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Pre-eclampsia with new-onset systemic lupus erythematosus during pregnancy: A case report

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Abstract

BACKGROUND

New-onset systemic lupus erythematosus (SLE) during pregnancy and in the postpartum period is rare, especially when complicated with pre-eclampsia, which is difficult to diagnose accurately. Here, we report a patient with new-onset SLE and antiphospholipid syndrome during pregnancy, which presented as pre-eclampsia at admission.

CASE SUMMARY

A 28-year-old primigravid woman was admitted to our hospital in the 27th wk of gestation with the primary diagnosis of severe pre-eclampsia. Although spasmolysis and antihypertensive therapy were administered since admission, the 24-h proteinuria of the 2nd day after admission reached 10311.0 mg. In the 47th h of admission, immunologic examinations revealed increased levels of anti-double stranded DNA antibody, anti-nuclear antibody, anti-cardiolipin antibody, anti-Sjögren's syndrome-related antigen A antibody and anti-nucleosome antibody and decreased levels of complement C3 and C4. One hour later, ultrasonography of the lower limbs showed thrombus of the bilateral popliteal veins. The diagnosis of SLE and antiphospholipid syndrome was indicated. In the 54th h, the patient manifested with convulsion, dyspnea and blurred vision. Ten hours later, intrauterine death was revealed by ultrasonography. Emergent surgery consisting of inferior vena cava filter implantation and subsequent cesarean section was performed. Following glucocorticoid and anticoagulation therapy after delivery, the patient had an optimal response with improvements in symptoms and immunological markers.

CONCLUSION

Obstetricians should be aware of the symptoms and immunological examination results to distinguish pre-eclampsia and underlying SLE for optimal pregnancy outcomes.

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Core tip: New-onset systemic lupus erythematosus during pregnancy and in the postpartum period is rare, especially when complicated with pre-eclampsia, which is difficult to diagnose accurately. We report a patient with new-onset systemic lupus erythematosus and antiphospholipid syndrome during pregnancy, which presented as pre-eclampsia at admission, and intrauterine death was revealed by ultrasonography. The patient showed improvements in symptoms and immunological markers after emergent surgery and drug therapy. Obstetricians should be aware of the symptoms and immunological examination results to distinguish pre-eclampsia and underlying systemic lupus erythematosus for optimal pregnancy outcomes.

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INTRODUCTION

Systemic lupus erythematosus (SLE) is a rare autoimmune connective tissue disease involving multiple systems with the incidence of 1 to 10 per 100000 person-years and the prevalence of 20 to 70 per 100000^[1]. Characterized by a strong female predisposition, SLE mainly affects women of reproductive age with a female-to-male ratio of approximately 9:1, which is largely thought to be due to the effect of female sex hormones on the immune system^[2]. Pregnant women with SLE have a higher risk of complications than non-pregnant women. The risk of pre-eclampsia in pregnant women with SLE is 3-5 times higher, and SLE complicated with pre-eclampsia accounts for 16%-30% of all SLE pregnancies. Up to 25% of SLE patients will develop pre-eclampsia, although this ratio is only 5% in the general population^[3]. Distinguishing between pre-eclampsia and SLE is challenging as the clinical manifestations of pre-eclampsia can sometimes mimic SLE, and the management of the two conditions emphasizes the expectant delivery and medication, respectively. Here we present a case of pre-eclampsia complicated with new-onset SLE during pregnancy and review the literature of similar cases.

CASE PRESENTATION

Chief complaints

On January 8, 2019, a 28-year-old primigravid woman at 27th wk of gestation was admitted to our hospital with edema of both lower limbs for 4 d, elevated blood pressure (150/98 mmHg) and proteinuria (4+) for 1 d.

History of present illness

Examination results on the tenth week of gestation showed blood pressure of 110/60 mmHg, negative urine protein and a platelet count of $234 \times 10^9/L$. Antenatal checkup was conducted regularly and showed normal outcomes except for edema in both limbs, hypertension and proteinuria before admission.

History of past illness

There was no history of past illness.

Personal and family history

She was married without the history of pregnancy or contraception. Her spouse was healthy and her family history was unremarkable.

Physical examination upon admission

Physical examination revealed that her blood pressure was 141/90 mmHg,

temperature was 36.5 °C, pulse rate was 92 bpm and respiratory rate was 18 breaths/min. The uterine height was 24 cm and abdominal circumference was 96 cm. Fetal weight was estimated to be 800 g.

