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SLE and Pregnancy

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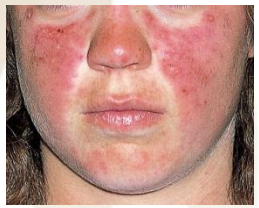
Introduction

Systemic lupus erythematosus (SLE) is a life-long, life-threatening autoimmune disease which can affect any organ in the body (Marks & Tullus, 2011). SLE disproportionately affects women in a ratio of 9:1 compared to men with most women being affected during child-bearing age (15-50 years) (Ferenkeh-Koroma, 2012). Pregnancy represents a challenge for the patient with SLE. Pregnant patients with SLE are considered high-risk for multiple medical and obstetric complications, as flares are related to increased irreversible organ damage (Ateka-Barrutia & Khamashta, 2013). Severe flares are also associated with poor fetal outcomes (Peart & Clowse, 2014). Successful pregnancies happen in 67% of women with lupus compared to 85% in the general population (Ferenkeh-Koroma, 2012). There is a 20-fold risk in maternal mortality and an increased rate of hypertension, pre-gestational diabetes, renal impairment, pulmonary hypertension, major infections, thrombotic events, and other hematological complications in patients with lupus (Ateka-Barrutia & Khamashta, 2013). There is a higher risk for preterm labor in patients with lupus with 25% of pregnancies resulting in delivery before 37 weeks gestation (Ferenkeh-Koroma, 2012). There is also a greater risk for pre-eclampsia, intrauterine growth restriction (IUGR) and cesarean section (Ateka-Barrutia & Khamashta, 2013).



Common butterfly rash in SLE
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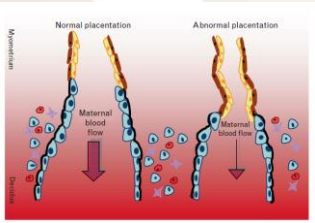
Pathophysiology

In SLE, the body produces auto-antibodies which attack healthy cells, tissues and organs (Ferenkeh-Koroma, 2012). "Common symptoms of lupus include extreme fatigue, kidney problems, painful or swollen joints and skin rashes" (Ferenkeh-Koroma, 2012, p. 49). A combination of genetic, environmental, immunoregulatory, hormonal, and epigenetic factors play a role in the predisposition and development of SLE (Tsokos, 2011). As with most autoimmune diseases, SLE is characterized by disease flares followed by periods of remission (Nalli et al., 2014).

SLE is caused by a complex assortment of immune abnormalities. Overactive B cells, abnormally activated T cells and antigen-presenting cells results in the productions of inflammatory cytokines, apoptotic cells, diverse autoantibodies and immune complexes (Marks & Tullus, 2011). Deposits of immune complexes are the main cause of organ injury in SLE. Immune complexes are formed in large amounts as antinuclear antibodies and bind to nuclear material in blood and tissues, but cannot be cleared easily because the Fc and complement receptors are deficient (Tsokos, 2011). The immune complexes can bind to tissue specific cells which triggers the complement cascade and other mediators of inflammation (Tsokos, 2011).



Decreased blood flow through placental spiral arteries in SLE
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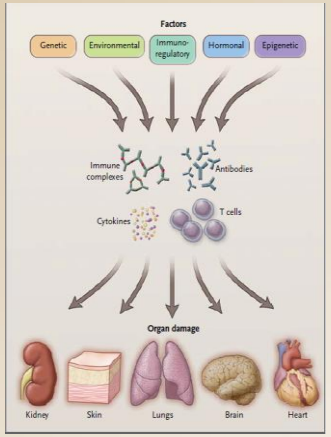

Pathogenesis of SLE
(Tsokos, 2011, p. 2112)

During pregnancy, the health of the fetus is dependent upon the health of the mother. An unhealthy mother can inhibit her ability to carry a pregnancy to full-term. Uterine-placental insufficiency caused by poor vascularization contributes to multiple pregnancy complications (Ostensen & Clowse, 2013). Endothelial dysfunction and cell damage leading to poor placental blood flow is the main cause of IUGR and possibly early preeclampsia (Ostensen & Clowse, 2013). Preterm labor may be triggered by several different routes in the SLE patient. The most significant predictor of preterm birth is lupus activity during the course of the pregnancy (Ostensen & Clowse, 2013). Inflammation from infection, oral prednisone, and elevated anti-dsDNA and hypocomplementemia are also associated with preterm birth (Ostensen & Clowse, 2013). Other antibodies, such as antiphospholipid antibodies, are associated with venous and arterial thrombosis and miscarriage, fetal death and preterm births (Stanhope, White, Moder, Smyth, & Garovic, 2012).

Significance of Pathology

There are 20-150 cases of SLE reported per 100,000 people with the highest prevalence in Brazil (Tsokos, 2011). In the U.S., people of African, Hispanic and Asian ancestry have a higher incidence than those of European ancestry (Tsokos, 2011). Multidisciplinary management of the pregnant SLE patient has improved disease management and pregnancy outcomes dramatically over the last 20 years (Ateka-Barrutia & Khamashta, 2013).

Many antibodies are present and measured in patients with SLE. Autoantibodies including antinuclear antibody (ANA), anti-double stranded (ds) DNA and anti-Smith have been found in 88% of SLE patients up to 9.4 years before the patient is ever diagnosed with SLE (Marks & Tullus, 2011). Anti-dsDNA antibodies are particularly important because they have a high specificity for disease activity (Marks & Tullus, 2011). Antiphospholipid antibodies are present in 30-40% of women with SLE (Tower, Mathen, Crocker, & Bruce, 2013). The presence of these antibodies places the pregnant SLE patient at greater risk for poor outcomes with approximately one third developing thrombotic events and miscarriages (Ferenkeh-Koroma, 2012). Low dose aspirin or low molecular-weight heparin can significantly decrease complications resulting from antiphospholipid antibodies and increase the live birth rate by sevenfold (Ferenkeh-Koroma, 2012).

