnormalities in one pregnancy helps predict APO in subsequent pregnancies.