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Original Research Article

A retrospective observational study of predictors of adverse maternal and fetal outcomes in mothers with systemic lupus erythematosus in pregnancy

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ABSTRACT

Background: SLE is a disease of reproductive age group and has implications on maternal and fetal health in pregnancy. This study was done to determine and analyze the predictors of adverse maternal and fetal outcomes in mothers with systemic lupus erythematosus in pregnancy.
Methods: Retrospective observational study was done to evaluate pregnancy outcomes in SLE mothers at Fernandez Hospital, Hyderabad, from January 2017 to December 2022. Data was obtained via electronic medical records. Descriptive analysis was done by mean and standard deviation for quantitative variables, and frequency and proportion for categorical variables. Chi-square test was used to test statistical significance between variables ($p<0.05$ was significant).
Results: SLE affects pregnancies causing adverse maternal (flares (18.95%), hypertensive disorders (22.18%), severe maternal morbidity (5.65%), fetal (preterm birth (38.71%), FGR (28.97%), perinatal death (3.17%)), and neonatal (congenital heart block (3.17%), neonatal lupus (3.96%), Binder's facies (5.16%)) outcomes- especially when period of remission was <6 months ($p<0.05$). Anti-Smith, RNP and Scl-70 Ab were significantly associated with Binder's facies and chondrodysplasia. Anti-Ro/La antibodies were associated with congenital heart block and endocardial fibroelastosis. Lupus nephritis was an independent risk factor for adverse outcomes. Chronic hypertension, past history of thrombosis and chronic kidney disease were risk factors for adverse outcomes ($p<0.05$). Heparin, HCQs and Azathioprine could also have a role in improving perinatal outcomes ($p<0.05$).
Conclusions: As SLE can cause adverse maternal, fetal and perinatal outcomes in pregnancy, all mothers with SLE should undergo preconceptional counselling to optimize outcomes.

Keywords: Adverse pregnancy outcomes, Binder's facies, Congenital heart block, SLE

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic, multisystem, inflammatory disease of autoimmune etiology characterized by relapses or flares and remission mainly affecting young women of reproductive age group. The skin, joints and kidneys are most commonly affected.[1] The reported prevalence of SLE in India is 14 to 60 per 1,00,000.[2]

The pathophysiology of SLE is complex and incompletely understood. It involves breakdown in tolerance of T and B cells to self-antigens. It also involves abnormalities in immunological processes of innate and adaptive immunity, mainly involving antinuclear antibodies (ANA). It may have some genetic component supported by studies done on affected twins. Environmental and increased levels of estrogen also play a role in the disease process.[3]

Diagnosis of SLE is done using criteria established by multiple international bodies like the European League Against Rheumatism (EULAR), American College of Rheumatology (ACR) and the Systemic Lupus International Collaborating Clinics (SLICC).[4,5]

Maternal morbidity and mortality are substantially increased in such mothers. SLE with APS is associated with a threefold increased risk of pregnancy loss which is further increased with presence of renal disease and active disease at the time of conception. Preeclampsia occurs in 15% to 35% of pregnant women with SLE. Pregnancy poses a risk for disease flares because of increased levels of estrogen, stress and increased physical demands of pregnancy. Fetal growth restriction (FGR) is common in SLE pregnancies and is associated with an increased risk for preterm birth.[6]

Neonatal lupus erythematosus is a syndrome entailing of congenital heart block, transient cutaneous lupus lesions, thrombocytopenia, hepatic and other systemic manifestations in children born to mothers with SLE with positive anti-Ro/anti-La antibodies.[7]

The anti Ro/La antibodies have been proven to be associated with congenital heart block while the antiphospholipid antibodies have been associated with early onset preeclampsia, fetal growth restriction and recurrent pregnancy losses.[7] The other factors that may affect pregnancy outcomes in mothers having SLE in are immunological drugs used in pregnancy, coexisting morbidities, organ systems involved in SLE and prepregnancy state of the disease.

The purpose of this study was to analyze the various predictors of SLE in pregnancy which might be associated with specific adverse maternal, fetal and neonatal outcomes so that appropriate anticipatory measures can be taken to optimize outcomes.

