RESEARCH ARTICLE

Impact of lupus nephritis on foeto-maternal outcomes: A retrospective case-control study

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Abstract

Objective: To evaluate the impact of lupus nephritis on foeto-maternal outcomes in patients with systemic lupus erythematosus, and to compare it with those without lupus nephritis.

Method: The retrospective, case-control study was conducted from June to December 2023 at Aga Khan University Hospital, Karachi, and comprised data of pregnant patients with systemic lupus erythematosus from January 1998 to December 2019. Patients with biopsy-confirmed lupus nephritis were placed in group A, while those without lupus nephritis were placed in control group B. Maternal and perinatal outcomes were compared between the groups. Data was analysed using SPSS 19.

Results: Of the 125 women, 49(39.2%) were in group A with mean age 30.33±4.34 years, and 76(60.8%) were in group B with mean age 29.87±4.06 years (p>0.5). Antenatal flare-ups occurred in 17(34.7%) group A patients during the third trimester compared to 13(17.1%) in group B. The rate of pre-eclampsia and eclampsia were significantly higher in group A women compared to group B women (p<0.05). Premature births were seen more often in group A (p<0.05).

Conclusion: Third-trimester flare-ups were seen more often among pregnant women with lupus nephritis, and they were more likely to have pre-eclampsia/eclampsia and preterm birth.

Keywords: Pre-eclampsia, Eclampsia, Premature birth, foetal growth retardation, Lupus nephritis, Lupus erythematosus, Systemic. (JPMA 76: 493; 2026) **DOI: https://doi.org/10.47391/JPMA.20094**

Introduction

Systemic lupus erythematosus (SLE) is an autoimmune disorder that affects various organ systems.[1] Worldwide prevalence rate now exceeds 50-100 per 100,000 adults.[2] In Asia, the incidence of SLE ranges from 2.8 to 8.6 per 100,000 person-years, with prevalence rates between 26.5 and 103 per 100,000 individuals.[3] Pregnant women with SLE face higher risks of adverse maternal and perinatal outcomes. Research shows proper care and low-dose aspirin significantly improve SLE outcomes, particularly pre-eclampsia.[4]

Lupus nephritis (LN), a severe form of glomerulonephritis, is one of SLE's most serious manifestations. Therefore, renal function should be assessed in all SLE patients at their initial presentation.[5]

A study found that LN in women with SLE was linked to a higher risk of pre-eclampsia, and active nephritis at conception was correlated with poor pregnancy outcomes.[6]

A prospective study proved that LN raises the risk of maternal complications, such as flares (19.7%), pre-eclampsia (8.4%) and haemolysis, elevated liver enzymes and low platelets (HELLP) syndrome (2.8%). Nonetheless, these complications were reversible with prompt treatment.[7]

Recently, the significance of renal biopsy in predicting the prognosis of LN has significantly decreased mortality rates.[8] A study has proposed that LN may increase maternal complications, but is not directly associated with adverse foetal outcomes.[9]

Another study has shown that adverse foetal outcomes are mainly in the form of spontaneous first-trimester abortions, stillbirths and prematurity.[10]

Although different studies conducted in Asia have assessed pregnancy outcomes among SLE patients,[11-13] the comparison of evidence of adverse outcomes in patients with LN and without LN, to our knowledge, is lacking. The current study was planned to fill the gap in literature by evaluating the impact of LN on foeto-maternal outcomes in women with SLE, and to compare the foeto-maternal outcomes in patients with SLE with and without LN.

Materials and Methods

The retrospective, case-control study was conducted from June to December 2023 at Aga Khan University Hospital, Karachi, and comprised data of pregnant patients with SLE from January 1998 to December 2019. Owing to the

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retrospective design, no formal sample size calculation was conducted. After approval from the institutional ethics review committee, data archive was accessed for all SLE patients who presented during pregnancy and met at least four elements of the revised and updated criteria of the American College of Rheumatology (ACR) for SLE[14] with the help of International Classification of Diseases, Ninth Revision (ICD-9) coding.[15] Patients with biopsy-confirmed LN were placed in group A, while those without LN were placed in control group B. SLE patients with incomplete data were excluded.

