A SUCCESSFUL OUTCOME OF TWIN PREGNANCY IN PATIENT WITH SYSTEMIC LUPUS ERYTHEMATOSUS – A CASE REPORT

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Abstract
Systemic lupus erythematosus (SLE) is an autoimmune disease which usually affects women of reproductive age and may co-exist with pregnancy. Its multisystem involvement and therapeutic interventions pose a high risk for both mother and the foetus. Therefore obstetrician should know about their effects on pregnancy and vice-versa and the therapeutic options available for optimizing maternal and foetal outcome.

Keywords: IUGR; LMWH; Proteinuria

Introduction
Systemic lupus erythematosus (SLE) is an autoimmune disease with considerable female predominance (9:1) with prevalence of 3/100,000. The usual disease onset is in reproductive years of women’s life. Women with SLE (incidence 0.29/1000 deliveries) are at higher risk for exacerbations of disease during pregnancy such as spontaneous abortions, intrauterine foetal death (IUF), preeclampsia, eclampsia, preterm delivery and intrauterine growth restriction (IUGR). However, over past few decades there has been trend towards favourable outcomes.¹ This case report summarizes management of an antenatal patient with SLE.

Case report
A 25-year-old lady G2P1L0 34 weeks with twin pregnancy with past history of SLE was admitted in our hospital with complaints of bleeding per vaginum since morning. On examination her pulse was 120/min and blood pressure was 110/70 mmHg. The patient’s respiratory and cardiovascular systems were normal. On per abdomen examination uterus was over distended both foetal heart sounds were regular. She had regular antenatal checkups. She was on low-molecular-weight heparin (LMWH) and aspirin. Her obstetric history revealed that she had previous history of intrauterine death (IUD) one year back with history of severe pregnancy induced hypertension (PIH). Decision of caesarean section was taken with consent. APGAR score of both babies was good. Babies were kept in neonatal intensive care unit due to low birth weight. Postoperative period was uneventful. She was discharged on day five and continued to be on heparin.

In our case she was diagnosed as a case of SLE a year back when she complained of bluish extremities and gangrenous changes in lower extremities suddenly. Her lower and upper limb angiogram was done suggestive of thrombo-occlusion of right dorsalis pedis artery. Her ds-DNA was positive. She was put on nifedipine and heparin. She also had one seizure attack but CT scan was normal. She was put on eptoin. She came to us with urine pregnancy test positive. She was started on aspirin and LMWH. She was also detected to be hypothyroid so thyroxine replacement was given. She had regular antenatal visits. Her growth scans and Doppler were normal. At 34 weeks she came in emergency with ante-partum haemorrhage for which emergency caesarean section was done.

Discussion
Systemic lupus erythematosus (SLE) is an inflammatory autoimmune disorder that may affect multiple organ system. Many of its clinical manifestations are secondary to the trapping of the antigen antibody complexes in capillaries of visceral structures or to autoantibody mediated destruction of host cells (e.g. thrombocytopenia). The clinical course is marked by spontaneous remission and relapses.² The classic presentation of a triad of fever, joint pain, and rash in a woman of childbearing age should prompt investigation into the diagnosis of SLE. Patients may present with any of the following manifestations:

- Constitutional (e.g. fatigue, fever, weight changes)
- Musculoskeletal (e.g. arthralgia, arthropathy, myalgia, frank arthritis, avascular necrosis)
- Dermatologic (e.g. malar rash, photosensitivity, discoid lupus)
- Renal (e.g. acute or chronic renal failure, acute nephritic disease)
- Neuropsychiatric (e.g. seizure, psychosis)
- Pulmonary (e.g. pleurisy, pleural effusion, pneumonitis,
pulmonary hypertension, interstitial lung disease)
· Gastrointestinal (e.g. nausea, dyspepsia, abdominal pain)
· Cardiac (e.g. pericarditis, myocarditis)
· Haematologic (e.g. cytopenias such as leukopenia, lymphopenia, anemia, or thrombocytopenia)

The diagnosis of SLE is based on a combination of clinical findings and laboratory evidence. The presence of 4 of the 11 American College of Rheumatology (ACR) criteria yields a sensitivity of 85% and a specificity of 95% for SLE.

