ORIGINAL RESEARCH PAPER

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A CASE OF LUPUS NEPHRITIS IN PREGNANCY- ALL HYPERTENSION IN PREGNANCY IS NOT JUST PRE ECLAMPSIA

KEY WORDS: SLE, lupus, lupus nephritis, glomerulo nephritis

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ABSTRACT

Historically, pregnancy for patients with SLE has been contraindicated. However, in the past several decades, diagnosis, treatment and management during pregnancy for patients with SLE has improved to the point that good outcomes are achievable for many of these patients. They are still considered to have high-risk pregnancies, who should be managed at tertiary care centre ideally under the coordinated care of a maternal-foetal medicine specialist, a rheumatologist and other specialists as needed. Lupus nephritis in pregnancy can lead to hypertension and renal failure, which can adversely affect the health of the mother and foetus. One such case was managed at our tertiary care centre is reported here.

INTRODUCTION

Systemic lupus erythematosus (SLE) is a serious multisystem disease that predominantly affects women of childbearing age. Flare-ups of SLE during pregnancy is an intimidating clinical scenario for both the treating physicians and patients[1]. Pregnancy can predispose patients to a lupus flare, especially if the disease is not adequately controlled at the onset of conception. Thus, patients are advised not to conceive until the disease has been quiescent for at least 6 months[2]. Disease onset in a younger age group, coupled with improved survival, makes pregnancy a likely occurrence in the setting of SLE[3]. Although outcomes have improved over time and successful live births can now be achieved in most cases, pregnancy still remains a high-risk situation in SLE. Both maternal and fetal mortality and morbidity are significantly increased, along with health care utilization and costs. A multidisciplinary coordinated approach with involvement of appropriate specialists and close monitoring is essential for optimal outcomes[3]. One of the most important causes of death in patients with lupus in the first decade is renal involvement. This defect is predominantly evidenced as lupus nephritis during pregnancy. The rate of active lupus nephritis in pregnancy is 4% to 30%, and in a person with a history of the previous recurrence of lupus nephritis is estimated to be 20% to 30%[4]. One such case which was managed in our hospital is reported in this article.

CASE REPORT

26 years old G3P1L1A1 with 7 months ANC came with absent foetal movements perception since 2 days. Also complains of headache, giddiness, blurring of vision, nausea, vomiting since 6 hours. She had one previous caesarean delivery 5 years ago which was done for meconium-stained liquor and it was uneventful and she also had one spontaneous abortion at 4 months. The index pregnancy was booked and 4 obstetric ultrasound examinations were done. She was a known case of hypothyroidism since 15 years and she was on tab eltroxin 100mcg once a day. About 7 years back she also had history of complaints of swelling in the legs, puffiness in the face, rashes on face, blood in urine when she was investigated and found to have lupus nephritis on renal biopsy. She was on treatment for lupus nephritis and after 2 months of diagnosis of lupus nephritis she had complaints of left sided tingling sensation, left sided weakness and loss of balance and on imaging (MRI) there was right posterior cerebral artery thrombus with absent flow with multiple acute infarcts. Her records show that she was treated with steroids, antihypertensives, diuretics, statins and anticoagulants. She took treatment for 8 months after which she regained the normal functioning of paralyzed arms and legs. Since then, she is not on any treatment for lupus nephritis. On examination, BP- 140/90mmhg taken on right arm in sitting position, pulse-98bpm. Pedal oedema of grade 2 was present. Uterus height-18-20 weeks size, relaxed, previous LSCS scar present, no scar tenderness, no contractions felt. There was blurring of vision, deep tendon reflexes were brisk and urine albumin was +++.

Provisional diagnosis was made as G3P1L1A1 with 27 weeks of gestation with hypothyroidism with previous LSCS with intra uterine foetal demise with severe pre-eclampsia in a known case of lupus nephritis. Tablet labetalol 100mg and inj. Magnesium sulphate according to Pritchard regimen was given. Meanwhile relatives were explained about the prognosis. Fundus examination was done which was within normal limits. Physician opinion was taken in view of lupus nephritis and history of stroke in the past. Her previous renal biopsy report showed diffuse proliferative lupus nephritis with crescentic transformation with grade 4- G(A), with activity score 15/24. Investigations revealed haemoglobin-19.3g/dl, platelet-1.5lakh/cmm, TLC-8000/cmm, Renal and liver function test, thyroid profile, coagulation profile were within normal limits. Patient was posted for hysterotomy with indication-impending eclampsia as she continued to have warning signs in spite of treatment. Surgery and postoperative stay were uneventful. Patient was vigilantly monitored for vitals, input and output. On post operative day 5, wound was healthy and patient went home healthy. This patient might look simply as she did not develop any complications, but whenever a pregnant woman presents with severe preeclampsia with atypical presentation like presenting with severe preeclampsia at early gestational age and warning signs and symptoms not relieving with magnesium sulphate; think of underlying cause not just pre-eclampsia perse.