METHODS

This was a retrospective observational study conducted over a period of 6 years from 1st January 2017 to 31st December 2022 at Fernandez Hospital, Hyderabad- a tertiary perinatal care center in South India. All mothers who had SLE and who birthed in Fernandez Hospital between 1st January 2017 to 31st December 2022 were included in the study. Mothers who lost to follow up were excluded from the study. All participants were recruited through institutional electronic medical records. A waiver of consent was obtained from the institutional ethics committee as it was a retrospective study and none of the participants were directly contacted. All the participants were managed based on standard protocols and confidentiality of data was maintained.

The maternal outcomes analyzed were hypertensive disorders complicating pregnancy, severe maternal morbidity (WHO maternal near miss criteria), SLE flare in pregnancy, thromboembolic events in pregnancy and delivery done for worsening maternal condition.[8] The fetal outcomes analyzed were fetal growth restriction (fetal weight <10th centile), preterm birth, stillbirths, neonatal death, neonatal lupus, congenital anomalies. The predictors of SLE used to assess outcomes were autoimmune antibodies, immunological drugs used in pregnancy, coexisting morbidities, organ systems involved in SLE and prepregnancy state of the disease.

Descriptive analysis was done by mean and standard deviation for quantitative variables, and frequency and proportion for categorical variables. The association between categorical variables and the quantitative outcome was assessed by comparing the mean values. The mean differences along with their 95% confidence interval was presented. The association between explanatory variables and categorical outcomes was assessed by cross-tabulation and chi-square test was used to test statistical significance. P value <0.05 was considered statistically significant. Data was analysed by using CoGuide software, V.1.01 and Microsoft Excel.

RESULTS

A total of 210 mothers with SLE having 248 pregnancies with 252 births including 4 pair of twin pregnancies were included in the study. Five pregnancies (2%) were following assisted reproductive techniques and 2 out of them were twin pregnancies. The mean age of study population was 28.29 years with a standard deviation of 3.79 years with majority of the population between 21 to 30 years (67.74%).

Table 1: Maternal outcomes in study population.

Pregnancy complications	N	%
Pregnancy related HTN	55	22.18
Thromboembolic events	3	1.21
Delivery for worsening maternal condition	76	30.65
Severe maternal morbidity	14	5.65
SLE Flares in pregnancy	47	18.95

Antinuclear antibodies were positive in all the mothers (100%, n=248) as it is one of the hallmarks of SLE. Anti dsDNA was present in 50.4% (n=125) of the mothers indicating active disease or flare during pregnancy. Lupus anticoagulant, anticardiolipin antibodies and beta 2 glycoprotein 1 were detected in 19.76% (n=49), 16.94% (n=42) and 6.85% (n=15) of the population respectively. Anti Smith and anti RNP antibodies were detected in 16.13% (n=40) and 16.53% (n=41) each. Anti Ro and anti La antibodies were positive in 41.13% (n=102) and 20.97% (n=52) of study population respectively. Other less commonly detected autoantibodies (<5% each) were Anti histones, anti-nucleosomes, Scl 70/100, Rib P, anti CCP and anti Jo antibodies.

Table 2: Perinatal outcomes in study population.

Gestational age	N	Livebirth (%)	Stillbirths (%)	Neonatal death (%)	FGR (%)
<28 weeks	3	1 (33.33)	2 (66.67)	1 (33.33)	3 (100)
28-32 weeks	14	14 (100)	0 (0)	0 (0)	8 (57.14)
32-34 weeks	25	25 (100)	0 (0)	0 (0)	11 (44)
34-37 weeks	56	54 (96.42)	2 (3.57)	0 (0)	22 (39.28)
>37 weeks	152	151 (99.34)	1 (0.66)	2 (1.31)	6 (3.94)
Total	252	244 (96.82)	5 (1.98)	3 (1.19)	0 (0)

Majority of the mothers were taking low dose aspirin and HCQs (95.16% (n=236) and 92.74% (n=230) respectively). Of all the mothers, 67.74% (n=168) of the mothers were taking prednisolone, 49.19% (n=122) were taking azathioprine, 24.19% (n=60) were taking heparin/LMWH injections and 8.06% (n=20) were taking tacrolimus. Other drug exposures to our mothers included sulfasalazine, phosphodiesterase-5 inhibitors (PDE-5 I), acitrom (<5% each).

