

Treatment challenges of severe hematologic manifestations of SLE in early pregnancy in a limited setting: A case report

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Abstract: Hematologic abnormalities are among the most common initial manifestations of systemic lupus erythematosus (SLE), primarily including anemia, thrombocytopenia, and leukopenia. Notably, pregnancy can influence SLE disease activity, and conversely, SLE is associated with adverse pregnancy outcomes. Several treatment strategies—such as high-dose corticosteroids, immunosuppressants, intravenous immunoglobulin, and splenectomy—have been proposed to manage severe hematologic manifestations during pregnancy. However, treatment remains particularly challenging in resource-limited settings. We report a case of a young woman at 12–13 weeks of gestation who presented with severe anemia and thrombocytopenia and was subsequently diagnosed with SLE. An intravenous pulse dose of methylprednisolone and a steroid-sparing agent were prescribed, resulting in improved hemoglobin levels and platelet counts, although thrombocytopenia recurred. Following a medical decision to terminate the pregnancy, an incomplete abortion occurred. An emergency uterine curettage under general anesthesia was subsequently performed despite low platelet counts. Given the resource-limited setting, this case illustrates the complexity of managing SLE with severe hematologic manifestations during early pregnancy. It underscores the importance of timely multidisciplinary care, a careful assessment of the safety of invasive procedures, and the development of clear clinical guidelines to improve maternal outcomes in similar cases.

Keywords: Anemia, Pregnancy, Systemic lupus erythematosus, Thrombocytopenia.

1. Introduction

Systemic lupus erythematosus (SLE) is a condition which occurs when the body's immune system targets healthy cells and tissues. The excessive response of B cells and T cells and the loss of immune tolerance against self-antigens are hallmarks of immune activation in SLE [1]. Globally, the incidence of SLE is about 5.14 per 100,000 person-years and about 400,000 people per year are newly diagnosed with SLE. In Indonesia, the estimated incidence is higher, reaching 7.4 cases per 100,000 person-years [2]. Critically, SLE is more prevalent in women of reproductive age. One theory proposed to explain this dynamic revolves around the X chromosome's association with immune system dysregulation. Due to its comparably higher rates of manifestation in this population of women, pregnancy has become an important issue in managing SLE. Pregnancy can affect SLE disease activity, and conversely, SLE can affect pregnancy outcome [3].

Hematologic involvement in SLE has been frequently observed, especially during the first year of diagnosis, and is associated with significant morbidity. Anemia is the most frequent form, seen in more than 50% of patients. It may present as anemia of chronic disease, iron deficiency anemia, and hemolytic anemia [4]. Thrombocytopenia in SLE has been linked to the presence of antiplatelet antibodies and circulating immune complexes causing peripheral platelet destruction [5]. Beyond its association with higher disease activity, thrombocytopenia also appears as one of the risk factors for poor pregnancy

outcomes, including pregnancy loss, preterm birth, and intrauterine growth restriction [3, 5]. To help expand our understanding of hematologic complications in SLE during pregnancy, we report a case of a young woman in early pregnancy who presented with severe anemia and thrombocytopenia, which was later diagnosed with SLE.

2. Case Report

A 30-year-old woman at 12–13 weeks of gestation was referred to the emergency room (ER) due to uncontrollable nose and gum bleeding for three days. The patient had no history of trauma. She had been given nasal packing and oral drugs, including tranexamic acid, to relieve her symptoms. Despite the treatment, the bleeding had not subsided, and initial laboratory examinations revealed that she had severe anemia (hemoglobin 5.0 g/dL) and thrombocytopenia (platelet counts 5000/mm³), which prompted clinicians to refer her to a tertiary hospital. She also had a low-grade fever for two days and tiny red spots on both her upper and lower extremities. There were no complaints of hair loss or joint pain and no rash in the cheeks.

The patient had no history of previous medical conditions, including experiencing similar symptoms, hospitalization, or a blood transfusion. She had been married for 14 years with one child aged 11 years old. There was no history of complications during the previous pregnancy. During this pregnancy, she routinely went to the primary health center to receive antenatal care. Basic routine laboratory examinations done at 9–10 weeks of gestation revealed a hemoglobin level of 9.2 g/dL.

The physical examination done in the ER revealed that she was pallor, with no icterus or hepatosplenomegaly. There was pitting edema in her feet and petechiae on both her upper and lower extremities. Laboratory findings at the time of admission revealed that she had severe hypochromic microcytic anemia (3.6 g/dL) and severe thrombocytopenia (3000/mm³). She also had hypoalbuminemia (1.62 g/dL). No abnormal results were found in the urinalysis, urinary sediment analysis, or serum bilirubin. The iron blood test found results in the normal range and a peripheral blood smear showed no abnormal findings. Immunological testing revealed a high ANA titer accompanied by decreased complement levels (C3 and C4).