Laboratory examinations

On the 1st day of admission, routine blood examination showed a white blood cell count of $8.43 \times 10^9/L$, red blood cell count of $3.37 \times 10^{12}/L$ and platelet count of $86 \times 10^9/L$. An examination of blood coagulation function showed a D-dimer level of 1309 ng/mL (fibrinogen equivalent units). Liver and renal function examinations showed decreased albumin (33 g/L) and increased lactate dehydrogenase (345 U/L), urea nitrogen (9.1 mmol/L) and uric acid (539 μ mol/L) with normal levels of alanine transaminase (17 U/L) and aspartate aminotransferase (32 U/L).

Imaging examinations

Color Doppler ultrasound examination at admission showed a second trimester pregnancy equivalent to 26 wk of gestation as well as normal fetal movement and a fetal heart rate of 160 bpm. The fetal head was located at the uterine fundus. The placenta of Grade I was in the anterior uterine wall. The fetal weight was estimated as 790 g and conditions of the fetus growth were as following: biparietal diameter: 6.6 cm; femur length: 4.6cm; humeral length: 4.5 cm; head circumference: 24.8 cm; and abdominal circumference: 20.1 cm. Systolic/diastolic ratio of the umbilical artery and the fetal middle cerebral artery was 3.10 and 3.30, respectively. The amniotic fluid index was 12.9 cm.

FINAL DIAGNOSIS

The patient was primarily diagnosed with severe pre-eclampsia according to her clinical manifestations and laboratory results on admission.

TREATMENT

Other relevant examinations were completed after admission and medications including magnesium sulfate, labetalol and glucocorticoid were prescribed for spasmodic, antihypertension and promotion of fetal lung maturation. Magnesium sulfate was administered with the loading dose of 5.0 g, followed by the 15.0-20.0 g pumped every day upon admission. The patient's vital signs as well as fetal movement and heart rate were closely monitored to assess disease status. However, her blood pressure remained higher than normal with a maximum of 166/98 mmHg and the lower limbs showed obvious edema in the 25th h.

On the 3rd day morning, the 24-h proteinuria of the 2nd day after admission was reported as 10311.0 mg. We suspected there was an underlying autoimmune disease and then prescribed an immunological examination. In the afternoon of the 3rd day (47th h of admission), the lupus anticoagulant assays showed that the standardized ratio of dilute Russell's viper venom time and silica clotting time were increased at 2.40 and 2.06, respectively. The dsDNA antibody, ACL-immunoglobulin (Ig) G and ACL-IgM were also elevated (61.5, 76.5 and 83.9 IU/mL, respectively). Positivity for dsDNA antibody, anti-nuclear antibody, anti-Sjögren's syndrome-related antigen A antibody, anti-nucleosome antibody and decreased levels of complement C3 and C4 were also observed. One hour later, the ultrasonography of the lower limbs showed mural thrombus and stenosis of the bilateral popliteal veins. The underlying SLE flare and antiphospholipid syndrome were indicated and nadroparin was administered then.

However, the disease developed so rapidly and on the third night (54th h of admission), the fetal heart monitoring showed 110 bpm with a non-reactive pattern. Ten minutes later, the patient presented with blurred vision and involuntary convulsion of her entire body but was conscious and able to answer questions. Her vital signs at that time revealed a temperature of 37.1 °C, pulse rate of 94 bpm, respiratory rate of 17 breaths/min and elevated blood pressure of 172/102 mmHg. When the patient showed convulsion, magnesium sulfate of 1.5 g/h was pumped continuously and was administered with the purpose of spasmodic for 3 d since admission. We did not consider the superaddition of magnesium sulfate in case of magnesium poisoning. In addition, thrombosis in both lower limbs indicated to us that the convulsion may also be caused by a potential cerebral embolism and hemorrhage besides preeclampsia. So urapidil and mannitol were used to reduce blood pressure as well as intracranial pressure. Her blood pressure decreased to

166/100 mmHg with slight convulsions and blurred vision.