Among the pregnant patients with SLE, biopsy-proven LN was classified as per the 2003 International Society of Nephrology/Renal Pathology Society (ISN/RPS) classification.[16] All the patients were on immuno-suppressive therapy, including azathioprine, hydroxy-chloroquine, steroids and tacrolimus, before pregnancy and continued with it during pregnancy.

LN flare was identified by active urinary sediment, persistent haematuria, cellular casts, increased proteinuria, a ≥30% rise in serum creatinine, elevated anti- double-stranded deoxyribonucleic acid (dsDNA) titers, and low complement component 3 (C3) and C4 levels.[17] Worsening proteinuria was defined as a 2g/24h increase from baseline or doubling in those with prior nephrotic range proteinuria or ≥1g/24h.[18] The distinction between pre-eclampsia and LN was established if an increase in proteinuria during pregnancy was accompanied by hypertension (HTN) in the latter half of the pregnancy without haematuria, red cell casts, or any sign of extra-renal flare. In the non-LN SLE group, disease activity was assessed using the Systemic Lupus Erythematosus Disease Activity Index 2000 (SLEDAI-2K) scoring system.[19] A score of 4 or higher showed active disease, while a score <4 signified inactive disease. Flare-ups were detected on the basis of new onset of anaemia, lymphocytopenia, increased anti-dsDNA titer, and decreased levels of C3 and C4.[20] Anticardiolipin antibodies immunoglobulin G (IgG) and IgM, results were expressed in terms of phospholipid units (GPL) and (MPL), respectively. They were defined as positive if IgG and IgM were >40. Values <20 were taken as weakly positive, 20-40 moderately positive, and >40 strongly positive. Each enrolled patient's lupus anticoagulants (LA) were evaluated, and positive values were set at ≥1.2.[21]

Patient demographics included age, height, weight, body mass index (BMI), parity and gestational age at booking and delivery. Obstetrical history included miscarriages. Maternal outcomes included thrombocytopenia, pre-eclampsia, eclampsia, preterm birth, intrauterine death (IUD), delivery mode, and LN flare-ups. Perinatal outcomes included SGA, IUGR, Appearance-Pulse-Grimace-Activity-Respiration (APGAR) scores, neonatal intensive care unit (NICU) stay and neonatal death.

Medical records showed that disease activity had been assessed at pregnancy onset, during pregnancy, and postpartum. Immunological markers included anticardiolipin IgG/IgM, anti-dsDNA, C3/C4, lupus anti-coagulant, and proteinuria levels. Routine lab tests, such as serum creatinine, uric acid, complete blood count (CBC), alanine transaminase (ALT) and proteinuria, had been done every 6-8 weeks during pregnancy.

Data was analysed using SPSS 19. Kolmogorov-Smirnov test was used to assess data normality. Frequencies and percentages were calculated for all categorical variables. Group comparisons were performed using chi-square test or Fisher's exact test. Continuous variables were expressed as mean±standard deviation or median with interquartile range (IQR; 25th-75th percentiles) and they were compared using independent samples t-test for normally distributed data, and Mann-Whitney U test for non-parametric data. P<0.05 was considered statistically significant.

Results

Of the 125 women, 49(39.2%) were in group A with mean age 30.33±4.34 years, and 76(60.8%) were in group B with mean age 29.87±4.06 years (p>0.5). The median gestational age at delivery in group A was 34 weeks (IQR: 29.5-36 weeks compared to 36 weeks (IQR: 34-38 weeks) in group B (p=0.001) (Table 1). The rate of preterm caesarean section (CS) was higher in group A compared to group B but the difference was not significant (p>0.5).

SLE flare-up was found in 86(68.9%) patients (95% confidence interval [CI]: 59.9-76.8%), and intergroup difference was significant (p<0.05). Preconception flairs

Table-1: Baseline characteristics of women with and without lupus nephritis (n=125).