SLE can also have an effect on the growing fetus in the form of neonatal lupus (NL). NL is mediated by maternal anti-Ro/SSA and anti-La/SSB antibodies which can cross the placenta affecting the fetus (Nalli et al., 2013). Only 1% of infants will develop NL with the majority having cutaneous lesions that are photosensitive to direct sunlight (Johnson, 2014). However, congenital heart block (CHB) is the most severe form of NL with a 10-20% perinatal death rate and most surviving children in need of a permanent pacemaker (Ateka-Barrutia & Khamashta, 2013).

Treatments typically include topical applications for skin rash, NSAIDS, corticosteroids, immunosuppressants, and antimalarial drugs (Ferenkeh-Koroma, 2012). Management is based on symptoms and the degree of organ involvement and the treatment regimen may change relative to flares and remissions (Ferenkeh-Koroma, 2012).

Nursing Considerations

Due to the various and vast effects SLE can have on the body, pregnancy represents an incredible challenge for healthcare providers. Preconception counseling is recommended for women with SLE hoping to become pregnant. It is encouraged for the woman to not get pregnant until the disease has been in remission for at least 6 months as this is associated with better outcomes for the mother and infant (Nalli et al., 2013). A multidisciplinary approach including an obstetrician experienced in high risk pregnancies in collaboration with a rheumatologist familiar with SLE should be utilized for the management of the patient (Ferenkeh-Koroma, 2012). Screenings for antibodies such as the ANA, anti-Ro/La, anti-dsDNA, and ASA should be done during the initial evaluation of the pregnant SLE patient. Anti-dsDNA and hypocomplementemia also have been proven to be very helpful in determining pregnancy risks in a clinically active SLE (Clowse, Magder, & Petri, 2011). Most lupus flares during pregnancy occur during the first trimester and up to 30% may have flares between 2-8 weeks postpartum (Ferenkeh-Koroma, 2012). Treatment for SLE during pregnancy is critical and varies on a case by case basis. Multiple medications may be used, although some are contraindicated during pregnancy. Poor control of the disease during pregnancy may have damaging effects on the outcome for the mother and infant (Nalli, et al., 2013). It is encouraging, though, that most women who become pregnant during remission can expect a normal pregnancy without any major complications (Ferenkeh- Koroma, 2012). Close monitoring and management of the pregnant SLE patient is imperative to ensure the best possible outcome for the mother and infant.

Conclusion

The exact etiopathogenesis of SLE is still unknown and there is no gold standard of treatment for the autoimmune disease. Female patients diagnosed with SLE should be counseled on the risks of pregnancy as the majority of SLE patients are women of child-bearing age. Since powerful immunosuppressants and cytotoxic drugs are used for treatment of SLE, pregnancy presents a unique challenge for medical management. Although pregnancy places the patient at higher risk for complications, most patients can have successful pregnancies. Providers must have a good understanding of the disease and provide patients with appropriate education and support to enable SLE patients to maintain wellbeing and lead active lives (Ferenkeh-Koroma, 2012).

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References

Ateka-Barrutia, O., & Khamashta, M. A. (2013). The challenge of pregnancy for patients with SLE. *Lupus*, *22*(12), 1295-1308. doi:10.1177/0961203313504637

Clowse, M. B., Magder, L. S., & Petri, M. (2011). The clinical utility of measuring complement and anti-dsDNA antibodies during pregnancy in patients with systemic lupus erythematosus. *Journal of Rheumatology*, *38*(6), 1012-1016.doi:10.3899/jrheum.100746

Ferenkeh-Koroma, A. (2012). Systemic lupus erythematosus: nurse and patient education. *Nursing Standard*, *26*(39), 49-57.

Johnson, B. (2014). Overview of neonatal lupus. *Journal of Pediatric Healthcare*, *28*(4), 331-341. doi:10.1016/j.pedhc.2013.07.015

Marks, S., & Tullus, K. (2012). Autoantibodies in systemic lupus erythematosus. *Pediatric Nephrology*, *27*(10), 1855-1868. doi:10.1007/s00467-011-2078-4

Nalli, C., Iodice, A., Andreoli, L., Lojacono, A., Motta, M., Fazzi, E., & Tincani, A. (2014). The effects of lupus and antiphospholipid antibody syndrome on foetal outcomes. *Lupus*, *23*(6), 507-517. doi:10.1177/0961203313501402

Ostensen, M., & Clowse, M. (2013). Pathogenesis of pregnancy complications in systemic lupus erythematosus. *Current Opinion in Rheumatology*, *25*(5), 591-596. doi:10.1097/BOR.0b013e328363ebf7

Peart, E., & Clowse, M. B. (2014). Systemic lupus erythematosus and pregnancy outcomes: an update and review of the literature. *Current Opinion in Rheumatology*, *26*(2), 118-123. doi:10.1097/BOR.0000000000000030

Stanhope, T., White, W., Moder, K., Smyth, A., & Garovic, V. (2012). Obstetric nephrology: lupus and lupus nephritis in pregnancy. *Clinical Journal of the American Society of Nephrology*, *7*(12), 2089-2099. doi:10.2215/CJN.12441211

Tower, C., Mathen, S., Crocker, I., & Bruce, I. N. (2013). Regulatory T cells in systemic lupus erythematosus and pregnancy. *American Journal of Reproductive Immunology (New York, N.Y.: 1989)*, *69*(6), 588-595. doi:10.1111/aji.12081

Tsokos, G. (2011). Systemic lupus erythematosus. *New England Journal of Medicine*, *365*(22), 2110-2121.