**Journal of Neonatal Surgery**
***ISSN(Online): 2226-0439***
***Vol. 14, Issue 21s (2025)***
*https://www.jneonatalsurg.com*

OPEN ACCESS

# A Severe Case of Postpartum Systemic Lupus Erythematosus: Clinical Challenges After Delivery

**Dyah Peni Puspitasari[1,2*], Awalia[3,4]**

[1]Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia;

[2]Department of Internal Medicine, Dr Soetomo General Academic Hospital, Surabaya, Indonesia;

[3]Division of Rheumatology, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia;

[4]Division of Rheumatology, Department of Internal Medicine, Dr Soetomo General Academic Hospital, Surabaya, Indonesia

***Corresponding Author:** Dyah Peni Puspitasari; Faculty of Medicine, Department of Internal Medicine, Universitas Airlangga, Dr Soetomo General Academic Hospital, Surabaya, Indonesia; dyah.peni.puspitasari-2018@fk.unair.ac.id

Cite this paper as: Dyah Peni Puspitasari, Awalia (2025). A Severe Case of Postpartum Systemic Lupus Erythematosus: Clinical Challenges After Delivery. *Journal of Neonatal Surgery*, 14 (21s), 1118-1124.

## ABSTRACT

**Background:** Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that frequently affects women of reproductive age. The disease can flare during pregnancy or postpartum, presenting complex clinical challenges due to hormonal fluctuations, immune system modulation, and multi-organ involvement. Severe postpartum flares, especially those involving neuropsychiatric and renal systems, require prompt diagnosis and targeted immunosuppressive therapy.

**Case Presentation:** We report a case of a 21-year-old primigravida woman who developed severe postpartum manifestations of SLE, three days following cesarean delivery due to preeclampsia, HELLP syndrome, and oligohydramnios at 32/33 weeks of gestation. The patient presented with seizures, altered mental status, and was diagnosed with neuropsychiatric SLE (NPSLE), lupus nephritis, peripartum cardiomyopathy (EF 45%), and systemic inflammatory response syndrome (SIRS) suspected due to urinary tract infection. Laboratory evaluation showed elevated ANA, hypocomplementemia (C3 and C4), and significant proteinuria. The patient met the SLICC criteria and scored 18 on the SLEDAI, indicating severe disease activity. She was treated with intravenous pulse methylprednisolone (750 mg/day for 3 days), followed by tapering doses, with noted clinical improvement. Cyclophosphamide was considered but postponed due to gastrointestinal symptoms. At discharge, she was continued on hydroxychloroquine, corticosteroids, mycophenolic acid, and supportive therapies. EEG and CT scan findings were unremarkable, while echocardiography confirmed PPCM. The patient remained clinically stable and was transitioned to outpatient follow-up.

**Conclusion:** This case highlights the complexity of diagnosing and managing severe postpartum SLE, particularly in primigravida patients with multiorgan involvement. Early recognition and aggressive immunosuppressive treatment are critical to improving outcomes. Multidisciplinary care and close postpartum monitoring remain essential in the management of high-risk SLE patients.

**Keywords:** *Systemic lupus erythematosus, postpartum flare, neuropsychiatric SLE, lupus nephritis, peripartum cardiomyopathy, immunosuppressive therapy*

## INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease involving immune complex-mediated damage to various organs and tissues (Kasper et al., 2015). It presents with a broad range of clinical, immunological, and laboratory abnormalities and most commonly affects women of reproductive age, with a prevalence of at least 1 in 1,000 in this population (Skorpen et al., 2016; Davis-Porada et al., 2020). The disease course is influenced by hormonal changes such as menstruation, menopause, and especially pregnancy (Jara et al., 2014). Flare rates during pregnancy and the postpartum period vary significantly, ranging from 13% to 74% (Jara et al., 2008; Skorpen et al., 2016; Eudy et al., 2018; Davis-Porada et al., 2020), with severe organ involvement such as nephritis or neuropsychiatric symptoms reported in 5% to 46% of cases (Zen et al., 2010). Risk factors for flares include active disease at conception, renal involvement, prior flares, and prednisone use (Eudy et al., 2018). Hormonal fluctuations may influence immune response, contributing to increased disease activity during pregnancy and postpartum (Zen et al., 2010). Although maternal and fetal outcomes have improved, active SLE remains a concern due to its potential complications (Jara et al., 2014). This paper presents a case report of a patient with severe postpartum SLE.