Based on the clinical and laboratory findings, she was diagnosed with SLE with moderate activity (SLEDAI score of 7). Following the diagnosis, she was given a pulse dose of intravenous methylprednisolone of 500 mg per day for three days. A total of 10 bags of thrombocyte concentrate (TC) and two bags of packed red cell (PRC) transfusions were also prescribed.

The repeated laboratory examination showed that there was an increase in hemoglobin levels to 5.3 g/dL and platelet counts to 30,000/mm³. Serum albumin was also increased to 2.7 g/dL following an albumin transfusion. Treatment was then continued with a methylprednisolone injection of 62.5 mg per day and three bags of PRC transfusions. After three days, another laboratory examination showed that there was an increase in hemoglobin levels to 10.4 g/dL. However, the platelet count decreased again to 9000/mm³. The methylprednisolone injection was continued with the same doses, and she was given cyclosporine oral tablets of 100 mg/day. A multidisciplinary discussion concluded that the pregnancy was to be terminated after optimizing the patient's general condition for one to two weeks, including achieving a minimal platelet count of 50,000/mm³.

During in-patient treatment following four days of cyclosporine prescription and seven days of methylprednisolone injection, the patient developed hematuria. Laboratory examinations revealed a hemoglobin level of 9.8 g/dL, increased platelet count of 14,000/mm³, and decreased serum albumin of 2.48 g/dL. A repeated urinalysis found proteinuria (4+), erythrocytes (3+), and casts. The methylprednisolone injection was then stopped, with treatment transition to 32 mg per day of oral methylprednisolone. A 24-hour urine protein excretion was collected and resulted in a protein urine excretion of 3.75 grams per day. She was then diagnosed with lupus nephritis. Repeated platelet counts showed that there was an increase to 44,000/mm³. The patient was then referred to the obstetrics department for pregnancy termination preparation.

However, three days later, the patient had an incomplete abortion. Repeated complete blood counts (CBCs) showed hemoglobin levels of 8.8 g/dL and platelet counts of 37,000/mm³. The patient was then prepared for an emergency uterine curettage with general anesthesia. A stress dose of methylprednisolone injection with a dose of 62.5 mg was prescribed. During curettage, she was given 10 bags of TC. One day later, she had no bleeding. CBCs showed a decrease in hemoglobin level (6.8 g/dL) and platelet counts of 45,000/mm³. A packed red cell transfusion was then prescribed and treatment was continued with 32 mg/day of methylprednisolone and 100 mg/day of cyclosporine delivered orally. After receiving three bags of PRCs, she had no complaints of bleeding. Repeated CBCs showed that there were increases in hemoglobin levels to 9.6 g/dL, and platelet counts to 95,000/mm³. She was then discharged from in-patient treatment with 32 mg/day of methylprednisolone tablets, 100 mg/day of cyclosporine tablets, and 4 mg/day of candesartan.

At the one-month outpatient follow-up after discharge, the patient reported no episodes of bleeding. Complete blood count results were within normal limits, with a hemoglobin level of 11.3 g/dL and a platelet count of 192,000/mm³. The follow-up urinalysis revealed 3+ proteinuria and trace erythrocytes. Steroid therapy was gradually tapered to 4 mg/day, and the dose of candesartan was increased to 8 mg/day.

3. Discussion

Hematologic involvement is common in newly diagnosed patients with SLE, with anemia being one of the most frequently observed abnormalities. The types of anemia seen in SLE include anemia of chronic disease, iron deficiency anemia, and hemolytic anemia [4, 6]. Anemia of chronic disease in SLE is marked by normocytic, normochromic anemia with low serum iron levels, low transferrin, and normal to elevated serum ferritin levels. Hemolytic anemia, meanwhile, should be suspected based on the presence of elevated indirect bilirubin, lactate dehydrogenase, and reticulocyte count, along with decreased serum haptoglobin levels. In hemolytic anemia, the Coombs test is usually positive and is mediated by immunoglobulin G anti-erythrocyte antibodies. Additionally, a peripheral blood smear often reveals spherocytosis [7].

In the present case, based on the clinical findings and the nature of the disease, severe anemia was most likely aggravated by excessive bleeding secondary to severe thrombocytopenia. However, due to limited resources, immune-mediated causes could not be definitively confirmed. The initial clinical and laboratory evaluations did not support hemolytic anemia, and therefore, no additional diagnostic testing was performed.

Thrombocytopenia is another common hematologic finding in SLE. However, severe thrombocytopenia, defined as a platelet count below 50,000/mm³, is relatively rare, presenting in only 10% of cases. The most common cause is immune thrombocytopenia [4]. Other causes include decreased platelet production in the bone marrow and hypersplenism. Most immune-related causes involve antiplatelet antibodies, with others resulting from more complex mechanisms, such as interactions with antiphospholipid antibodies [4, 8]. In pregnancy, the causes of thrombocytopenia vary in each trimester. Gestational thrombocytopenia is the most frequent in later stages of pregnancy, while immune-related causes are frequently seen in the first trimester [9].