Almost 33% of the mothers had bad obstetric history (Neonatal deaths, stillbirths and recurrent pregnancy losses). Twenty-seven mothers (12.8%) had history of thromboembolic events before pregnancy. Maximum mothers who were affected with SLE had mucocutaneous, musculoskeletal and hematological manifestations [74.19% (n=184), 57.66% (n=143) and 37.50% (n=93) respectively]. A total 78 women had lupus nephritis (31.45%). A total of 166 mothers (67%) had multisystem manifestations of SLE. A total of 47 mothers (18.95%) had flare of SLE during pregnancy. Maximum SLE flares involved renal systems (31.91%). This was followed by hematological (29.78%, n=14) and mucocutaneous flares (23.40%, n=11) respectively. Mean duration of disease prior to conception was 5.18 years with a standard deviation of 4.2 years. Seventy-two mothers had been in remission for less than 6 months before conception and this was significantly associated with the incidence of flares during pregnancy (p value <0.0001, OR=4.68). A total of 37 mothers attended prepregnancy counselling before planning pregnancy which was also significantly associated with reducing the incidence of flares in pregnancy (p value 0.0226).

Among the study population, 55 mothers (22.18%) developed pregnancy related hypertension with varying severity and 3 mothers (1.21%) had thromboembolic events in pregnancy. Seventy-six mothers (30.65%) had to be birthed for maternal medical condition while 5.65% (N=14) mothers had severe maternal morbidity as per the WHO maternal near miss criteria (Table 1).[8]

Of the study population, 38.71% mothers had preterm birth. One baby had neonatal death at 25 weeks of gestation due to complications of prematurity. One baby born at 39 weeks of gestation succumbed due to respiratory depression and right pneumothorax. One baby had spinal muscular atrophy type I at 38 weeks of gestation. Five mothers had intrauterine fetal demise at 25, 26, 34, 35, 37 weeks respectively. All of these babies had fetal growth restriction (Table 2).

Table 3: Congenital anomalies in babies of study population.

Anomalies	N	%
Binder's facies	13	5.16
Chondrodysplasia punctata	3	1.19
Hypospadiasis	3	1.19
Ventricular septal defect	2	0.79
Club foot	2	0.79
Ebstein's anomaly	1	0.40
Tracheosophageal fistula	1	0.40
VACTERL	1	0.40
Congenital diaphragmatic hernia	1	0.40
Duplex renal system	1	0.40
Total	28	11.11

The total of 28 babies (11.11%) had congenital anomalies. The most common anomaly detected was Binder's facies (5.16%, n=13). Table 3 shows distribution of congenital anomalies in study population.

The total of 28 babies (11.11%) had neonatal lupus. The most common manifestations were endocardial fibroelastosis (5.95%, n=15) followed by 8 babies (3.17%) having congenital heart block. Out of them, 5 babies (1.98%) had complete heart block and 3 babies (1.19%) had first degree heart block. Two of these mothers received antenatal dexamethasone therapy, however, none had advancement or reversion of heart block. One baby with complete heart block required IVIg, steroids and Ionotropic support after birth. None required pacemaker in the immediate postpartum period.

Of all the babies, 2 babies (0.8%) had cutaneous manifestations and both were under dermatologist follow up and did not require treatment. One baby (0.4%) had neonatal autoimmune thrombocytopenia and the mother had immunological thrombocytopenia. This baby required platelet transfusion and IVIg transfusion at birth. Two babies (0.8%) had autoimmune hemolytic jaundice. However, none of them required blood transfusions (Table 4).

Table 4: Neonatal lupus manifestations in study population.

Anomalies	N	%
Endocardial fibroelastosis	15	5.95
Congenital heart block	8	3.17
Cutaneous Lupus	2	0.79
Neonatal Thrombocytopenia	1	0.40
Hemolytic jaundice	2	0.79
Total	28	11.11

On comparing outcomes with predictors of SLE, presence of preexisting chronic hypertension was significantly associated with hypertensive disorders of pregnancy. Use of hydroxychloroquine (HCQs), azathioprine and heparin significantly reduced risk of hypertensive disorders in pregnancy. Remission of disease for <6 months was a significant risk factor for severe maternal morbidity and SLE flares in pregnancy. History of thromboembolic events in the past was also linked to severe maternal morbidity in pregnancy. Hematological flare was significantly related to presence of beta 2 GP1 antibodies while mucocutaneous flare was significantly related to presence of lupus anticoagulant (LAC). Renal flare in pregnancy was significant in mothers who had CKD while used of HCQs, azathioprine and tacrolimus significantly helped in reducing these flares.