**Case Report**

A 21-year-old Javanese female was referred from the Department of Cardiology. The referral was prompted by the onset of seizures occurring three days following childbirth. Based on anamnesis, the patient was referred from a hospital in Pasuruan with a diagnosis of primigravida at 32/33 weeks gestation with suspected intrauterine fetal death (IUFD), complicated by preeclampsia, HELLP syndrome, and severe oligohydramnios. The patient was admitted under the care of the Obstetrics and Gynecology (Obgyn) team and subsequently underwent a cesarean section due to indications of severe preeclampsia and oligohydramnios. Postoperatively, she was transferred to the Cardiology team with a working diagnosis of peripartum cardiomyopathy. During hospitalization under the Cardiology team, the patient remained alert and communicative, without complaints of nausea, vomiting, dyspnea, or vaginal bleeding. However, on the second day of treatment, she began to report bilateral lower limb edema. Her urine output was recorded at 1200 mL over 24 hours. The therapeutic regimen included intravenous Furosemide 20 mg once daily, Furamine injection twice daily, Ceftriaxone 1 g IV twice daily (Day 1), oral Bromocriptine 2.5 mg twice daily, oral Bisoprolol 1.25 mg once daily, antacid syrup 15 mL three times daily, and fluid intake was restricted to a maximum of 1000 mL per 24 hours.

On the third day of hospitalization, the patient developed complaints of abdominal bloating and a sensation of fullness, followed by increasing restlessness and difficulty speaking, which progressed to a decrease in consciousness. A nasogastric tube was inserted, yielding approximately 50 mL of blackish fluid. The patient was administered a bolus of intravenous lansoprazole (2 ampoules), followed by a continuous lansoprazole infusion at 6 mg/hour. Later that night, the patient experienced a seizure episode characterized by ocular deviation and clonic movements of the upper and lower extremities, lasting less than five minutes. Postictally, the patient regained consciousness but remained somnolent, though still able to communicate. The patient was initiated on loading therapy with intravenous phenytoin 500 mg, followed by a maintenance dose of phenytoin 100 mg three times daily and oral folic acid 1 mg twice daily. A non-contrast head CT scan and electroencephalogram (EEG) were planned for further evaluation. There was no prior history of seizures, photosensitivity, arthralgia, oral ulcers, or alopecia. During the antenatal period, the patient underwent routine prenatal care and was informed of elevated blood pressure, although no antihypertensive therapy had been initiated. The patient denied any previous history of diabetes mellitus, chronic hypertension, autoimmune disease, renal or hepatic disorders. There was no family history of seizures, diabetes mellitus, hypertension, or autoimmune disorders.

On physical examination, the patient appeared in a generally weak condition. The Glasgow Coma Scale (GCS) score was 3-4-5. Vital signs were as follows: blood pressure 111/72 mmHg, heart rate 120 beats per minute, respiratory rate 22 breaths per minute, axillary temperature 36.8°C, and oxygen saturation of 97% on room air. Anthropometric measurements showed a body weight of 62 kg and a height of 155 cm. Head and neck examination revealed no signs of anemia, icterus, dyspnea, or cyanosis. Cardiovascular examination demonstrated regular heart sounds (S1 and S2) without the presence of murmurs or gallops. Pulmonary auscultation revealed bilateral vesicular, with no rhonchi or wheezing. Abdominal examination showed normal bowel sounds and no evidence of hepatomegaly or splenomegaly. Examination of the extremities revealed warm, dry, erythematous acral regions with a capillary refill time of less than 2 seconds, and pitting edema was noted in both lower limbs. Laboratory findings are summarized in Table 1. A chest radiograph (figure 1) revealed: (1) left-sided pleural effusion, and (2) cardiomegaly. Echocardiographic findings are presented in Table 2.

**Tabel 1. Laboratory Findings**

| | Day 2 | Day 5 | Day7 | Day 9 | Day 15 |
|---|---|---|---|---|---|
| Hemoglobin (g/dL) | 10,6 | | 7,7 | | 12,1 |
| Hematocrit (%) | 32,5 | | 24,5 | | 36,1 |
| MCV (fL) | 89,3 | | 91,1 | | 91,3 |
| MCH (pg) | 29,1 | | 28,6 | | 29,2 |
| MCHC (g/dL) | 32,6 | | 31,4 | | 32,4 |
| Leucocytes (uL) | 20260 | | 22080 | | 14450 |
| Neutrofils (%) | 88,5 | | 89,5 | | 86,6 |
| Lymphocytes (%) | 8,2 | | 7,4 | | 9,8 |
| Platelets (uL) | 141000 | | 100000 | | 165000 |
| Reticulocytes (%) | 2,31 | | | | |
| Ureum (mg/dL) | 54 | | 51 | 42,1 | 10,2 |
| Creatinine serum (mg/dL) | 2 | | 1,26 | 0,78 | 0,48 |
| AST (U/L) | 27 | | 25 | | 14,8 |
| ALT (U/L) | 8 | | 9 | | 6,9 |
| Albumin serum (g/dL) | 2,1 | 2,2 | 2 | 2,4 | 2,96 |
| Sodium (mmol/l) | 136 | 146 | 142 | 142 | 146 |
| Chloride (mmol/l) | 107 | 107 | 109 | 109 | 107 |
| Potassium (mmol/l) | 4,8 | 3,4 | 2,9 | 2,8 | 3,1 |