Variables	Total	With lupus nephritis (n=49)	Without lupus nephritis (n=76)	*p*-value
Age (years)	125	30.33±4.34	29.87±4.06	0.550
Age at SLE	125	23.67±5.52	23.87±5.38	0.843
Weight (kg)	125	58.25±9.94	60.72±11.35	0.216
Height (cm)	125	156.51±5.23	157.79±6.29	0.239
BMI (kg/m^2)	125	23.75±3.59	24.41±4.39	0.369
Gestational age at booking (weeks)†	125	12[11-16]	12[9-18]	0.733
Gestational age at delivery (weeks) †	125	34[29.5-36]	36[34-38]	0.001*
Parity,		n (%)	n (%)	
Primigravida	38	17(34.7)	21(27.6)	0.214
Multigravida	83	32(65.3)	51(67.1)	
Grand Multigravida	4	0(0)	4(5.3)	

Data are presented as mean ± standard deviation, median [interquartile range], or n (%), as appropriate. Comparisons were made using an independent sample t-test, Mann–Whitney U test, or chi-square test; SLE: Systemic lupus erythematosus, BMI: Body mass index.

Table-2: Lupus nephritis flare-ups in relation to pregnancy trimesters.

Variables	Total	With lupus nephritis n(%) (n=49)	Without lupus nephritis n(%) (n=76)	*p*-value
Disease flare-up	86	42(85.7)	44(57.9)	0.001
Pre-conceptional	52	24(49)	28(36.8)	0.006
Antenatal	30	17(34.7)	13(17.1)	0.001
Postnatal	04	1(2)	3(3.9)	0.732
Pre-conceptional		24	28	
Within six months of conception	10	5(20.8)	5(17.9)	0.049
Six months year before conception	12	2(8.3)	10(35.7)	
More than one year before conception	30	17(70.8)	13(46.4)	
Antenatal		17	13	
1st Trimester	3	1(5.9)	2(15.4)	0.635
2nd Trimester	11	6(35.3)	5(38.5)	
3rd Trimester	16	10(58.8)	6(46.2)	

were reported in 52(41.6%) patients. They were significantly more in group B (p<0.05). Of the patients who had flare-ups before pregnancy, 30(56.6%) had it more than one year ago, and the intergroup difference was significant (p<0.05). Antenatal flare-ups occurred in 17(34.7%) group A patients compared to 13(17.1%) in group B (Absolute difference: 27.8%; 95% CI: 13.1-42.6%; p=0.001) (Table 2).

SLE manifestations, like fever, arthritis and lymphocytopenia, were significantly more in group B compared to group A, while seizure, proteinuria, haematuria, spike in creatinine, low C3 level, and anaemia were significantly more in group A compared to group B (Table 3).

Maternal outcomes revealed a significantly higher rate of pre-eclampsia in group A compared to group B (Absolute difference: 39.9%; 95% CI: 23.9-55.8%; p<0.01). A similar pattern was seen in patients with eclampsia (Absolute difference: 8.2%; 95% CI: 0.04-15.8%; p=0.02). There were 4(8.2%) maternal deaths in group A due to organ failure and sepsis. Premature births (<37 weeks) were more prevalent in group A than in group B (Absolute difference: 26.3%; 95% CI: 11.1-41.4%; p<0.05). Incidence of eclampsia was also significantly different between the groups (Table 4; Figure 1).

Among the neonates, IUGR 18(45%) and low birth weight 31(77.5%) were significantly higher in group A than group B (p<0.5). Neonatal deaths were more in group B compared to group A (p>0.05) (Table 4; Figure 2).

The foeto-maternal outcomes of group A women in the active and remission phases were compared, and the rate of pre-eclampsia was considerably more significant in the active phase compared to the remission phase (p=0.002). The active phase had a significantly higher frequency of thrombocytopenia (p=0.022) and preterm birth <34 weeks (p=0.002) than the remission phase. IUGR rate (p=0.006]

Table-3: Clinical characteristics of pregnant women with and without lupus nephritis.