Neonatal deaths were significantly associated with presence of lupus anticoagulant and anti-histone antibodies while anticardiolipin antibody (ACLA) was associated with preterm birth. Intake of HCQs significantly reduced risk of stillbirths, azathioprine significantly reduced risk of fetal growth restriction and preterm birth while heparin significantly reduced risk of stillbirth, fetal growth restriction and preterm birth. History of vasculitis was significantly associated with fetal growth restriction and lupus nephritis was significantly associated with preterm birth.

Table 5: Significant statistical associations with p value of maternal outcomes with predictors in study population.

Outcome	Predictors	P value
Hypertension in pregnancy	HCQs	0.0032
	Azathioprine	0.006
	Heparin	0.0338
	Chronic HTN	0.0026
Maternal morbidity (WHO near miss criteria)	Remission <6 months	0.0192
	History of Thrombosis	0.0006
Hematological flare	Beta 2 GP1	0.0263
	Remission <6 months	0.0003
Renal Flare	HCQs	0.0028
	Tacrolimus	0.0063
	Azathioprine	0.0027
	CKD	0.0001
	Remission <6 months	0.0342
Mucocutaneous flare	LAC	0.003
	Remission <6 months	0.0097

Table 6: Significant statistical associations with p value of fetal outcomes with predictors in study population.

Outcome	Predictors	P value
Neonatal death	LAC	0.0376
	Anti Histone	0.0001
Stillbirth	HCQs	0.004
	Heparin	0.0436
Fetal growth restriction	Heparin	0.0001
	Azathioprine	0.071
	History of vasculitis	0.0408
Preterm birth	ACLA	0.0113
	Heparin	0.0002
	Azathioprine	0.002
	Lupus nephritis	0.0028

Anti Ro and anti La antibodies were significantly associated with congenital heart block and endocardial fibroelastosis while anti Smith, anti RNP and anti Scl 70 Ab were significantly associated with Binder's facies and chondrodysplasia punctata. Use of HCQs significantly reduced the risk of congenital heart block. Azathioprine significantly reduced the risk of endocardial fibroelastosis and Binder's facies in our study. Use of PDE-5 inhibitors significantly reduced the risk of Binder's facies and chondrodysplasia punctata in our study.

Table 7: Significant statistical associations with p value of congenital fetal anomalies and neonatal lupus in study population.

Outcome	Predictors	P value
Congenital heart block	Anti Ro	0.0489
	Anti La	0.0034
	HCQs	0.0463
Endocardial fibroelastosis	Anti Ro	0.0017
	Anti La	0.0001
	Azathioprine	0.0034
Binder's Facies	Anti Smith	0.0001
	Anti RNP	0.0002
	Anti Scl 70	0.0001
	Azathioprine	0.0014
	PDE-5 I	0.0001
Chondrodysplasia punctata	Anti Smith	0.0174
	Anti RNP	0.0194
	Anti Scl 70	0.0012
	PDE-5 I	0.0012

Tables 5, 6 and 7 show important statistical associations along with p value between maternal and fetal outcomes with SLE predictors in pregnancy.

DISCUSSION

SLE is a disease of the reproductive age group. Only 2% of all the pregnancies in our study were conceptions following assisted reproductive techniques which reflects the fact that SLE does not affect the fertility. We also found that maximum number of women conceive within the first 5 years of SLE diagnosis putting them at a risk of complications as the disease may not be in remission for more than 6 months at the time of conception. All the flares of SLE in pregnancy in our study were treated by increasing the doses of immunosuppressant medications. Prepregnancy counselling significantly helped our mothers to go through pregnancy with minimal maternal and fetal complications.

In a study by Swee et al, they retrospectively studied 131 pregnancies over a period of 12 years.[9] They observed that 13% of all women had lupus nephritis while this number was 31.45% in our study. HCQs, prednisolone and low dose aspirin were the most commonly used medications which was also the case in our study. The incidence of preterm birth in their study was 24% which was lesser than that in our study while the incidence of fetal growth restriction was comparable with our study. In both the studies, 18% of the women experienced flare of SLE during pregnancy. The pattern of positive autoantibodies was similar in both the studies for ANA, anti Ro, anti La, ACLA, LA, beta 2 GP 1. Preeclampsia was significantly associated with chronic hypertension, antiphospholipid antibody syndrome and preexisting renal disease.