| | | | | | |
|---|---|---|---|---|---|
| Calcium (mg/dL) | 3,96 (corrected 5.64) | 3,95 (corrected 5,64) | 6,08 (corrected 7,3) | 6,6 (corrected 7,5) | 6,89 (corrected 7.3) |
| Magnesium (mg/dL) | 3,36 | 2,89 | 1,93 | | |
| Uric acid (mg/dL) | 12,77 | | | | |
| PPT | 14,9 | | | | |
| APTT | 32,4 | | | | |
| BGA results | pH 7,35<br>$pCO_2$ 25 mmHg<br>$pO_2$ 98 mmHg<br>$HCO^{3-}$ 13,8 mmol/l<br>BEecf -11.8mmol/l<br>SO2 97%. | | | | |
| Procalcitonin (ng/mL) | 0,17 | | | | |
| ANA test (AU/ml) | 205,4 | | | | |
| C3 (mg/dL) | 30 | | | | |
| C4 (mg/dL) | 9,5 | | | | |
| TSH (IU/ml) | 2,139 | | | | |
| FT4 (ng/dL) | 0,48 | | | | |
| Urinalysis results | pH 5<br>SG 1.018<br>Nitrite (-)<br>Glucose (-)<br>Erythrocyte 3+<br>Bilirubin (-)<br>Urobilinogen normal<br>Protein 2+<br>Leukocyte 1+<br>Ketone (-)<br>A:C >300<br>P:C >0.5 | | | | |

**Tabel 2. Echocardiographic Findings**

(1) Dilated left ventricular dimensions (LVIDd 5.0 cm) with eccentric LVH (LVDMi 99.63 g/m2; RWT 0.371), decreased left ventricular systolic function (EF by TEICH 45 %), left ventricular hypokinetic anteroseptal (B-M) and septal segmental analysis (A), the other segments are normokinetic. Left ventricular diastolic function grade I diastolic dysfunction
(2) Normal right ventricular dimensions, normal right ventricular systolic function (TAPSE 2.7 cm)
(3) Normal left atrial and right atrial dimensions
(4) No thrombus or intracardiac vegetation was found
(5) Heart valves have no abnormalities in morphology or function
(6) Minimal pericardial effusion (0.7cm basal; 0.7cm right lateral, and 0.9cm anterior), no pleural effusion was found.
(7) Echocardiographic results of hemodynamic parameters

PCWP 12.72mmHg

SVR 2077,05dynes.sec/cm5

mPAP 25.9 mm Hg

PVR 264,89dynes.sec/cm5

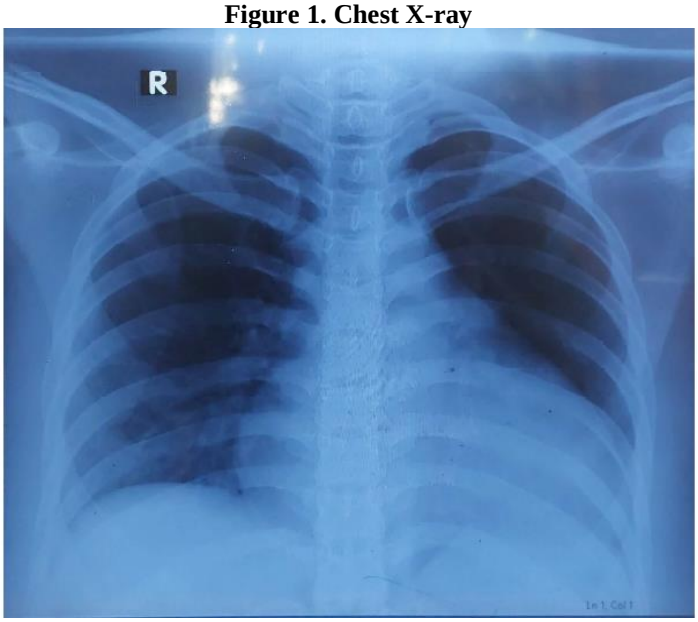
CO 3.98L/min

CI 2.47L/min.m2

LVOT VTI 13.3cm

EstRAP 10.00mmHg.