The patient in the present case was in her first trimester of pregnancy when she was diagnosed with SLE and developed severe thrombocytopenia. Three weeks prior, laboratory examinations during antenatal care showed normal platelet counts. The patient had no organomegaly. Based on these clinical findings, the most likely cause of thrombocytopenia in this case was increased peripheral destruction due to immune thrombocytopenia.

Platelet counts below 20,000/mm³ or the presence of active bleeding are indications for immunosuppressive treatment in patients with thrombocytopenia associated with SLE [10]. Both international and national guidelines recommend initiating treatment for severe autoimmune thrombocytopenia in SLE with high-dose corticosteroids, particularly pulse intravenous methylprednisolone, either alone or in combination with intravenous immunoglobulin (IVIG),

rituximab, or cyclophosphamide during the acute phase [6, 11]. However, most treatment guidelines do not specifically address thrombocytopenia management during pregnancy. This gap in the guidelines presents a critical issue, as consideration must be given to both the management of disease activity and the possible harm to the fetus when selecting a treatment plan [8]. Despite this, some general principles for managing SLE in pregnancy are available. All pregnant women with SLE should receive hydroxychloroquine unless contraindicated, and corticosteroid doses should be maintained below 20 mg/day of prednisone [3, 12]. Notably, though, for pregnant women with high disease activity and in need of therapy, starting or continuing treatments with the appropriate dose of steroids is recommended, as high disease activity and continuous prescription of high-dose steroids could affect pregnancy outcomes [12]. IVIG may be administered during the acute phase if there is an inadequate response to steroids and can also be considered in patients with contraindications to corticosteroids, such as uncontrolled diabetes, psychosis, or acute infection [6, 10]. For patients who do not respond to steroids—defined as the failure to achieve platelet counts above 50,000/mm³—or who experience relapse, rituximab should be considered, especially if cyclophosphamide has already been administered. The early initiation of steroid-sparing agents, including azathioprine, mycophenolate mofetil, and cyclosporine, is also recommended to facilitate dose reduction of steroid [10].

Of the drugs mentioned above, azathioprine and cyclosporine are considered safe for use during pregnancy; mycophenolate mofetil, however, is contraindicated and should be discontinued prior to conception [3, 12]. Rituximab also presents potential problems in pregnant patients, as it can cross the placenta and impair neonatal B-lymphocyte development, potentially leading to prolonged lymphopenia in the newborn. Additionally, maternal use of rituximab is associated with an increased risk of infection in both the mother and infant [8]. Therefore, rituximab should only be given in life- or organ-threatening conditions. Cyclophosphamide should also be discontinued before conception and should only be used in the second or third trimester for life- or organ-threatening conditions [3, 12]. Meanwhile, thrombopoietin receptor agonists (TPO-RAs) or a splenectomy may be considered when other treatment options fail [10]. TPO-RAs are preferred over a splenectomy due to the potential complications and long-term sequelae of the latter. TPO-RAs can, however, increase the risk of thromboembolism and should therefore be avoided in patients with antiphospholipid antibodies [6]. Platelet transfusion is generally not recommended for immune thrombocytopenia but may be considered in cases of life-threatening bleeding or when emergency cesarean delivery is indicated [8]. National guidelines in Indonesia allow for platelet transfusion in patients with platelet counts below 10,000/mm³ or those undergoing invasive procedures [6].

In this case, the patient presented with active bleeding and platelet counts below 20,000/mm³. She was treated with pulse-dose intravenous methylprednisolone (500 mg/day) for three days, followed by 62.5 mg/day of methylprednisolone injection for six days. Cyclosporine, as a steroid-sparing agent, was initiated immediately after the pulse steroid therapy. During hospitalization, TC transfusions were administered only when the patient's platelet counts dropped below 10,000/mm³ or in the presence of active bleeding. Throughout treatment until near the time of delivery, the platelet counts remained below 50,000/mm³. IVIG could not be administered due to limited resources. Rituximab and cyclophosphamide were not prescribed because of the patient's pregnancy.

