Summer 2015

SLE and Pregnancy

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Introduction

Systemic lupus erythematosus (SLE) is a life-long, life-threatening autoimmune disease that can affect any organ in the body (Marks & Tullus, 2012). SLE disproportionately affects women in a ratio of 9:1 compared to men, with most women being affected during their child-bearing age (15-50 years) (Ferenkeh & Khamashta, 2013). Pregnancy represents a challenge for the patient with SLE. Pregnant women with SLE are at increased risk for multiple medical and obstetric complications, as flares are related to increased irreversible organ damage (Ateka-Barrutia & Khamashta, 2015). Severe flares are also associated with poor fetal outcomes (Pearl & Clowse, 2014). Successful pregnancies happen in 67% of women with lupus compared to 85% in the general population (Ferenkeh & Khamashta, 2012). There is a 20-fold risk in maternal mortality and an increased rate of hypertension, pre-eclampsia, gestational diabetes, renal impairment, pulmonary hypertension, major infections, thrombotic events, and other hematologic complications in patients with lupus (Ateka-Barrutia & Khamashta, 2013). This is particularly true for the immune labor in patients with lupus with 25% of pregnant women dying in delivery between 37 weeks, gestation and 39 weeks, in 2012. There is also a greater risk for pre-eclampsia, intraventricular hemorrhage (IVH), and ovarian cysts (Ateka-Barrutia & Khamashta, 2015).

Pathophysiology

In SLE, the body produces auto-antibodies which attack healthy cells, tissues and organs (Ferenkeh & Khamashta, 2012). “Common symptoms of lupus include extreme fatigue, kidney problems, pain or swelling in joint and skin rashes” (Ferenkeh & Khamashta, 2012). A combination of genetic, environmental, immunoregulatory, hormonal, and epigenetic factors play a role in the perpetuation and development of SLE (Tsokos, 2011). As with most autoimmune diseases, SLE is characterized by disease flares followed by periods of remission (Hall et al., 2014). SLE is caused by a complex assortment of immune abnormalities. Discoidal flares, abnormally activated T cells and antigen presenting cells results in the production of inflammatory cytokines, acutologic 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 receptor are defected. There is a higher risk for the immune complex disease in SLE patients compared to tissue specific cell death which triggers the complement cascade and other mediators of inflammation (Tsokos, 2011).

During pregnancy, the health of the fetus is dependent upon the health of the mother. An unhealthy mother can inhibit the ability for any pregnancy to fulfill the uterine-placental insufficiency caused by poor vascularization, contribute to multiple pregnancy complications (Ostensen & Cloowe, 2013). Endothelial dysfunction and cell damage leading to poor placental blood flow is the main cause of IUGR and possibly early pre-eclampsia (Ostensen & Cloowe, 2013). Immune system may be affected by several different routes in the SLE patient. The most significant predictor of preterm birth is lupus during the course of the pregnancy (Ostensen & Cloowe, 2013). Infections from infections, oral prednisone, and elevated anti-SSA/A and anti-SSB antibodies in mothers are associated with preterm birth (Cloowe & Clowse, 2011). Other antibodies, such as antiphospholipid antibodies, are associated with venous and arterial thromboembolic complications (Marks & Tullus, 2011). Fetal death and preterm births (Stanhope, White, Molder, Smyth, & Garwin, 2012).

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Nursing Considerations

References

To the due to the variances and 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 5 months for the benefit of the mother and infant (Tsokos et al., 2013). A multidisciplinary approach including obstetrical care, through high risk pregnancies in collaboration with a rheumatologist familiar with SLE should be utilized for the management of the pregnant (Ferenkeh-Koroma, 2012). Screenings for infections such as the HIV, anti-Ro/SSA and anti-DNA, 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 (Cloowe, Mageer, & Petti, 2011). Most lupus flares during pregnancy occur during the first trimester and up to 10% may have flares between 2-6 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 treatment for the autoimmune disease. Female patients diagnosed with SLE are at increased risk on the odds 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 many women (Barrutia, 2013). Providing patients at high risk for complications, most patients can have a successful pregnancy with a multidisciplinary approach that understands the disease and provide patients with appropriate education and support to enable SLE patients to maintain wellbeing and lead active lives (Ferenkeh-Koroma, 2012).