**Figure 1. Chest X-ray**




The patient was diagnosed with SLE with severe multi-organ manifestations (NPSLE, lupus nephritis). The management plan included correction of hypoalbuminemia, evaluation of urinary protein loss using the Esbach method, lipid profile assessment, and 24-hour urine output monitoring. The patient was initiated on intravenous pulse-dose methylprednisolone therapy at 750 mg once daily for three consecutive days. Additionally, intravenous calcium gluconate was administered at 1 ampoule five times daily via slow bolus. The patient was planned to be transferred to the high care unit of Internal Medicine.

By the fifth day of hospitalization, the patient's consciousness improved, with spontaneous eye opening and reduced restlessness. Seizure activity had ceased. She received pulse-dose intravenous methylprednisolone (750 mg/day), which led to gradual neurological recovery. By day 7, she was alert, responsive, and began sitting with assistance. Laboratory findings revealed dyslipidemia [low-density lipoprotein (LDL) cholesterol of 63 mg/dL, high-density lipoprotein (HDL) cholesterol of 16 mg/dL, total cholesterol of 127 mg/dL, and triglyceride level of 296 mg/dL] and low albumin. On day 9, methylprednisolone was tapered to 62.5 mg/day, and the patient was transferred to low-care for further recovery. By the 12th day of hospitalization, the patient was asymptomatic and able to sit independently. Physical examination showed an adequate general condition with a GCS score of 4-5-6 and stable vital signs. No abnormalities were found on systemic examination. Methylprednisolone was adjusted to 16 mg every 8 hours, and other treatments were continued. On day 15, the patient remained clinically stable with no new complaints. Vital signs and general physical examination findings remained within normal parameters. Laboratory findings are summarized in Table 1.

The final diagnosis included: severe SLE with altered consciousness suspected secondary to cerebral edema related to hypoalbuminemia versus neuropsychiatric SLE (NPSLE); serial generalized-onset clonic seizures; improved sepsis; peripartum cardiomyopathy (PPCM) with heart failure with reduced ejection fraction (HFrEF, EF 45%); hypertriglyceridemia (296 mg/dL); improved hypocalcemia (corrected calcium: 7.3 mg/dL); lupus nephritis; minimal pericardial effusion; hematemesis suspected secondary to stress-related mucosal disease (SRMD); and controlled hypertension post-emergency hypertensive crisis associated with preeclampsia. The patient was deemed clinically stable for outpatient follow-up and was scheduled for continued management in the internal medicine and rheumatology polyclinic. Discharge medications included Hydroxychloroquine 200 mg once daily, Methylprednisolone 16 mg twice daily, Furosemide 40 mg twice daily, Spironolactone 200 mg twice daily, Ciprofloxacin 500 mg twice daily, Potassium Slow Release 600 mg three times daily, Calcium Carbonate 1 tablet every 8 hours, Bromocriptine 2.5 mg every 12 hours, Bisoprolol 1.25 mg once daily, Ramipril 2.5 mg once daily, Phenytoin 100 mg every 8 hours, and Fenofibrate 300 mg once daily.

Following hospital discharge, she was evaluated in the rheumatology outpatient clinic. Subsequent investigations included an electroencephalogram (EEG), which showed normal findings without evidence of epileptiform discharges or abnormal slowing; a cranial CT scan, which revealed no signs of infarction, hemorrhage, or mass effect in the brain parenchyma; and an echocardiographic assessment that demonstrated concentric left ventricular hypertrophy, left ventricular dilation, and suspected peripartum cardiomyopathy (PPCM) with an ejection fraction of 35%. The patient's treatment regimen was then adjusted to include mycophenolic acid 360 mg three times daily, methylprednisolone 4 mg once daily, hydroxychloroquine 200 mg once daily, ramipril 2.5 mg once daily, furosemide 40 mg once daily, simvastatin 20 mg at bedtime, calcium carbonate one tablet three times daily, and folic acid one tablet daily. In addition, based on cardiology recommendations, bromocriptine 2.5 mg once daily and bisoprolol 1.25 mg once daily were included in her ongoing therapy.