Variables	Total n(%) (n= 125)	With lupus nephritis n(%) (n=49)	Without lupus nephritis n(%) (n=76)	*p*-value
Disease activity during pregnancy				
Active	29(23.2)	15(30.63)	14(18.4)	0.115
Remission Phase	96(76.8)	34(69.4)	62(81.6)	
Rashes	30(24)	8(16.3)	22(28.6)	0.107
Fever	52(41.6)	7(14.3)	45(59.2)	0.0005
Arthritis	52(41.6)	7(14.3)	45(59.2)	0.0005
Cerebrovascular system				
Seizure	11(8.8)	8(16.3)	3(3.9)	0.024
Stroke	1(1.3)	0(0)	1(1.3)	0.999
Visual disturbances	3(2.4)	2(4.1)	1(1.3)	0.561
Cardiovascular system				
Pericarditis and Myocarditis	7(5.6)	2(4.1)	5(6.6)	0.704
Chest pain	2(1.6)	0	2(2.6)	0.519
Shortness of Breath	1(0.8)	1(2)	0	0.392
Renal system				
Proteinuria >1g/day	42(33.3)	30(61.2)	12(15.8)	0.0005
Haematuria	19(15.2)	17(34.7)	2(2.6)	0.005
Creatinine >1.42 (mg/dl)	17(13.6)	16(32.7)	1(1.3)	0.0005
Immunology				
Anti-dsDNA positive (IU/ml)	97(77.6)	38(77.6)	59(77.6)	0.999
Low complement levels				
C3 (<0.8): (mg/dl)	24(19.2)	14(28.6)	10(13.2)	0.033
C4 (<0.1): (mg/dl)	19(15.2)	10(20.4)	9(11.8)	0.211
Haematology:				
Anaemia (Hb: < 11): (gram/dl)	65(52)	36(73.5)	29(38.2)	0.0005
Leukopenia (<1.5×10^9 lymphocytes/L)	7(5.6)	3(6.1)	4(5.3)	0.999
Lymphopenia (<3000/mm)	64(48.8)	31(36.7)	33(56.6)	0.030
Thrombocytopenia (<1Lac)	12(9.6)	7(14.3)	5(6.6)	0.153
ITP	3(2.4)	0	3(3.9)	0.279
APLA antibodies				
IgG positive (GPL)	52(41.6)	17(34.7)	35(46.1)	0.265
IgM positive (MPL)	50(40)	16(32.7)	34(44.7)	0.195
Lupus Anticoagulant	19(15.2)	9(18.4)	10(13.2)	0.428
Anti-Ro Antibodies (IU)	45(36)	15(30.6)	30(39.5)	0.314
Anti La Antibodies (IU)	15(12)	7(14.3)	8(10.5)	0.528
Positive ANA (IU/ml)	84(67.2)	33(67.3)	51(67.1)	0.978

Data are presented as n (%) for categorical variables. Comparisons between groups were made using chi-square test; dsDNA: Double-stranded deoxyribonucleic acid, Hb: Haemoglobin, C3.4: Complement component 3/4, APLA: Antiphospholipid antibody, ITP: Immune thrombocytopenia, Ig: Immunoglobulin, ANA: Antinuclear antibodies.

and APGAR score <7 at one minute (p=0.017) were significantly high in the active phase compared to the remission phase (Table 5).

Within group A, women with proteinuria >1g had significantly higher rates of preeclampsia (p=0.004], CS (p=0.005), preterm birth ≤34 weeks (p=0.013) and NICU admission (p=0.010) than those with <1g (Table 6).

Discussion

The current study highlights the significant impact of LN on pregnancy outcomes. Patients with LN faced higher

risks of adverse maternal outcomes, including lupus flare-ups, pre-eclampsia, eclampsia, IUGR and preterm birth. Laboratory markers, such as serum creatinine, proteinuria, haematuria, anaemia, lymphocytopenia and hypocomplementemia, were also significantly worse in LN patients. Perinatal outcomes were similar between the groups, except for low-birth-weight babies, IUGR, and NICU admission. Different studies have shown a correlation between pre-conceptional flare and poor maternal outcomes.[22,23] In the current study, the most SLE pre-conceptional flare-ups were seen in the LN group (70.8%) compared to the non-LN group (46.4%) and occurred more than one year before conception, whereas flares in <1 year before conception were more common in the non- LN group (Table 2).