In a Korean study by Ko et al, they found statistically significant difference in adverse neonatal outcomes and preterm births with presence of antiphospholipid antibodies and active disease at the time of conception.[10] They also found significant association between lupus flare and preeclampsia and fetal growth restriction. Active disease at the time of conception in their study also increased the incidence of congenital anomalies in the fetus (10% versus 4%) but the difference was not statistically significant. They concluded that the disease has to be in remission for at least 4 months prior to conception to reduce complications in pregnancy.

Analysis of a large California database that included 555 lupus pregnancies (of a reported 1.2 million pregnancies) found that overall, SLE patients had significantly increased rates of hypertension, renal disease, preeclampsia, preterm delivery, cesarean delivery, fetal growth restriction (FGR), and neonatal death.[11]

In the recent PROMISSE study (Predictors of pregnancy outcome: biomarkers in antiphospholipid syndrome and systemic lupus erythematosus), a large prospective observational study of over 700 women with SLE, antiphospholipid antibodies and controls, the rate of severe flare was 3% in the second trimester and 3% in the third trimester, and the rate of mild or moderate flare was less than 15%. Importantly, these favorable outcomes may be caused by most enrolled SLE patients having fairly inactive disease with normal or near normal renal function.[12] Overall, patients with active disease during the 6 months before conception are at highest risk for flare during pregnancy.

A 2023 study by Singh et al observed increased risks of several adverse outcomes.[13] Women with SLE more often required rehospitalization, most notably at <6 months postpartum (6% versus 2%). Maternal postpartum rehospitalization was greatest for musculoskeletal flares. Infants of women with SLE more often had malformations (9% versus 6%), and increased mortality at <2 years. Hence, close follow-up during these time periods is crucial to minimize adverse outcomes especially in the initial months.

A retrospective study by Zamani et al evaluated fetal outcomes included FGR, stillbirth, abortion and preterm labor.[14] Fetal loss occurred in 43.8% of pregnancies. The most common laboratory findings in SLE patients were antinuclear antibody (81.4%) and anti-ds DNA positivity

(54.2%) which was similar to our study. Renal involvement, anti-double-stranded DNA positivity, anti-phospholipid antibody (APA) positivity and younger age at disease onset were significantly correlated with unfavorable pregnancy outcomes. A significant difference was observed between duration of SLE and FGR and still birth. The most unfavorable pregnancy outcome in women with SLE was spontaneous abortion. Our study did not analyze miscarriages as an outcome.

Nield et al described 13 cases of children with complete atrioventricular block associated with fibroelastosis of the endocardium, mainly affecting the left ventricle. Six mothers had positive titers of anti-SS-A and anti-SS-B antibodies, and seven mothers had only anti-SS-B antibodies. Endocarditis fibroelastosis developed from 6 to 12 weeks after prenatal diagnosis of CHB and from 7 months to 5 years after birth. Severe left ventricular dysfunction was found in all cases; in nine cases, it resulted in death, and in two cases in heart transplantation.[15] There are reports of dilated cardiopathy associated with anti-Ro antibodies, however, more definitive studies are required to establish association.

In studies done by Zuppa et al, the incidence of neonatal lupus in mothers with SLE with anti Ro antibodies was 50% which was quite high as compared to our study.[16] This is probably because most of our mothers were on HCQs, which has a protective effect on development of neonatal lupus. In the study by Zuppa et al, the incidence of congenital heart block was 16%.[16]

Past concerns regarding hydroxychloroquine (HCQ) being associated with fetal ocular toxicity and ototoxicity have not been confirmed by studies published within the past 15 years. Moreover, the continuation of HCQ during pregnancy may be beneficial, with one retrospective analysis showing that women who remained on HCQ during pregnancy had less severe SLE flares and required lower doses of prednisone.[17] There is also evidence that HCQ may reduce the likelihood of congenital heart block in at-risk fetuses. In a landmark study by Peter et al in 2020 published in the American Journal of Cardiology, they found that hydroxychloroquine (HCQ), when orally administered, through its Toll-like receptor antagonist activity reduced the recurrence rate of congenital heart block by 18%.[18] Given the apparent and possible benefits and the apparent lack of harm, many experts now prefer that women on HCQ who become pregnant should continue this medication during pregnancy. It is also a favored agent for the treatment of SLE flare in pregnancy.