**Discussion**

Systemic lupus erythematosus (SLE) is a multifactorial autoimmune disorder characterized by the involvement of multiple organ systems. Although its exact etiology remains unclear, a combination of genetic predisposition, immune dysregulation, hormonal influences, and environmental exposures is known to contribute to disease development. The pathogenesis of SLE is intricate and continues to be refined. In genetically susceptible individuals, exposure to environmental triggers can lead to a loss of self-tolerance and subsequent immune activation. This process involves antigen presentation following cellular injury—often due to infection or other environmental insults—which provokes an aberrant immune response involving T and B lymphocytes. The sustained autoimmune activity results in chronic inflammation, marked by cytokine release, complement activation, and the production of autoantibodies, all of which contribute to progressive tissue and organ damage (Indonesian Rheumatology Association, 2019; Vaillant et al., 2022).

SLE predominantly affects women of reproductive age, with female-to-male prevalence ratios ranging from 1.2:1 up to 15:1 (Rees et al., 2017). The clinical presentation is heterogeneous, ranging from mild mucocutaneous involvement and arthritis to severe manifestations involving major organs such as the kidneys, lungs, heart, and central nervous system (Vaillant et al., 2022).

Diagnosis of SLE relies on a combination of clinical features and laboratory investigations. The use of standardized classification criteria can aid diagnostic accuracy. One widely used system is the Systemic Lupus International Collaborating Clinics (SLICC) criteria, which requires either: (1) the presence of at least four criteria, with at least one clinical and one immunological criterion, or (2) histologically confirmed lupus nephritis in the presence of antinuclear antibody (ANA) or anti-double-stranded DNA (anti-dsDNA) positivity. Clinical criteria include cutaneous manifestations, oral ulcers, non-scarring alopecia, synovitis, renal involvement, neuropsychiatric symptoms (e.g., seizures, psychosis), hematologic abnormalities (hemolytic anemia, leukopenia, lymphopenia, thrombocytopenia), among others. Immunological criteria encompass ANA, anti-dsDNA, anti-Sm, antiphospholipid antibodies, low complement levels (C3, C4), and a positive direct Coombs test in the absence of hemolytic anemia (Petri et al., 2012; Kasper et al., 2015; Indonesian Rheumatology Association, 2019).

In the present case, the patient fulfilled the SLICC classification for SLE. Immunological findings revealed a positive ANA (205.4 AU/mL) and hypocomplementemia (C3: 30 mg/dL; C4: 9.5 mg/dL). Clinically, the patient exhibited lupus nephritis, as evidenced by a protein-to-creatinine ratio >0.5, as well as neuropsychiatric manifestations including seizures and an acute confusional state. Therefore, the presence of these features meets the minimum four-point threshold required by the SLICC criteria, supporting the diagnosis of SLE.

Evaluating disease activity in SLE is essential to ensure that therapeutic strategies are appropriately tailored to the individual patient. This evaluation should begin at the time of diagnosis. A number of validated tools have been developed to assess disease activity, among which the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) is widely utilized. The SLEDAI includes 24 clinical and laboratory parameters encompassing nine organ systems, allowing for a comprehensive overview of disease involvement. Scores are stratified into the following categories: no activity (score 0), mild activity (scores 1–5), moderate activity (scores 6–10), severe activity (scores 11–19), and very severe activity (scores ≥20) (Gladman et al., 2002; Indonesian Rheumatology Association, 2019; Bartels, 2022).

In this case, the patient was diagnosed with SLE and obtained a SLEDAI score of 18, with contributing factors including seizures, organic brain syndrome, and decreased complement levels. According to the scoring system, this places the patient in the category of severe disease activity.

SLE activity may increase during pregnancy or the postpartum period (Doria et al., 2008). Several studies have documented an elevated risk of disease flares during pregnancy and within 6 weeks to 3 months after delivery (Ruiz-Irastorza et al., 1996; Eudy et al., 2018), with reported flare rates ranging between 13.5% and 65% (Doria et al., 2008; Saavedra et al., 2014). Flares during these periods are of particular concern, especially postpartum, as they may impair the mother's ability to care for her newborn (Mouyis, 2020). Although fewer studies have examined the postpartum period specifically, one study reported an increased flare rate during the first 3 months postpartum, though not over a 12-month period, indicating that the heightened immune activity during pregnancy can persist into early postpartum (Eudy et al., 2018).

SLE flares are thought to occur due to an imbalance between regulatory and activating immune mechanisms. In genetically predisposed individuals, the immune system is primed toward activation, and external triggers can further elevate this activity beyond a clinical threshold, resulting in flares. Triggers include ultraviolet light, infections, hormonal changes (e.g., elevated estrogen or prolactin), and certain medications that affect DNA methylation (Fernandez and Kirou, 2016). Consequently, the occurrence of severe disease flares during pregnancy or the postpartum period can be understood through immunologic, neuroendocrine, and clinical frameworks (Jara et al., 2014).