The active disease at the beginning of pregnancy foresees the consequent flares during pregnancy.[24] Rahman et al.

Table-4: Foeto-maternal outcomes in pregnant women with and without lupus nephritis.

Outcomes	Total (n=125)	With lupus nephritis n(%) n=49	Without lupus nephritis n(%) n=76	*p*-value*
Maternal Outcome				
Gestational diabetes	27	11 (22.4)	16(21.1)	0.856
Pre-eclampsia	36	26(53.1)	10(13.2)	0.0005
Eclampsia	4	4 (8.2)	0 (0)	0.022
Thrombocytopenia	12	7(14.3)	5(6.6)	0.214
Maternal mortality	6	4(8.2)	2(2.6)	0.209
Caesarean section	72	32(65.3)	40(52.6)	0.162
Miscarriage	35	10(20.4)	25(32.9)	0.129
IUD	21	9(18.4)	12(15.8)	0.707
- 2nd trimester	15	7(77.8)	8(66.7)	0.659
- 3rd trimester	6	2(22.2)	4(33.3)	
Preterm birth†				
≤34 weeks birth	24	16/43(37.2)	8/66(12.1)	0.002
<37 weeks birth	79	38/43(88.4)	41/66(62.1)	0.003
Foetal and neonatal outcome		n=40	n=64	
SGA	10	5(12.5)	5(7.8)	0.146
- IUGR	34	18(45)	16(25)	0.034
Apgar score < 7 at 1 minute	7	3(7.5)	4(6.3)	0.805
Low Birth Weight	58	31(77.5)	27(42.2)	0.0005
NICU* admissions	35	16(40)	19(29.7)	0.279
Neonatal death	7	1(2.5)	6(9.4)	0.245

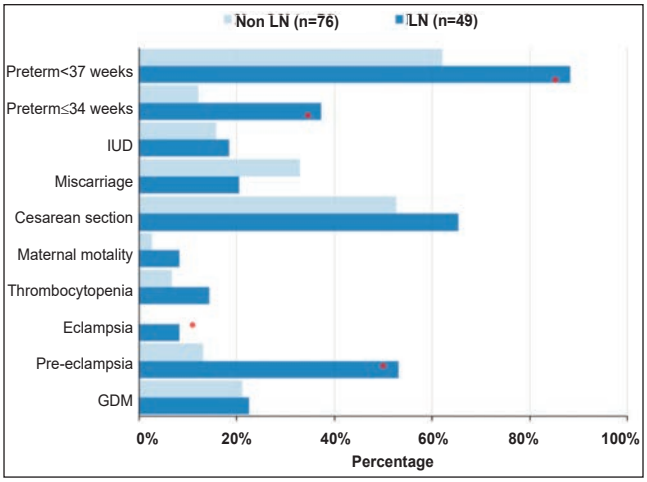

Figure-1: Comparison of maternal outcomes between lupus nephritis (LN) and non-LN groups.

**Significant at p<0.05; IUD: Intrauterine death; GDM: Gestational diabetes mellitus.*

Table-5: Foeto-maternal outcomes of pregnant women with lupus nephritis (LN) in active and remission phases (n=49).

Outcomes	Total (n=49)	Active (n=15)	Remission Phase (n=34)	*p*-value*
Maternal Outcome				
Gestational diabetes	11(22.4)	2(13.3)	9(26.5)	0.464
Pre-eclampsia	26(53.1)	13(86.7)	13(38.2)	0.002
Eclampsia	4(8.2)	3(20)	1(2.9)	0.079
Thrombocytopenia	7(14.3)	5(33.3)	2(5.9)	0.022
Maternal mortality	4(8.2)	3(20)	1(2.9)	0.079
Caesarean section	32(74.4)	10(83.3)	22(71)	0.698
Miscarriage	10(20.4)	1(6.7)	9(26.5)	0.145
IUD	9(18.4)	4(26.7)	5(14.7)	0.427
Preterm birth†				
≤34 weeks birth	16/43(37.2)	8/12(66.7)	8/31(25.8)	0.002
<37 weeks birth	38(88.4)	11/12(91.7)	27/31(87.1	0.999
Foetal and neonatal outcome	(n=40)	(n=11)	(n=29)	
SGA	5(12.5)	1(9.1)	4(13.8)	0.999
-IUGR	18(45)	9(81.8)	9(31)	0.006
APGAR score < 7 at 1 minute	3(7.5)	3(27.3)	0(0)	0.017
Low Birth Weight	31(77.5)	9(81.8)	22(75.9)	0.999
NICU* admissions	16(40)	7(63.6)	9(31)	0.080
Neonatal death	1(2.5)	1(9.1)	0(0)	0.275