In a study done by Vinet et al and published in the Journal of American Heart Association, they found statistically increased incidence of cardiac septal defects (ASD and VSD), cardiac valve anomalies and other cardiac anomalies in offsprings of mothers with SLE.[19] These babies were also significantly associated with more than 1 extracardiac anomalies. In our study, the cardiac anomalies found were Ebstein's anomaly, VACTERL syndrome and VSD.

In a study done in our own institute about the implications of antenatally diagnosed Binder's syndrome, 10% of all Binder's syndromes were associated with connective tissue disorders and 75% of these connective tissue disorders were SLE.

Binder's facies and connective tissue disorders have been reported in the literature in case reports. No reports have been published in the literature as to association with specific autoantibodies. There have been case reports of isolated Binder's facies as well as chondrodysplasia punctata with premature ossifications in SLE fetuses. Roy et al in 2013 published a case report of chondrodysplasia punctata.[20] The mother of the baby developed systemic lupus erythromatosus (SLE) with Ro/SSA antibodies 11 months after delivery. They postulated that Ro/SSA antibodies may generate calreticulin antibodies causing characteristic skeletal changes. A case series by Chitayat et al reported a series of eight cases of maternal collagen vascular disease associated with fetal chondrodysplasia punctata.[21] They concluded that chondrodysplasia punctata may be associated with maternal collagen vascular disease. They suggested that maternal autoimmune diseases should be part of the differential diagnosis and investigation in newborns/fetuses with CDP and all fetuses/newborn to mothers with autoimmune diseases should be examined for absent/hypoplastic nasal bone, brachydactyly, shortened long bones and epiphyseal stippling. In the mother reported by Schultz et al in 2010 serological studies failed to show the presence of anti-Ro/SSA or anti-La/SSb autoantibodies and instead disclosed high titers of anti-RNP antibodies.[22] This observation suggests that the transplacental crossing of anti-RNP or possibly another yet unidentified antibody mediate chondrodysplasia punctata (CDP).

In our study, use of drugs was decided on the basis of SLE status and organ systems involved. Doses were escalated and reduced as per SLE disease activity and flares. For drugs like tacrolimus and PDE-5 inhibitors, our study cannot establish definite association to prevent certain maternal and fetal outcomes as the number of mothers taking these drugs was less. Similarly, the sample size for anti Scl-70 Ab and anti-histone antibodies was small in our study. Hence, further studies with larger sample size are necessary to establish definite associations in these cases.

The society of maternal fetal medicine in March 2023 has laid down guidelines for management of SLE in pregnancy.[1] They recommend low-dose aspirin beginning at 12 weeks of gestation until delivery to decrease the occurrence of preeclampsia. They recommend all patients with SLE, other than those with quiescent disease, either continue or initiate HCQs in pregnancy. They suggested treatment with a combination of prophylactic unfractionated or low-molecular-weight heparin and low-dose aspirin for patients without a previous thrombotic

event who meet obstetrical criteria for APS and therapeutic dose for those with history of thrombosis. As also proven in our study, they also recommend that patients with SLE undergo prepregnancy counselling to optimize outcomes. Those with severe maternal risk, including patients with active nephritis; severe pulmonary, cardiac, renal, or neurologic disease; recent stroke; or pulmonary hypertension should be discouraged from getting pregnant. They recommend antenatal testing and serial growth scans in pregnant patients to monitor for FGR and avoid stillbirth.

CONCLUSION

From our study, we concluded that systemic lupus erythematosus adversely affects pregnancy, especially so if the disease has been in remission for less than 6 months and preconceptional counselling significantly helps to reduce adverse outcomes. Lupus nephritis is an independent risk factor for adverse pregnancy outcomes. Major maternal risks include hypertensive disorders of pregnancy, severe maternal morbidity and worsening flares of SLE in pregnancy. Major fetal outcomes include fetal growth restriction, preterm birth, perinatal mortality and neonatal lupus. Binder's facies and chondrodysplasia punctata are associated with autoimmune conditions with presence of autoantibodies- anti Smith, anti RNP and Scl 70. Endocardial fibroelastosis and congenital heart block was associated with anti-Ro and anti La antibodies. Mechanical PR interval monitoring and fetal 2D echo are essential to diagnose these issues in such cases. Use of HCQs, heparin and azathioprine may have a role in prevention of adverse maternal and perinatal outcomes. Multidisciplinary management of such mothers along with rheumatologists and fetal medicine consultants helps to optimize outcomes.

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