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Case Report

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PREGNANCY WITH SYSTEMIC LUPUS ERYTHEMATOSUS: A CASE REPORT

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ABSTRACT

Systemic Lupus Erythematosus (SLE) is a chronic inflammatory and autoimmune connective tissue disorder that can affect multiple organ systems, including the skin, joints, kidneys, central nervous system, heart, lungs and liver. It is more prevalent among women than men and most women who are affected by the disease manifest it at some point during their reproductive years. Women with SLE are at higher risk of exacerbations of the disease during pregnancy. There is an increased risk of spontaneous abortions, intrauterine foetal death, preeclampsia, eclampsia, intrauterine growth restriction (IUGR) and preterm delivery. I report a case of 30 year $G_3P_{1+1}(L_0)$, who was referred from a primary health centre at 24 weeks of gestation in view of low platelet count. Patient was admitted and after careful history taking, examination and investigations diagnosis of SLE was made. Regular fetomaternal monitoring was done and patient was induced at 38 weeks of gestation. She delivered a live male child with birth weight 2.75kg with no gross congenital anomaly. Postnatal period was uneventful. This case report highlights the importance of preconceptional counselling and multidisciplinary approach needed in lupus pregnancies for optimal maternal and foetal outcome.

KEYWORDS: Systemic lupus erythematosus, autoimmune, chronic inflammatory, pregnancy.

INTRODUCTION

SLE is a chronic inflammatory and multisystem disease which damages the cells and tissue by autoimmunity and immune complex formation.[1] Significant racial differences are apparent in disease prevalence: black women have a prevalence of 405 per 100,000 compared to 164 per 100,000 among white women.[2] Pregnancy with SLE is a high risk condition and is associated with high maternal and foetal morbidity and mortality. The best predictor of the course of disease during gestation is the state of disease activity at the onset of pregnancy. Approximately 1/3rd of women who are in remission for atleast 6 months prior to pregnancy suffered an SLE flare compared with 2/3rd of women with active disease at the beginning of pregnancy.[3] Hence preconceptional counselling and delaying conception until the women has been in remission for atleast 6 months is of paramount importance. SLE tends to flare during pregnancy and disease flare is associated with increased risk of prematurity, pregnancy loss, IUGR and eclampsia.[4] Nephritis is known to be one of the most serious complications of SLE and a strong predictor of poor outcome.[5] Multidisciplinary approach with medical, obstetric and neonatal monitoring is essential for optimal outcomes.

CASE REPORT

A 30 year $G_3P_{1+1}(L_0)$ presented to the outpatient department of a tertiary care hospital at 24 weeks of gestation. She was referred from a primary health centre in view of low platelet count of 60,000/ µL. She was married for 4 years and conceived for the first time 1 year after marriage. At 26 weeks of gestation she went into spontaneous labor, labor pains lasted for 3 hours with delivery a female child with birth weight 750 grams. The baby died on 4th day of life in NICU due to respiratory distress. The patient conceived again 1 year after the first child birth but had an induced abortion at 12 weeks of gestation due to IgM rubella positivity and foetal affection. She again conceived spontaneously 1 year after the last abortion with the present pregnancy. The patient was admitted for further investigations and in view of spontaneous preterm delivery at around the same gestation. At the time of admission her blood pressure (BP) was 124/80 mm of Hg and pulse rate was 84/ minute. On per abdominal examination height of uterus (HOU) was corresponding to the period of gestation (POG) with relaxed uterus and foetal heart sound~144/ minute. She gave history of erythema on the face on exposure to sunlight (malar rash) along with excessive dryness of eyes. There was a positive history of bluish discoloration of digits on exposure to cold weather (Raynaud's phenomenon). There was no history of easy bruisability, ecchymosis, bleeding from gums, oral ulcers, seizure, fatigue and chest or flank pain. Bowel and bladder habits were normal. Her routine antenatal investigations were normal. Manual platelet count was 90,000/ µL and on peripheral smear megakaryocytes