*chi-square or Fisher exact test of association, *p*-value less than 0.05 taken as significant; NICU: Neonatal intensive care unit, SGA: Small gestational age, IUGR: Intra-uterine growth restriction, IUD: Intra-uterine death, APGAR: Appearance-Pulse-Grimace-Activity-Respiration; † Terminate cases were not in the denominator ¥ Denominator changed due to IUD.

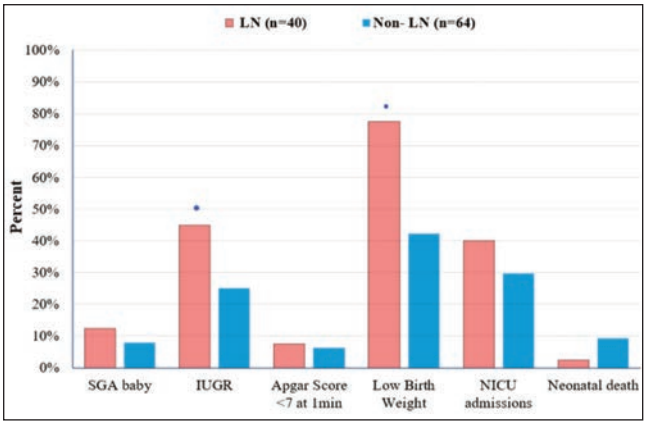

Figure-2: Comparison of foetal and neonatal outcomes between lupus nephritis (LN) and non-LN groups.

**Significant at p<0.05. NICU: Neonatal intensive care unit, SGA: Small gestational age, IUGR: Intra-uterine growth restriction, APGAR: Appearance-Pulse-Grimace-Activity-Respiration.*

Table-6: Foeto-maternal outcomes of pregnant women with lupus nephritis (LN) in relation to proteinuria.

Outcomes	Total (n=49)	Proteinuria ≤1g (n=19)	Proteinuria >1g (n=30)	p-value*
Maternal Outcome				
Gestational diabetes	11(22.4)	5(26.3)	6(20)	0.606
Pre-eclampsia	26(53.1)	5(26.3)	21(70)	0.004
Eclampsia	4(8.2)	1(5.3)	3(10)	0.555
Thrombocytopenia	7(14.3)	2(10.5)	5(16.7)	0.691
Maternal mortality	4(13.3)	0	4(13.3)	0.148
Caesarean section	32(74.4)	10(52.6)	22(91.7)	0.005
Miscarriage	10(20.4)	5(26.3)	5(16.7)	0.480
IUD	9(18.4)	1(5.3)	8(26.7)	0.127
Preterm birth†				
≤34 weeks birth	16/43(37.2)	3/19(15.8)	13/24(54.2)	0.013
<37 weeks birth	38/43(88.4)	17/19(89.5)	21/24(87.5)	0.999
Foetal and neonatal outcome	(n=40)	(n=18)	(n=22)	
- SGA	5(12.5)	2(11.1)	3(13.6)	0.999
- IUGR	18(45)	6(33.3)	12(54.5)	0.216
Apgar score < 7 at 1 minute	3(7.5)	1(5.6)	2(9.1)	0.999
Low Birth Weight	31(77.5)	14(77.8)	17(77.3)	0.999
NICU* admissions	16(40)	3(16.7)	13(59.1)	0.010
Neonatal death	1(2.5)	0	1(4.5)	0.999