The marked female predominance and peak disease onset during adolescence and early adulthood suggest a significant hormonal component in SLE pathogenesis, with estrogen being particularly implicated (Fernandez and Kirou, 2016). Although mechanisms remain under investigation, elevated levels of estrogen, progesterone, and prolactin during pregnancy and postpartum are believed to contribute to immunologic shifts that exacerbate SLE activity (Fernandez and Kirou, 2016; Jara et al., 2008). Interestingly, one study suggests that administration of bromocriptine for 14 days postpartum may reduce SLE flare rates for up to one year (Fernandez and Kirou, 2016).

Hormonal changes during pregnancy, particularly in the third trimester, influence cytokine expression and immune

responses. Elevated levels of cortisol, estrogen, progesterone, and vitamin D suppress Th1 cytokines (e.g., IFN-γ, IL-1, IL-2, IL-12, TNF-α), which are associated with cell-mediated immunity, while promoting Th2 cytokines (e.g., IL-4, IL-5, IL-6, IL-10) that support humoral immunity (Jara et al., 2008; Zen et al., 2010; de Jesus et al., 2015). This shift may explain the worsening of Th2-mediated diseases like SLE during pregnancy (Zen et al., 2010; de Jesus et al., 2015).

SLE is characterized by dysregulated T and B cell tolerance, leading to the production of pathogenic autoantibodies. Sex steroid hormones, particularly estrogen, enhance B cell activation and Th2 immune responses. During pregnancy, high concentrations of estrogen and gestagens further stimulate IL-4, IL-10, TGF-β, and IFN-γ while reducing TNF-α levels. In patients with SLE, IL-6 levels fail to rise during the third trimester as in healthy pregnancies, whereas IL-10 levels remain persistently high, leading to continuous B cell stimulation (Cutolo et al., 2004; de Jesus et al., 2015). Postpartum flares are influenced by similar mechanisms, including immune hyperactivity, elevated autoantibodies, high prolactin levels, active lupus nephritis, and a rebound in Th1 cytokines such as IL-12 and TNF-α (Jara et al., 2008; Jara et al., 2014).

Clinically, active SLE at the onset of pregnancy—particularly with manifestations such as thrombocytopenia, lupus nephritis, hypertension, antiphospholipid syndrome, and preeclampsia—has been linked to higher flare rates and adverse fetal outcomes (Jara et al., 2014). On average, 2.4 organ systems are involved during flares, most commonly presenting with systemic symptoms (e.g., fever, fatigue), cutaneous lesions, renal involvement, and musculoskeletal symptoms (Petri et al., 1991; Saavedra et al., 2014). Flares during pregnancy are associated with increased risk of preeclampsia and preterm delivery. In one multivariate analysis, primigravida status was identified as a risk factor for any flare during pregnancy (OR 2.3) (Saavedra et al., 2014).

In this case, the patient was a primigravida diagnosed with SLE three days postpartum. Multiorgan involvement was observed, including lupus nephritis and NPSLE. The patient also had preeclampsia and delivered preterm at 32/33 weeks of gestation.

The selection of therapy in systemic lupus erythematosus (SLE) is primarily guided by the level of disease activity and the specific organs involved (Tsokos, 2011; Bartels, 2022). The European Alliance of Associations for Rheumatology (EULAR) provided treatment guidelines in 2008, which were later updated in 2019. These guidelines emphasize remission as the primary goal of therapy; however, when remission is unattainable, the objective shifts to reducing disease activity across all affected organ systems. Hydroxychloroquine is recommended for all SLE patients due to its favorable safety profile and ability to reduce flares. Glucocorticoids are useful for rapid symptom control, though the long-term strategy should focus on minimizing the daily dose to 7.5 mg or less (prednisone equivalent) or discontinuing it entirely. To support steroid tapering and reduce relapse risk, the timely initiation of immunosuppressive therapy is advised. The choice of immunosuppressive agents should be individualized based on the patient's disease manifestations, age, reproductive considerations, drug safety, and cost (Bertsias et al., 2008; Fanouriakis et al., 2019). In cases of severe SLE, a comprehensive assessment is needed to rule out alternative causes such as infections. Management strategies should be tailored to the underlying etiology, utilizing immunosuppressants and, where appropriate, anticoagulants. For severe disease activity, immunosuppressive regimens may include intravenous methylprednisolone or oral prednisolone at doses up to 1 mg/kg/day, with pulse methylprednisolone therapy (500–1,000 mg/day for three consecutive days) as an option in acute settings (Indonesian Rheumatology Association, 2019). In patients with neuropsychiatric SLE (NPSLE), glucocorticoids and/or immunosuppressive agents are recommended if inflammation is the suspected mechanism. Lupus nephritis is typically managed with an induction phase followed by long-term maintenance, with mycophenolate mofetil or cyclophosphamide as the preferred agents for induction (Indonesian Rheumatology Association, 2019; Bartels, 2022). Postpartum SLE flares are frequently managed effectively with corticosteroids (Mouyis, 2020).