Meanwhile, we informed the patient and her families of the disease status and the following treatment plan. However, considering the small gestational age of 27⁺² wk and for fear of low infant survival rate, they did not have high expectations for the premature infant and decided not to deliver emergently. Furthermore, the head magnetic resonance imaging showed long T1 and T2 signals in the frontoparietal lobe, basal ganglia and pons, which indicated posterior reversible encephalopathy syndrome. Therefore, we monitored the maternal and fetal condition besides medications and consulted with the vascular surgeon for preparations of the inferior vena cava filter.

On the fourth morning (63rd h of admission), the blood biochemical testing and routine blood examination were carried out for further evaluation of the disease status, which indicated high lactic acid dehydrogenase of 318 U/L and increased white blood cell count of 10.07×10^9 /L and decreased red blood cell, hemoglobin and platelet count of 2.99×10^{12} /L, 101 g/L and 84×10^9 /L, respectively. One hour later (64th h of admission), the fetal heart was not detected, and emergent ultrasonography was performed, which showed intrauterine death and pleural effusion in the fetus with an estimated gestational age of 26⁺ wk. In the 68th h, the patient underwent surgery consisting of inferior vena cava filter implantation for prevention of the potential occurrence of pulmonary embolism and subsequent cesarean section followed by hydrocortisone therapy. The dead fetus weighing 980 g was delivered. Methylprednisolone was prescribed during the postoperative period along with magnesium sulfate and enoxaparin.

OUTCOME AND FOLLOW-UP

Laboratory testing on the third postpartum day revealed an increased white blood cell count and decreased red blood cell count, hemoglobin, platelet count and prothrombin time. On the sixth postoperative day, 24-h urinary protein decreased to 8463.0 mg and complement C3 was slightly lower than normal at 68.10 mg/dL with normal C4 and anti-dsDNA, which improved significantly compared with antepartum values. The patient's hypertension improved and was in the normal range without recurrence of convulsions. Oral prednisone acetate (50 mg) was continued for SLE control. The patient was discharged on the 13th postoperative day with further follow-up in the Department of Rheumatology.

DISCUSSION

The relationship between pregnancy and SLE is a matter of great concern and is mainly related to the effect of pregnancy on SLE and the impact of SLE on adverse pregnancy outcomes. Pregnancy is considered a high-risk period for patients with SLE, and there is no consensus on whether pregnancy increases the risk of SLE flares. Although the prognosis of patients with SLE has significantly improved and is no longer a contradiction for pregnancy due to continuous developments in obstetrics and rheumatology, pregnancy is still challenging with a high risk of maternal and fetal complications, such as hypertensive disorders, fetal loss, preterm birth, intrauterine growth retardation and neonatal lupus. Pre-eclampsia is one of the most common complications in pregnant patients with SLE. However, it is difficult to distinguish pre-eclampsia and active SLE in clinical practice as clinical manifestations such as hypertension, proteinuria and impaired renal function often occur in both conditions.

In the present study, we report a patient with pre-eclampsia as the initial manifestation of SLE during pregnancy. Such cases are rarely reported; thus, we comprehensively searched the relevant literature in PubMed from the database establishment to June 2019 and identified a total of eight published studies involving nine cases. The clinical characteristics and information on all included patients are summarized in Table 1. All of these patients were diagnosed with pre-eclampsia during pregnancy with new-onset SLE. Three patients (plus the present case)^[4,5] had SLE before delivery and six patients^[5-10] developed SLE in the postpartum period. In one study^[11], the time of SLE diagnosis was unclear, but the patient showed SLE-related manifestations immediately after delivery. The mean age of the included patients was 29 years and ranged from 20 to 35 years. The most common pregnancy outcome was preterm delivery, which occurred in six patients. In addition, two patients developed abortion, and two had term delivery. Only one case of intrauterine death was observed. Maternal death did not occur, but the following pregnancy

complications in addition to pre-eclampsia and SLE were reported: Eclampsia, acute fatty liver, HELLP syndrome, acute myocardial infarction, acute heart failure, pulmonary edema, lupus nephritis, vasculitis, thrombosis, dilated cardiomyopathy, nephrotic syndrome, thrombocytopenic purpura and antiphospholipid syndrome. Lupus nephritis is a common complication and indicates the high incidence of renal involvement. Besides positive immunological examinations, symptoms supporting the diagnosis of SLE were as follows: Fever, skin rash and macules, oral ulcer, arthritis, proteinuria, hematuria, anemia, thrombocytopenia, leukopenia, renal disorder, convulsion and thrombus.

