Systemic lupus erythematosus and worsening severe preeclampsia: Does it have correlation?

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Abstract. Acute exacerbation of systemic lupus erythematosus in pregnancy complicated by severe preeclampsia was difficult to diagnose and treat. This is the case of Mrs Y, 26 years old in 24 weeks gestational age with severe preeclampsia with active exacerbation of systemic lupus erythematosus. She had suffered from systemic lupus eryhtematosus for three years before she got pregnant. When she came the blood pressure was 180/110 mmHg, protein urine +3. There was complaint of stiffness on muscle joints, facial flares, general weakness, and photosensitivity. She was hospitalized for seven days before she was discharged. Her blood pressure was controlled and lupus symptoms were controlled by antihypertensive agents and antiinflamation agent metil prednisolone. A week later she came back to our hospital. Her blood pressure was 190/110 mmHg despite having antihypertensive. She also complaint of joints ache, general weakness, facial flares, and no fetal movement. On ultrasound examination there was intra uterine fetal death. From this case, we would like to assess the patophysiology of systemic lupus erythematosus and worsening symptoms of severe preeclampsia which cause the mortality and morbidity of mother and baby.
Keywords: systemic lupus erythematosus, severe preeclampsia, acute exacerbation

Introduction

Systemic lupus erythematosus (SLE) is a chronic,inflammatory multiorgan autoimmune disorder characterized by periods of remissions and relapse. The patophysiology of SLE is as follows.Antigen-antibody complexes are formed with a resulting secondary inflammatory response. The combination of the immune complexes and the secondary inflammatory response within the glomerulus leads to irreversible renal damage. Deposits also occur within the skin and other endothelial surface. During pregnancy, the most common clinical manifestations include arthralgia, fever, skin lesions and renal disease. SLE has effect on pregnancy while pregnancy also exacerbates the symptoms of SLE. There is increased rate of pregnancy loss in patients with SLE. The most important factor associated with SLE and pregnancy loss is the presence of antiphospholipid antibodies, other factors include renal disease, disease activity, and history of previous pregnancy loss. Antiphospholipid antibody, renal disease, and preeclampsia are risk factors for SLE. This paper is purposed to assess and review the patophysiology of systemic lupus erythematosus and its correlation with severe preeclampsia.

Material and Methods

This is the case of Mrs Y, 26 years old, had been suffering from systemic lupus erythematosus for 3 years while she got pregnant. She came to our hospital with chief complaint of malar rash, joint stiffness in her 24 weeks of pregnancy. She did antenatal care at obgyn every month. When she came, her blood pressure was 180/110 mmHg, protein urine +3. From ultrasound examination there was intra uterine singleton live fetus with severe oligohydramnion. We informed her and her family. They chose to conserve the pregnancy and denied the idea of pregnancy termination since it was her first pregnancy. She was hospitalized for seven days before she was discharged. Her blood pressure was controlled and lupus symptoms were controlled by antihypertensive agents and antiinflamation agent metil prednisolone. A week later she came back to our hospital. Her blood pressure was 190/110 mmHg despite having antihypertensive. She also complaint of joints ache, general weakness, facial flares, and no fetal movement. On ultrasound examination there was intra uterine fetal death. Laboratory finding showed hemoglobin 12.3 g/dl, leukocyte 11.500 /UL, thrombocyte 71.000 /UL, increased level of ureum and creatinine, increased level of liver enzyme AST and ALT, the albumin level 1.9 g/dl. The peripheral blood smear showed anisocytosis, Burr cell (+) and leococytosis. Immunoserology showed Ig M and Ig G ACA was within normal limit. There was also disfunction in hemostasis which showed slightly longer coagulation test prothrombine time and activated partial prothrombine time as well as significantly evelated D-dimer 3594 ng/ml exceeding the normal value of 500 ng/ml. Regretfully we had to terminate her pregnancy by pregnancy induction. By inducing pregnancy, born baby boy, 870 grams, died maserated grade II.

Results and Discussion

Systemic lupus erythematosus was chronic autoimmune disorder. Effect of pregnancy due to increased level of estrogen can create acute exacerbation of pregnancy. It was difficult to distinguish between acute exacerbation of SLE in early pregnancy and early onset of preeclampsia because both of them may have the manisfestation of hypertension, proteinuria, renal function deterioration, leucopenia, thrombocytopenia, as well as edema. Those characteristic presented in that patient in 24 weeks of gestational age. The strategy to distinguish between acute exacerbation and worsening of severe preeclampsia in patient with SLE who developed hypertension was the presence of hemolysis and elevated liver enzymes. It was shown in this patient that there was leukocytosis and the presence of Burr cell and anisocytosis in peripheral blood smear. It was highly suggestive that this patient suffer from severe preeclampsia exaggerated by HELLP syndrome. Patients with SLE have high risk of developing preeclampsia wih incidence of 15-32% with risk factors lupus nephritis, antiphospholipid syndrome (APS), and chronic hypertension. SLE can increase the risk of pregnancy loss due to hypertension, placental insufficiency, as well as may cause fetal and neonatal heart block. The patient in this study also suffered from acute exacerbation of SLE characterized by the presence of malar rash, joint ache, stomatitis, and fatigue syndrome. There was severe hypertension which could not be controlled by medication, proteinuria, renal function deterioration, hypoalbuminemia. There was also coagulation disorder with increased level of fibrinogen degradation products ex D-dimer which was suggestive of the disseminated intravascular coagulation (DIC).

The patient in this study suffered from acute exacerbation of SLE which was compromised by many complications such as severe preeclampsia with uncontrolled hypertension, HELLP syndrome, and DIC. There was massive endothelial destruction with comsumptive coagulopathy which caused DIC. Because there was manifestation of severe preeclampsia, HELLP syndrome, the cause of intra uterine fetal death was because of severe placental insufficiency. The best management for this patient was termination of pregnancy. Severe endothelial destruction can deteriorate organs such as heart, vascular, renal, liver, and central nervous system. T would create bad prognosis for mother if pregnancy was prolonged. We still could not exclude the presence of APS although Ig M and Ig G ACA were within normal limit, because it should be added by Ig M and Ig G beta 2 glicoprotein examination which was quite expensive and unavailable in our center.

Conclusions

Patient with acute exacerbation of SLE complicated by severe preeclampsia, HELLP syndrome, was due to placental insufficiency. It created worse prognosis for the fetus.

References

1. Clowse ME, Jamison M, Myer E, James AH. A national study of the complications of lupus in pregnancy. Am J Obstet Gynecol 2008; 199: 127.e1-127.e6.
2. Harnett M, McElrath T. Autoimmune Disease. Dalam: Datta S (editor). Anesthetic and Obstetric Management of High Risk Pregnancy. 3rd Edition. 2006. USA: Spinger –Verlag.
3. Leguizamon GF, Reece EA. Rheumatologic and Connective Tissue Disordes in Pregnancy. Dalam: Reece EA, Hobbins JC (editor). Clinical Obstetrics: The Fetus and The Mother. 3rd Edition. 2007. UK: Blackwell Publishing
4. Yuen SY, Krizova A, Ouimet JM, Pope JE. Pregnancy outcome in systemic lupus erythematosus is improving: results from a case control study and literature review. The Open Rheumatology Journal 2008; 2: 89-98.