If a patient requires emergency surgery, the procedure may be performed immediately, even if the patient is undergoing immunosuppressive therapy [13, 14]. Continuous glucocorticoid use can suppress the hypothalamic-pituitary-adrenal axis, reducing endogenous cortisol production. Specifically, surgery induces physiological stress, which increases the body's demand for cortisol. Depending on the type of surgery, corticosteroid supplementation may be given [6]. Stress-dose corticosteroid administration is based on the estimated daily cortisol secretion relative to the invasiveness of the procedure. For superficial procedures, such as dental surgery or biopsy, the patient's usual daily dose is typically sufficient. For major surgeries, such as esophagectomy or labor, a supplemental dose of 100 mg hydrocortisone intravenously should be administered and followed by a continuous infusion of 200 mg hydrocortisone over 24 hours, or 50 mg every 8 hours for 24 hours. Uterine curettage is considered a

minor procedure, for which 50 mg of hydrocortisone intravenously before the incision, in addition to the regular daily dose, is recommended [15]. Vaginal delivery is considered safe at a platelet count of 50,000/mm³ or more, whereas spinal or epidural anesthesia and cesarean section require a platelet count of 75,000/mm³ or more [6].

In this case, the patient had an incomplete abortion and was scheduled for uterine curettage under general anesthesia. Prior to the procedure, she received a stress dose of 62.5 mg intravenous methylprednisolone. During the procedure, 10 units of TC were transfused, as her preceding CBC showed a platelet count of 37,000/mm³. As noted earlier, TC transfusion is generally contraindicated in immune thrombocytopenia, except when invasive procedures are planned.

Pregnant women with SLE are at an increased risk of poor pregnancy outcomes, including a prolonged hospital stay, hypertension, intrauterine growth restriction, and a higher rate of cesarean delivery. Additionally, pregnant women with SLE have an elevated risk of preeclampsia, infection, thrombosis, and maternal mortality [3]. Fetal complications in SLE include fetal death, prematurity, fetal growth retardation, stillbirth, low birth weight, and neonatal lupus [6]. Fetal death has been reported in up to 43% of pregnancies in women with SLE, and the risk of fetal loss is approximately twice as high compared to non-SLE pregnancies [3]. Although less common than anemia, thrombocytopenia during pregnancy in women with SLE is associated with higher disease activity, early-onset preeclampsia, and an increased risk of pregnancy loss [16]. Other risk factors for poor pregnancy outcomes in SLE include the presence of lupus anticoagulant, antiphospholipid antibodies, low complements, use of antihypertensive medications, flares, active disease—particularly lupus nephritis—and a reduced rise in C3 levels [3, 16].

In this case, refractory thrombocytopenia likely contributed to both the poor pregnancy outcome and increased disease activity. Hematologic abnormalities were among the earliest clinical manifestations. As the disease progressed, significant proteinuria was observed, reaching 3.75 g/day. The combination of thrombocytopenia and active lupus nephritis further complicated the pregnancy and ultimately led to fetal loss.

The management of SLE with severe hematologic manifestations during early pregnancy is particularly challenging, especially in resource-limited settings. In this case, the limited availability of certain treatment options, such as IVIG, necessitated a more individualized therapeutic approach. The limited guidance available for thrombocytopenia management in pregnancy also required clinicians to rely on clinical judgment while balancing maternal and fetal risks. The timely involvement of a multidisciplinary team—including rheumatologists, obstetricians, anesthesiologists, and medicolegal specialists—was essential for making informed decisions regarding therapy and the safety of invasive procedures, including the decision to terminate the pregnancy. This case highlights the importance of collaborative decision-making in complex cases and reinforces the need for clearer clinical pathways to optimize maternal outcomes in similar settings.

4. Conclusion

A 30-year-old woman in early pregnancy came to the ER with nasal and gingival bleeding due to severe thrombocytopenia. Laboratory examinations revealed that the severe thrombocytopenia was secondary to SLE. Despite treatment with pulse doses of intravenous methylprednisolone and an early initiation of cyclosporine as a steroid-sparing agent, the platelet count could not be maintained above 50,000/mm³. A multidisciplinary discussion concluded that pregnancy termination was necessary. During preparation, the patient had an incomplete abortion requiring emergency uterine curettage. She received a stress dose of corticosteroids and TC transfusion during the procedure. Following the pregnancy termination, her platelet counts gradually improved, and she was discharged with oral corticosteroids and cyclosporine.

Abbreviations:

ANA – antinuclear antibody, CBC – complete blood counts, dL – deciliter, ER – emergency room, g – gram, IVIG – intravenous immunoglobulin, kg – kilogram, mg – milligram, mm – millimeter, PRC – packed red cell, SLE – systemic lupus erythematosus, SLEDAI – Systemic Lupus Erythematosus Disease Activity Index, TC – thrombocyte concentrate, TPO-RA – thrombopoietin receptor agonist.

Institutional Review Board Statement:

The study was reviewed and approved by the Ethics Committee of RSUD Dr Soetomo General Academic Hospital (Approval No. 2120/LOE/301.4.2/IX/2025).

Transparency:

The authors confirm that the manuscript is an honest, accurate, and transparent account of the study; that no vital features of the study have been omitted; and that any discrepancies from the study as planned have been explained. This study followed all ethical practices during writing.

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