Of the ten reported cases, only two patients^[4,5] were diagnosed with SLE prior to the diagnosis of pre-eclampsia, and pre-eclampsia-related symptoms were absent on initial admission. The remaining eight patients initially manifested with pre-eclampsia before the diagnosis of SLE. In these eight patients, the first symptoms, such as edema, proteinuria, hypertension and seizure, are common in pre-eclampsia, which made it difficult to distinguish the underlying SLE from pre-eclampsia. However, the typical manifestations of SLE (such as rash, arthritis or thrombus) and severe complications (such as vasculitis, thrombocytopenic purpura or lupus nephritis) occurred as the disease progressed and appeared as SLE. Furthermore, positive immunological test results (especially anti-dsDNA and antinuclear antibodies) further contributed to the diagnosis of SLE. In addition, as renal involvement often occurs in both pre-eclampsia and SLE, it is very important to distinguish between pre-eclampsia and renal damage caused by SLE. Patients with SLE often have a low level of C3 and increased dsDNA antibody in the blood, both of which are found in pre-eclampsia patients. Moreover, proteinuria rapidly decreases after delivery in pre-eclampsia, but is present or even increases in patients with renal disorder due to SLE.

Improvements in maternal and fetal outcomes in SLE patients have been achieved in the past few decades^[12], but is still a great challenge for both obstetricians and rheumatologists. Multidisciplinary coordination including a comprehensive assessment before conception, close monitoring during pregnancy as well as termination of pregnancy at the appropriate time is essential for optimal prognosis. Once a patient with SLE becomes pregnant, regular prenatal examinations and monitoring per the following are necessary for us to learn the disease process: (1) Whether there is the maternal clinical manifestations that indicate the SLE flare, such as fever, facial erythema, arthralgia or photosensitivity; (2) Regular blood, urine, hepatic, renal and immunological analysis including anti-nuclear antibody, anti-dsDNA, ACL lupus, complement C3 and C4 and lupus anticoagulant assays; and (3) Regular ultrasound for detection of the growth and heart developments of the intrauterine fetus.

In particular, when SLE is complicated with pre-eclampsia, it is crucial to differentiate these disorders as each condition requires different treatment. Strict attention to symptoms and immunological tests is necessary for a definite diagnosis and timely treatment. In our case, hypertension, edema in both legs and proteinuria led to the diagnosis of pre-eclampsia and treatment with spasmolysis and antihypertensive therapy. Immunological tests were conducted to determine whether underlying SLE was present in view of the patient's severe proteinuria. The unrelieved symptoms by treatment for pre-eclampsia and the results of immunological tests along with findings such as thrombosis and convulsions contributed to the diagnosis of an SLE flare and antiphospholipid syndrome. Fetal distress developed following suspected lupus encephalopathy. Regrettably, intrauterine death occurred even though an emergency cesarean section was performed. However, the patient had a good prognosis following postpartum glucocorticoid and anticoagulation therapy.

CONCLUSION

New-onset SLE during pregnancy and in the postpartum period is rare and difficult to differentiate from pre-eclampsia. Close attention should be paid to the symptoms, and timely immunological examinations should be performed, especially in patients with an initial diagnosis of pre-eclampsia and underlying SLE, which are essential for a rapid diagnosis and effective treatment.

Table 1 Case reports of pre-eclampsia with new-onset systemic lupus erythematosus during pregnancy