*chi-square or Fisher exact test of association, p-value less than 0.05 taken as significant; NICU: Neonatal intensive care unit, SGA: Small gestational age, IUGR: Intra-uterine growth restriction, IUD: Intra-uterine death, APGAR: Appearance-Pulse-Grimace-Activity-Respiration; † Terminate cases were not in the denominator; ¥ Denominator changed due to IUD.

showed that inactive LN at the beginning of pregnancy suggested good pregnancy outcomes, and endorsed that disease activity should be controlled before pregnancy.[25] This was in contrast to the present study, which found that even with conception in remission, many LN patients experienced antenatal flare-ups compared to non-LN patients (Table 2).

It is challenging to differentiate active LN from pre-eclampsia. LN flare-ups can be established by declining complements, rising dsDNA antibody levels, proteinuria, haematuria and increasing creatinine.[26] The current study found antenatal flares more in the LN group (34.7%) than in the non-LN group (17.1%), especially in the third trimester. These patients had severe pre-eclampsia, resulting in preterm births. The distinction between pre-eclampsia and LN was made by monitoring HTN in the second half of the pregnancy with the escalation of proteinuria without rising creatinine, haematuria, red cell casts, or any sign of an extra-renal flare. A study has shown that patients with pre-existing LN have a 30% flare rate during pregnancy or postpartum.[27]

Bremme K et al. concluded in a case-control study in Sweden that LN in SLE patients exhibited increased rates of pre-eclampsia, eclampsia, IUGR and preterm birth, and these patients were also at high risk for CS.[28] The current study had similar findings.

Besides, the current study found that LN patients had more proteinuria, haematuria, rising creatinine levels, low complement levels more anaemia compared to the non-LN group (Table 3). These differences were significant, and the findings were consistent with earlier reports.[29]

Regarding preterm births, the current study found that the LN group had more preterm births at ≤34 weeks and at <37 weeks compared to the non-LN group, and the difference was significant. This was consistent with earlier findings reported by meta-analysis of 2,751 pregnancies[30] and a study by L Soubassi et al.[31]

The current study showed a higher rate of NICU admissions in the LN group compared to the non-LN group (Figure 2). This was due to increased preterm births in the LN group. Petri M. et al. reported comparable results regarding preterm births.[32] This difference might be due to the direct involvement of maternal-foetal medicine consultants in patient care and regular foetal scans from foetal medicine specialists.

Pregnant women with LN had higher rates of adverse perinatal outcomes, including miscarriages, DGA foetuses, IUGR, stillbirth and neonatal deaths.[33]

The current study found that the CS rate was higher in the LN group compared to the non-LN group (Figure 1; Table 2). This aligned with another study showing that LN was associated with higher CS rates.[34]

A study in 2012 evaluated maternal mortality in lupus, and showed that maternal mortality was associated with active disease in pregnancy.[35] In the current study, there were more maternal deaths in the non-LN group, especially among those in the active disease phase.

Significant adverse maternal and neonatal outcomes were more common in those who conceived during the active phase (Table 5). Numerous studies have advocated planning pregnancies during the remission phase to enhance outcomes.[36]

Adverse maternal outcomes were seen more in the current patients who developed proteinuria >1g (Table 6). A study reported that proteinuria <1g was associated with good renal outcomes.[37]

The long-term follow-up of two decades is among the strengths of the current study, which brings it in line with other large-scale studies.[38]

The current study has its limitations, including its

retrospective design, which may have introduced potential recall and documentation bias. Besides, it was conducted at a single tertiary care institution, which may have limited the generalisability of the findings.

Conclusions

Pregnant SLE women with LN were more at the risk of flare-ups in the third trimester, and were more likely to have pre-eclampsia/eclampsia and preterm birth. The outcomes may improve with pre-conceptional counselling and a multidisciplinary approach.

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DS & DR: Concept, design, data acquisition, analysis, interpretation, drafting, revision, final approval and agreement to be accountable for all aspects of the work.
AR: Data analysis and final approval.
AM: Revision, final approval and agreement to be accountable for all aspects of the work.