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ORIGINAL ARTICLE

Clinical and serological risk factors of systemic lupus erythematosus outcomes during pregnancy

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KEYWORDS

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Abstract *Introduction:* SLE is an important risk factor for mother and fetus during pregnancy.

Aim of the work: To identify clinical and serological risk factors that may cause poor maternal and fetal outcomes in pregnant systemic lupus erythematosus (SLE) patients.

Patients and methods: Forty selected SLE pregnant women (group A) versus 35 non-pregnant SLE patients (group B). SLE disease activity index (SLEDAI) and flares were evaluated for both groups. Laboratory investigations included double stranded DNA, anticardiolipin antibodies (aCL), and complements (C3 & C4). SLE pregnant patients were followed up in the second and third trimesters by ultrasonography and fetal Doppler were done to assess fetal outcome. Risk factors for poor maternal and fetal outcome were recorded.

Results: SLEDAI was increased in both groups more in group A. Lupus flares were increased during pregnancy as it occurred in (62.5%) of group A compared to (37.14%) in group B where severe flares were more frequent in group A. Gestational hypertension and active SLEDAI were found statistically significant for poor maternal outcome. Fetal outcome included full term 37.5%, prematurity 25%, intra-uterine growth retardation (IUGR) 22.5%, stillbirth 12.5%, abortion 7.5% and congenital heart block (CHB) 2.5%. Factors significantly associated with poor fetal outcome were severe flares and active renal disease where fetal loss significantly associated with aCL antibodies. Full term was more common in patients with no flares.

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Conclusion: These data demonstrate that pregnancy in SLE patients should be considered as a high-risk pregnancy and conception should be planned during a quiescent period. Close monitoring for optimal disease control of flares, lupus nephritis, gestational hypertension and aCL antibodies is recommended.

1. Introduction

Systemic lupus erythematosus (SLE) is a chronic, multisystem, autoimmune disease that is well recognized to affect the child-bearing age females, characterized by periods of fluctuating disease activity [1].

In the past, women with SLE were advised against pregnancy. However, with good disease control and rigorous monitoring, there is no reason why the majority of these women should not have the opportunity to bear children. SLE is an important risk factor for mother and fetus during pregnancy. Lupus women are thought to experience disease deterioration due to pregnancy, but varying incidence and rates have been reported [2].

Multiple clinical and laboratory factors that have been identified in association with the less favorable outcome during pregnancy include previous nephropathy, maternal hypertension, flare-up of the disease and positivity for antiphospholipid antibodies (aPL) [3]. Obstetric complications that may be increased in lupus pregnancies include fetal loss, prematurity, preeclampsia, intrauterine growth retardation, and neonatal lupus syndromes [4].

The objective of this study was to identify clinical and serological risk factors that may cause poor maternal and fetal outcomes in SLE patients.

2. Patients and methods

2.1. Study subjects and design

Forty SLE pregnant women were selected from inpatient and outpatient clinics of Rheumatology & Rehabilitation and Obstetrics & Gynaecology Departments in Zagazig University Hospitals (group A), from March 2008 to October 2010.

Another 35 non-pregnant SLE patients attending Rheumatology outpatient clinics were taken as control group (group B) that were homogenous for age, disease duration, diagnostic and therapeutic protocol. All patients and controls met the revised criteria of American College of Rheumatology for SLE [5]. Exclusion criteria included therapeutic abortion, patients suffering from diseases other than SLE (e.g. diabetes or hypertension), preeclampsia, and patients escaped from follow up.

Patients in group A were first booked in the first trimester and followed up in second and third trimester. Maternal information included age, past obstetrical history, duration of SLE, previous and current manifestations of SLE and current medications. Active disease at conception was defined as the use of >10 mg of prednisone daily, the use of any immunosuppressive agent, or an SLE disease activity index (SLEDAI) score of >2 [6,7].

Non-pregnant SLE patients attending lupus clinic were followed up once every 3 months with inactive disease and more frequent with active disease (increased SLEDAI score of >2).

Both groups were also assessed for disease flare according to Petri et al. [8] in the first visit and once every trimester. Mild/moderate flare was defined as new or worsened cutaneous disease, nasopharyngeal ulcers, pleuritis, pericarditis, arthritis, fever attributable to SLE, the addition of non-steroidal anti-inflammatory drugs or an increase in prednisone up to a dose of 0.5 mg/kg/d. Severe flare was defined as new or worse CNS disease; vasculitis; nephritis; myositis; hemolytic anemia; platelet count less than 60,000/mL; the addition of azathioprine, hospitalization for SLE-related manifestations, or an increase in prednisone to >0.5 mg/kg/d.

Gestational hypertension was defined as a systolic blood pressure $\geqslant 140$ mm Hg and diastolic blood pressure $\geqslant 90$ mm Hg on at least two occasions, at least 6 h apart after 20 weeks' gestation and with normalization of blood pressure postpartum. Antihypertensive drugs were given to patients with blood pressure $\geqslant 160/100$ mm Hg [9,10]. We did not include patients with previously-induced hypertension as we aimed to study it as a maternal or fetal risk factor.

Written consents for ethical consideration were taken from all patients.

2.2. Laboratory procedures

Laboratory data for both groups included ANA, double-stranded DNA antibody, aCL antibodies, complements (C3 & C4), complete blood count, and urine analysis.

For group A, sequential ultrasound studies and Doppler analysis of uteroplacental circulation were performed every 2 weeks in the second trimester and weekly thereafter. Anti-SSA was detected in patients with abnormal ultrasonography suggesting CHB.

2.3. Therapeutic protocol

It included pre-conceptional treatment which was kept unchanged as possible. Cyclophosphamide was switched to azathioprine (100 mg/d). Prednisolone dosage was unchanged until there was a lupus flare, the dose was increased according to severity of flare. Cases with positive aCL antibody had got infantile aspirin (75 mg/d) once daily. Low molecular weight heparin was added to patients with previous thrombosis and discontinued in the last 10–15 days before delivery.

Statistical methods: Data analysis was performed by using SPSS version 15.0 (SPSS Inc., Chicago, IL, USA). Proportions were compared using the χ^2 test and Fisher exact test when appropriate. Quantitative variables were presented as mean ± SD and t test for qualitative variables statistical significance was set at $p < 0.05$.