The following are the ACR diagnostic criteria in SLE:
· Serositis
· Oral ulcers
· Arthritis
· Photosensitivity
· Blood disorders
· Renal involvement
· Antinuclear antibodies
· Immunologic phenomena (e.g., dsDNA; anti-Smith [Sm] antibodies)
· Neurologic disorder
· Malar rash
· Discoid rash

Testing
The following are useful standard laboratory studies when SLE is suspected:
· CBC with differential
· Serum creatinine
· Urinalysis with microscopy

Other laboratory tests that may be used in the diagnosis of SLE are as follows:
· ESR or CRP level
· Complement levels
· Liver function tests
· Creatine kinase assay
· Spot protein/spot creatinine ratio
· Autoantibody tests

Presence of antiphospholipid antibody during pregnancy is associated with significant risk of pregnancy morbidity and loss. Although antibodies are present in about a quarter to half of patients with SLE, only a fraction of these patients develop antiphospholipid syndrome (APS). APS is defined by the persistence of medium-to-high titre antiphospholipid antibodies (anticardiolipin, anti-β2 glycoprotein and/or the lupus anticoagulant) on at least two laboratory tests, 12 weeks apart, in the presence of at least one clinical criterion of thrombosis and/or pregnancy morbidity.

Lupus can flare during any trimester, as well as in the postpartum period. Flare may be defined as unpredictable bouts of disease after a period of remission. Two of the indices Pregnancy Disease Activity Index (SLEPDAI) and BILAG (British Isles Lupus Activity Group) 2004 have been predominant ones used for defining flares. Pregnancy may increase lupus activity but flares are usually mild. Patients with lupus nephritis (LN) and antiphospholipid antibodies are at risk of developing pre-eclampsia and should be monitored closely. Proteinuria may increase during pregnancy in women with underlying kidney disease. Very low serum complement, active urine sediment and evidence of generalized lupus activity favour the latter. Other features such as hypertension, thrombocytopenia, rise in serum uric acid levels and proteinuria may be observed in both conditions. The reason for such discordant study results most likely stems from different definitions of flare, difficulty in differentiating flare from hypertensive complications of pregnancy. It is a diagnostic challenge to differentiate lupus disease manifestations from normal pregnancy-related effects or pregnancy-related hypertensive complications. At times, even the most experienced clinician cannot differentiate these with certainty and ends up treating both. Preeclampsia and eclampsia can mimic lupus with both presenting as oedema, thrombocytopenia, hyperuricemia, anaemia, hypertension, proteinuria, renal impairment, hematuria and additionally seizures in eclampsia.

It is well known that the risk of adverse foetal outcomes is increased in pregnancies complicated by lupus. The frequencies of spontaneous abortions and stillbirths are increased in women with lupus with the stillbirth rate nearly five times greater than for non-lupus pregnancies. Preterm births occur in approximately 20% of lupus pregnancies and are associated with hypertensive medication use, corticosteroid use at conception, severe flare during pregnancy, nephritic range proteinuria and positive anticardiolipin antibody. Foetal growth restriction occurs more frequently in pregnancies complicated by lupus and must be monitored closely.

Foetal wastage may result from several factors, including disease activity, hypercoagulability and placental pathology.

Foetal growth is impaired when blood flow through the placenta is restricted by placental pathology characterized by ischemia/hypoxia, decidual vasculopathy, decidual and foetal thrombi, chronic villitis, decreased placental weight and placental infarctions along with deposits of fibrin, IgG, IgM, IgA and C3 in the trophoblastic membrane.

In short, SLE should be controlled prior to pregnancy. Patient should be informed about the risk of flares, pre-eclampsia and
foetal compromise. A multidisciplinary team should manage SLE during pregnancy. Antenatal care should begin early. Frequent antenatal visits every fortnight in the first and second trimester and weekly in the third trimester should be advised. Early dating USG should be performed. Low dose aspirin, in combination with prophylactic doses of heparin, significantly reduces the risk of pregnancy loss. Low-molecular weight heparin (LMWH) has similar efficacy to unfractionated heparin, but requires twice daily administration of all doses during pregnancy. Low-molecular weight heparin must be transitioned to unfractionated heparin prior to delivery. Heparin treatment needs to be continued for six weeks postpartum. The patients with prior systemic thrombosis should receive full therapeutic doses of heparin throughout pregnancy. Strict monitoring aimed at SLE flares, pre-eclampsia and IUGR should be continued throughout pregnancy. Foetal surveillance should be generally initiated at 30-32 weeks. SLE patients should be delivered at term. Postdatism should be avoided. 

Conclusion
We successfully managed the case of twin pregnancy with SLE. If planned properly when the disease is quiescent and monitored closely in a multidisciplinary setting, pregnancy outcomes can be favorable in women with lupus.

In conclusion, pregnancy is no longer considered an absolute contraindication in lupus.

Editor’s comment
This case report highlights the effect of SLE on pregnancy and vice-versa. Obstetrician should be aware of the potential complications and line of management. Moreover, proper referral and counseling regarding the prognosis of pregnancies is important.

References