Ref.	Age in yr, gravity, parity	Initial symptoms	Onset time of symptoms	Time of pre-eclampsia diagnosis	Symptoms supporting SLE diagnosis	Time of SLE diagnosis	Positive antibodies	Other pregnancy complications	Treatment of SLE	Fetal outcome	Birth gestation, fetal weight in g, and delivery method
Minakami <i>et al</i> ^[4] , 1994	34 G4P2	Facial rash	Early first trimester	NS	Facial rash, discoid rash, oral ulcer, arthritis, proteinuria	Before delivery	Anti-dsDNA, anti-Sm, ANA	Eclampsia, acute fatty liver, AMI	NS	NS	14 wk, Supravaginal amputation
Hildbrand <i>et al</i> ^[11] , 2005	34 G5P3	Severe dyspnea, hypertension, acute left heart failure	Immediately after delivery	NS	Anemia, thrombocytopenia, proteinuria, acute heart failure, renal disorder	NS	Anti-dsDNA, ANA, anti-cardiolipin	Acute heart failure	Prednisone, cyclophosphamide	Live birth	36 wk, 1640 g, NS
Yang <i>et al</i> ^[5] , 2006	22 G1P0	Facial malar rash, cold fingers, high fever	17 wk of gestation	Early third trimester	Facial malar rash, leukopenia, proteinuria	Before delivery	Anti-dsDNA, ANA	NS	Prednisolone, hydroxychloroquine, methylprednisolone, azathioprine, colchicine	Live birth	33 wk, 1420 g, cesarean section
	31 G1P0	Skin rashes, lower leg edema, dizziness, palpitation, leukopenia	26 wk of gestation	38 wk of gestation	Skin rash, leukopenia, proteinuria, pulmonary edema	After delivery	Anti-dsDNA, ANA	Pulmonary edema after delivery	Prednisolone, azathioprine, hydroxychloroquine, methylprednisolone, azathioprine	Live birth	38 wk, 3542 g, cesarean section
Matsuo <i>et al</i> ^[6] , 2007	27 NS	proteinuria, pre-eclampsia	17 wk of gestation	31 wk of gestation	Continuous proteinuria, hematuria	12 mo after delivery	Anti-dsDNA, ANA	Lupus nephritis	Prednisolone, cyclosporine	Live birth	31 wk, 1214 g, cesarean section
Borahay <i>et al</i> ^[7] , 2009	20 G1P0	Hypertension, headache, history of seizure	39 wk of gestation	39 wk of gestation	Fever, purpuric macules at the fingers, leukopenia, anemia, proteinuria	> 18 d after delivery	Anti-dsDNA, ANA	Leukocytoclastic vasculitis, seizure, partial thrombosis of the proximal left transverse sinus	Methylprednisolone, enoxaparin, prednisone, hydroxychloroquine	Live birth	39 wk, NS
Stepanková <i>et al</i> ^[8] , 2009	26 G1P0	Pre-eclampsia, HELLP syndrome	31 wk and 2 d of gestation	31 wk and 2 d of gestation	Proteinuria, anemia, thrombocytopenia, fever	> 3 wk after delivery	Anti-dsDNA, ANA	HELLP syndrome, dilated cardiomyopathy, NYHA class 3–4, lupus nephritis	Plasma exchange, steroids, cyclophosphamide, mycophenolate mofetil	Live birth	31 wk and 2 d, 1900 g cesarean section
Miyamoto <i>et al</i> ^[9] , 2014	32 G1P0	Pre-eclampsia	29 wk of gestation	29 wk of gestation	Thrombocytopenia, proteinuria, anemia	5 th day after delivery	Anti-dsDNA, ANA, anti-Sm, anti-RNP	NS	Prednisone	Live birth	29 wk, 1048 g, cesarean section

Karachalios 2018	35 G2P1	Edema in both legs, face and neck, history of RA for 6 yr and gestational hypothyroidism	34 wk plus 6 d of gestation	After delivery	Anemia, hypertension, microscopic hematuria, proteinuria, respiratory alkalosis, bilateral pleural effusion	6 th day after delivery	anti-dsDNA, ANA	Postpartum nephrotic syndrome, lupus nephritis	Cyclophosphamide, azathioprine, methylprednisolone	Live birth	34 wk and 6 d, 2340 g, cesarean section
Current case	28 G1P0	Hypertension, edema in both legs, proteinuria	27 wk of gestation	27 wk of gestation	Anemia, fever, convulsion, dyspnea, blurred vision, thrombus in bilateral popliteal veins, renal disorder	Before delivery	anti-dsDNA, ANA, anti-cardiolipin, anti-SSA, AnuA	Lupus nephritis, antiphospholipid syndrome	Nadroparin, Enoxaparin, hydrocortisone, methylprednisolone, prednisone	Intrauterine death	27 wk and 3 d, 980 g, cesarean section

AMI: Acute myocardial infarction; ANA: Anti-nuclear antibody; AnuA: Anti-nucleosome antibody; anti-SSA: Anti-Sjögren's syndrome-related antigen A antibody; NS: Not stated; RA: Rheumatoid arthritis; RNP: Ribonuclear protein; SLE: Systemic lupus erythematosus; NYHA: New York Heart Association.

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