DISCUSSION

Historically, pregnancy for patients with SLE has been contraindicated. However, in the past several decades, therapy and disease management for patients with SLE has improved to the point that good outcomes are achievable for many of these patients. As such, the current recommendations for pregnancy in patients with SLE are for planned conception after at least six months of disease quiescence, with the appropriate transition to non-teratogenic immunosuppression as needed to maintain quiescence. More recent studies have suggested that the necessary quiescence

period may only be four months rather than six months, but this is controversial[5]. Quiescent disease is the only well-established predictive factor for reducing the risk of SLE flare or other pregnancy complications. The risk factors associated with poor outcomes likely also affect the ideal length of remission before pregnancy, but these risk factors remain to be fully elucidated. Some of the implicated risk factors for poor outcomes like neonatal demise and IUGR, include lupus nephritis, anti-phospholipid syndrome, active disease during pregnancy or before conception, hypertension, and anti-dsDNA anti- bodies (especially anti-Ro/La which are associated with congenital heart block)[6].

DH Attia et al[7] studied pregnant lupus patients with prior lupus nephritis (LN) with those without nephritis and found the frequency of maternal complications, especially disease flares, was significantly higher in patients with LN. There were no significant differences between both groups regarding the frequency of pregnancy-related maternal complications, including pre-eclampsia. Regarding the frequency of foetal complications, there was no significant difference between groups. Quiescent LN at the onset of the conception does not add to the increased risk of maternal and foetal complications in SLE patients[7]. Koh et al[8] study also compared SLE patients with past LN and those with no history of nephritis, the renal group had more frequent obstetric complications including pregnancy-induced hypertension, pre-eclampsia, intrauterine growth retardation, and preterm delivery and a lower live birth rate. The adverse pregnancy outcome in patients with a history of LN was related to lupus activity prior to pregnancy and also showed that the independent predictors of renal flares during pregnancy were pre-existing LN, active disease prior to pregnancy. Saavedra et al[9] study also compared the outcome in pregnant women with lupus with or without prior lupus nephritis, the study concluded that women with quiescent LN have no increased risk of maternal and fetal complications as compared to non-renal patients; hence they recommended adequate control of the disease activity before conception. Even though our case had quiescent LN, she developed maternal and foetal complications. It is very much essential for women with known case of lupus nephritis to consult nephrologist and obstetrician before planning pregnancy.

A mini-review conducted by Mowla et al[4] states that the risk of preeclampsia in patients with lupus is between 10% and 35% and in women with creatinine levels above 2.8 mg/dL, the risk of preeclampsia and loss of pregnancy increases dramatically. In women with underlying kidney disease, the blood pressure should be controlled in a range lower than 140/90 mm Hg. The proposed drugs for pregnancy include methyldopa, nifedipine, diuretics, labetalol and hydralazine. Due to the possibility of embryonic anomalies, angiotensin-converting enzyme (ACE) inhibitors and angiotensin-receptor blockers (ARBs), should be avoided. Additionally, the administration of atenolol due to the possibility of an intrauterine growth retardation of the foetus should be avoided. Aspirin should be prescribed to prevent preeclampsia. The administration of aspirin in mothers with lupus causes a better prognosis for foetus. In our case, she was not on aspirin, which might have contributed to preeclampsia and foetal loss.

Mok et al[10] study states- differentiation between pre-eclampsia and nephritic flare during pregnancy can be difficult. Both conditions can cause hypertension, proteinuria, oedema and worsening renal function, and may coexist in the same patient. Distinction between relapse of nephritis and pre-eclampsia is important because the management is different. There are some clinical clues that may help. First, in patients with pre-eclampsia, the serum C3 and C4 levels normally rise steadily during pregnancy whereas a drop in C3 and C4, coupled with a rising anti-dsDNA titre, is likely to be associated with disease flare in a SLE patient with proteinuria. Second, the presence of active urinary sediments like white cell, red cell, or granular casts and disease activity in other organs such as true arthritis, vasculitis, oral ulcers, and lymphadenopathy in a patient with worsening proteinuria points to a lupus flare. Third, prednisone treatment will typically worsen pre-eclampsia while renal flare will respond to increasing dose of prednisone. It also states that hypertension was not present in most of their patients with relapse of nephritis while raised blood pressure occurred almost universally in patients with pre-eclampsia. Our case looks more like preeclampsia than nephritic flareup.

CONCLUSION

It is important to differentiate pre-eclampsia from nephritic flare during pregnancy as both the conditions present with similar clinical picture and the management is different. Quiescent LN prior to conception is found to have good maternal and foetal outcomes and successful pregnancy is similar with patients without nephritis. So, it is very important to have nephrologist and obstetrician consultation before planning pregnancy.

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