In the case presented, the patient was treated with pulse intravenous methylprednisolone at 750 mg daily for three days, resulting in clinical improvement in neuropsychiatric symptoms. Although cyclophosphamide was initially planned for further treatment, it was postponed due to the patient experiencing abdominal pain. Upon discharge and transition to outpatient care, the patient was prescribed hydroxychloroquine 200 mg daily and methylprednisolone 16 mg twice daily. Due to ongoing immunosuppressive therapy, the patient was advised against breastfeeding.

**Conclusion**

This case highlights the complexity of managing severe postpartum systemic lupus erythematosus (SLE) in a primigravida patient with multiorgan involvement, including neuropsychiatric lupus and lupus nephritis, following preeclampsia and preterm delivery. Hormonal and immunological changes in the peripartum period can trigger disease flares, necessitating early recognition and aggressive treatment. Diagnosis in this patient was established using the SLICC criteria and a high SLEDAI score, indicating severe activity. Clinical improvement was achieved through prompt administration of intravenous pulse methylprednisolone, followed by continued immunosuppressive therapy. This case emphasizes the importance of a multidisciplinary approach in managing high-risk SLE patients during the postpartum period to optimize both maternal and neonatal outcomes.

## REFERENCES

[1] Bartels, C. M. 2022. Systemic Lupus Erythematosus (SLE) Treatment & Management. Available in https://emedicine.medscape.com/article/332244-overview.