were seen. Antiphospholipid antibody (APLA) profile revealed raised titres of IgM anticardiolipin antibody (ACA) and anti β_2 glycoprotein. Antinuclear antibody (ANA) levels were also raised 1:80 (homogenous). Specific investigation for SLE were done which revealed positive anti-smith (anti-Sm) antibodies and negative anti-double stranded DNA (anti-dsDNA). Anti-Ro/SSA and anti La/SSB antibody was negative. Her liver function tests (LFT), renal function tests (RFT) and 24 hour urine protein levels were normal. Patient was then referred to a rheumatologist and a preliminary diagnosis of SLE was made. Ultrasonography was done which revealed normal foetal growth parameters and no evidence of any gross congenital anomalies. Foetal echocardiography was done to rule out congenital heart block (CHB) and was found to be normal. Patient was started on daily subcutaneous injection of low molecular weight heparin (LMWH) 60mg, tablet ecosprin 150 mg OD and hydroxychloroquine (HCQ) 300 mg HS. Her complete haemogram (CHG), LFT, RFT and 24 hours urine protein were repeated every 3 weekly. At 31 weeks she was found to have increased levels of 24 hour urine protein (510mg/day) and low platelet count (60,000/µL). Patient was then started on tablet prednisolone 60mg/day. Regular fetomaternal monitoring was done and the patient was induced with dinoprostone gel at 38 weeks of gestation. Stress dose of injection hydrocortisone 100mg every 8 hourly was given at the time of labor. She delivered a healthy male child with birth weight 2.75kg. Postnatal period was uneventful and patient was advised to continue injection LMWH 60mg daily for 6 weeks postpartum. Tablet prednisolone was slowly tapered to 30mg/day over a period of 6 weeks. Patient along with her baby were discharged in good health on 7th postpartum day and was advised for continuous follow up at the department of rheumatology.

DISCUSSION

The peak incidence of SLE is between 15-40 years with male: female ratio of 1:9.[6] The etiology of the disease is not well understood. Genetic predisposition appears to be an important contributing factor to the development of SLE in that 5-12% of relatives of SLE patients also have the disease. Rare genetic factors such as deficiencies in complement components and mutation in *TREX1* gene, which encodes for DNA degrading enzymes are associated with the development of SLE.[7] Various exposures such as ultraviolet light, Ebstein Barr Virus and silica dust have also been associated with disease etiology.[8] Consistent with the higher prevalence of SLE among women hormonal factors appear to play an important role. Early menarche, oral contraceptives and postmenopausal hormone replacement have all been associated with an increased risk for SLE.[9] The clinical course of SLE is characterised by periods of disease "flares" interspersed with periods of remission. The most common presenting symptoms of SLE include arthralgia, fatigue, malaise, raynaud's phenomenon, photosensitive rash and alopecia. In this case report patient had positive finding of photosensitive rash and Raynaud's phenomenon. Pregnancy outcome is influenced by placental dysfunction, presence of APLA, preconceptional lupus activity, the severity of renal involvement and the course of SLE during pregnancy.[10] Positive ANA titres is used as screening test in SLE. An elevated ANA titre is not specific for SLE and can also be seen in other autoimmune conditions like sjogren syndrome, scleroderma and rheumatoid arthritis. Anti-dsDNA and anti-sm antibodies are highly specific for SLE, albeit less sensitive. Anti-dsDNA titres are frequently elevated in the setting of disease flare. In the present case patient was found to have raised ANA titres and positive anti-sm antibody. An important component in the disease is pre conceptional counselling and disease control prior to conception. Some of the drugs used in SLE are teratogenic and should be stopped prior to conception. Hence the risks involved may be minimized by appropriate timing of pregnancy and optimization of therapy prior to conception. Unnecessary steroid use should be avoided during pregnancy as it is associated with increased risk of premature rupture of membranes, gestational diabetes, and hypertension. However, in the case of disease flares, short courses of high doses and/or intravenous pulse methylprednisolone can be used.[11] Use of hydroxychloroquine (HCQ) can significantly reduce the risk of CHB and neonatal lupus syndromes.[12] Treatment of patients with APLA associated recurrent pregnancy loss with heparin and low dose aspirin have been shown to improve live birth rates.[13] In this case as well based on the history and investigations patient was started on LMWH and HCQ. Prednisolone was started only after there was increase in 24 hour urine protein levels at 31 weeks of gestation. In patients with disease flare and lupus nephritis azathioprine in combination with corticosteroids is the treatment if choice.[14] Other drugs like cyclophosphamide, mycophenolate mofetil and methotrexate are contraindicated in pregnancy and should be stopped at least three months prior to conception. Other immunosuppressive drugs that can be used safely in pregnancy are cyclosporine A and tacrolimus.[15] SLE is not a contraindication for pregnancy but the patient should be closely followed up by obstetrician, rheumatologist, nephrologist and neonatologist. Patient should be counselled regarding the worsening of disease in pregnancy. Proper preconceptional counselling and planned conception in the remission period is an important cornerstone in the disease management for best possible foetal and maternal outcome.

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