[2] Bertsias, G., Ioannidis, J. P., Boletis, J., Bombardieri, S., Cervera, R., Dostal, C., et al. 2008. EULAR recommendations for the management of systemic lupus erythematosus. Report of a Task Force of the EULAR Standing Committee for International Clinical Studies Including Therapeutics. *Ann Rheum Dis*; 67(2): 195-205. doi:10.1136/ard.2007.070367.
[3] Cutolo, M., Sulli, A., Capellino, S., Villaggio, B., Montagna, P., Seriolo, B., Straub, R. H. 2004. Sex hormones influence on the immune system: basic and clinical aspects in autoimmunity. *Lupus*; 13(9): 635-638. doi:10.1191/0961203304lu1094oa.
[4] Davis-Porada, J., Kim, M. Y., Guerra, M. M., Laskin, C. A., Petri5, M., Lockshin, M. D., Sammaritano, L. R., Branch, D. W., Sawitzke, A., Merrill, J. T., Buyon, J. P., Salmon, J. E. 2020. Low frequency of flares during pregnancy and post-partum in stable lupus patients. *Arthritis Research & Therapy*; 22: 52. https://doi.org/10.1186/s13075-020-2139-9.
[5] De Jesus, G. R., Mendoza-Pinto, C., de Jesus, N. R., Dos Santos, F. C., Klumb, E. M., Carrasco, M. G., Levy, R. A. 2015. Understanding and Managing Pregnancy in Patients with Lupus. *Autoimmune Dis*; 943490. doi:10.1155/2015/943490.
[6] Doria, A., Tincani, A., Lockshin, M. Challenges of lupus pregnancies. *Rheumatology*; 47(3): 9-12. doi:10.1093/rheumatology/ken151.
[7] Eudy, A., Siega-Riz, A. M., Engel, S. M., Franceschini, N., Howard, A. G., Clowse, M., Petri, M. 2018. Effect of pregnancy on disease flares in patients with systemic lupus erythematosus. *Ann Rheum Dis*; 77(6): 855–860. doi:10.1136/annrheumdis-2017-212535.
[8] Fernandez, D., Kirou, K. A. 2016. What Causes Lupus Flares? *Curr Rheumatol Rep*; 18: 14. doi:10.1007/s11926-016-0562-3.
[9] Fanouriakis, A., Kostopoulou, M., Alunno, A., Aringer, M., Bajema, I., Boletis, J. N., et al. 2019. 2019 update of the EULAR recommendations for the management of systemic lupus erythematosus. *Ann Rheum Dis*; 78(6): 736-745. doi:10.1136/annrheumdis-2019-215089.
[10] Gladman, D. D., Ibañez, D., Urowitz, M. B. Systemic lupus erythematosus disease activity index 2000. *J Rheumatol*; 29(2): 288-291. PMID: 11838846.
[11] Jara, L. J., Medina, G., Cruz-Dominguez, P., Navarro, C., Vera-Lastra, O., Saavedra, M. A., Blanco-Favela, F., Espinoza, L. R. 2008. Pregnancy, Hormones, and Autoimmune Rheumatic Diseases. *Handbook of Systemic Autoimmune Diseases*; 9: 185-197. doi:10.1016/S1571-5078(07)00216-4.
[12] Jara, L. J., Medina, G., Cruz-Dominguez, P., Navarro, C., Vera-Lastra, O., Saavedra, M. A. 2014. Risk factors of systemic lupus erythematosus flares during pregnancy. *Immunol Res*; 60(2-3): 184-92. doi:10.1007/s12026-014-8577-1.
[13] Kasper, D. L., Hauser, S. L., Jameson, J. L., Fauci, A. S., Longo, D. L. & Loscalzo, J. 2015. *Harrison's Principles of Internal Medicine*, McGraw-Hill.
[14] Mouyis, M. 2020. Postnatal Care of Woman with Rheumatic Diseases. *Adv Ther*; 37(9): 3723-3731. doi:10.1007/s12325-020-01448-1.
[15] Indonesian Rheumatology Association. 2019. Rekomendasi Perhimpunan Reumatologi Indonesia, Diagnosis dan Pengelolaan Systemic Lupus Erythematosus.
[16] Petri, M., Howard, D. and Repke, J. 1991. Frequency of lupus flare in pregnancy: The hopkins lupus pregnancy center experience. *Arthritis & Rheumatism*; 34: 1538-1545. https://doi.org/10.1002/art.1780341210.
[17] Petri, M., Orbai, A. M., Alarcón, G. S., Gordon, C., Merrill, J. T., Fortin, P. R., et al. 2012. Derivation and validation of the Systemic Lupus International Collaborating Clinics classification criteria for systemic lupus erythematosus. *Arthritis Rheum*; 64(8): 2677-2686. doi:10.1002/art.34473.
[18] Rees, F., Doherty, M., Grainge, M. J., Lanyon, P., Zhang, W. 2017. The worldwide incidence and prevalence of systemic lupus erythematosus: a systematic review of epidemiological studies. *Rheumatology*; 56: 1945-1961. doi:10.1093/rheumatology/kex260
[19] Ruiz-Irastorza, G., Lima, F., Alves, J., Khamashta, M. A., Simpson, J., Hughes, G. R. V., Buchanan, N. M. M. 1996. Increased Rate of Lupus Flare During Pregnancy and The Puerperium: A Prospective Study of 78 Pregnancies. *British Journal of Rheumatology*; 35(2): 133-138. doi:10.1093/rheumatology/35.2.133.
[20] Saavedra, M. A., Sanchez, A., Morales, S., Navarro-Zarza, J. E., Angeles, U., Jara, L. J. 2014. Primigravida is associated with flare in women with systemic lupus erythematosus. *Lupus*; 24(2): 180-185. doi:10.1177/0961203314552116.
[21] Skorpen, C. G., Lydersen, S., Gilboe, IM., Skomsvoll, J. F., Salvesen, K. A., Palm, O., Koksvik, H. S. S., Jakobsen, B., Wallenius, M. 2016. Disease activity during pregnancy and the first year postpartum in women with systemic lupus erythematosus. *Arthritis Care & Research*; 69 (8): 1201-1208. doi:10.1002/acr.23102.
[22] Tsokos, G. C. 2011. Mechanisms of Disease Systemic Lupus Erythematosus. *N Engl J Med*; 365: 2110-2121. doi:10.1056/NEJMra1100359.
[23] Vaillant, A. A. J., Goyal, A., Varacallo, M. 2022. Systemic Lupus Erythematosus. Available in https://www.ncbi.nlm.nih.gov/books/NBK535405/?report=printable. PMID: 30571026.
[24] Zen, M., Ghirardello, A., Iaccarino, L., Tonon, M., Campana, C., Arienti, S., Rampudda, M., Canova, M., Doria, A. 2010. Hormones, immune response, and pregnancy in healthy women and SLE patients. *Swiss Medical Weekly*; 140(13-14): 187–201. doi:10.4414/SMW.2010.12597.