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Case Report

New onset of systemic lupus erythematosus during pregnancy

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ABSTRACT

Systemic lupus erythematosus (SLE) is a chronic inflammatory disease that frequently affects young women. If SLE is first suspected during pregnancy, the diagnostic criterion is the same as for nonpregnant women and the treatment should be started as soon as the diagnosis is made. This study describes a 31-year-old pregnant woman who was hospitalized with shortness of breath, pain, and stiffness in phalangeal joints. The new onset of SLE was diagnosed. The disease was controlled by medical treatment. The patient had successful pregnancy and a healthy baby was delivered.

Keywords: Systemic lupus erythematosus, Pregnancy, New onset

INTRODUCTION

Systemic lupus erythematosus (SLE) is a multisystem autoimmune disorder which involves almost all organs and tissues, affecting young women in childbearing age. SLE is a predominantly a Th-2 mediated disease.[1] A progressive Th-2 cytokine predominance during pregnancy is caused by hormones such as progesterone, estradiol, cortisol, prostaglandin D2, and leukemic inhibitory factor.[1,2]

In general, pregnancy does not cause flares of SLE, but it could be a risk factor for SLE activity. The variable flare rate has been reported. Twenty five to 65 percent women will have the flare during pregnancy. Different organ systems may have variable response to pregnancy; the renal and hematologic flares are more common than musculoskeletal flares.[3]

This study presents a case of new-onset of SLE with pleuritis, pericarditis and arthritis in a pregnant woman at 22 weeks' gestation.

CASE REPORT

A 31-year-old multigravid woman, 22 weeks pregnant, present in the emergency room with shortness of breath and pain in the chest that has progressed over the past week. Shortness of breath was worse in the supine position. The patient had a three week history of pain and stiffness in phalangeal joints in the morning. Her medical history was significant showing, hypothyroidism, treated by 150 mg levothyroxim once a day, and conisation of cervix on account of CIN III. The previous delivery was at term, spontaneously baby was born, weighing 2900 g.

On examination patient was hypertensive (164/92 mm Hg) with a heart rate of 80 bpm and a respiratory rate of 23 breaths per minute with an oxygen saturation of 98 %. The heart sounds were regular without murmurs and both lungs were clear on auscultation. The patient had pitting edema of both legs and face. There was no evidence of skin rash. Body temperature was normal and foetal heart rate was 150 beats per minute.

The provisional diagnosis was pneumonia. Chest X-ray showed right sided pleural effusion. Echocardiography displayed a mild pericardial effusion. The initial laboratory tests revealed neutrophilia, lymphopenia, and elevated C-reactive protein (Table 1). The urinalysis was normal.

Empiric antibiotic treatment (ceftriaxone, clarithromycin) was started based on findings of pleural effusion. Despite antibiotic treatment shortness of breath, pain, and stiffness of fingers still persisted. Smear and culture for any bacteria, including *Mycobacterium tuberculosis* taken from pleural effusion were negative. The cytological examination of the effusion smears revealed neutrophils, a small amount of eosinophils, and lymphocytes. To exclude infections polymerase chain reaction for detection of influenza, *Mycoplasma pneumoniae, Chlamydophila pneumoniae, Legionella pneumoniae, Streptococcus pneimoniae, Haemophilus influenza, Bordetella pertussis in nasopharyngeal swab, serological* tests for HIV, *lues,* and *Toxoplasma gondii* were done. All tests were negative.

Table 1: Laboratory test.

Blood count	Result	References	Coagulogram	Result	References
Hemoglobin (g/dl)	12.1	12.0 – 16.0	APTT (s)	28.4	26.0 – 36.0
Red blood count (106/µL)	4.2	4.2 – 5.4	INR	0.9	0.8 – 1.2
MCV (fL)	83	80.0 – 100.0	Fibrinogen (g/L)	4.6	1.8 – 3.6
MCH (pg)	29	27.0 – 33.0	D-dimer (mg/L)	1.18	≤ 0.5
MCHC (g/dL)	34.3	32.0 – 36.0			
RDW (%)	13.7	11.5 – 14.5	**Biochemistry**		
White blood cell (103/µL)	7.5	4.0 – 10.0	C-reactive protein (mg/L)	27.0	0.0 – 0.5
Neutrophil (103/µL)	6.3	2.0 – 5.5			
Lymphocyte (103/µL)	1.0	1.5 – 3.5			
Monocyte (103/µL)	0.1	0.1 – 0.6			
Eosinophil (103/µL)	0.1	0.0 – 0.3			

Abbreviations: MCV, mean corpuscular volume; MCH, mean corpuscular haemoglobin; MCHC, mean corpuscular haemoglobin concentration; RDW, red blood distribution; APTT, Activated Partial Thromboplastin Time.

Chest CT showed bilateral axillary lymphadenopathy (2.5 cm in diameter). An excisional biopsy of right axillary lymph nodes was performed to exclude sarcoidosis and lymphoproliferative disease. Histologic examination of lymph node showed non-specific reactive changes.

Immunological screening was positive for ANA (> 1:3200 speckled), anti – Ro (SSA) (4.0), anti – La (SSB) (4.1) and perinuclear antineutrophil cytoplasmic antibody (p-ANCA). Serum C3 complement factor was low, respectively; 0.57 g/L (range 0.90–1.80 g/L).

The presence of pleural and pericardial effusion, lymphopenia, polyarthritis, and positive ANA suggested a diagnosis of SLE according to criteria proposed by the American College of Rheumatology.

The patient was treated with oral prednisolone 30 mg qd and hydroxychloroquine (HCQ) 200mg qd. The dyspnoea and arthritis resolved and the patient was discharged on the 23rd hospital day. Prednisolone 10 mg and hydroxychloroquine 200mg daily was prescribed to control the disease.

At 36th weeks of gestation patient presented with preterm premature rupture of membranes (PPROM) and spontaneously delivered a female baby, weighing 2320 g (low birth weight, <10th) with Apgar score of 8 and 9 at 1 and 5 minutes.

Repeated screening after delivery was positive for ANA and anti-double stranded *DNA* (anti-dsDNA) (47 U/ml). Lupus was controlled with prednisolone 10 mg qd and HCQ 200mg qd postpartum.

DISCUSSION

New onset SLE during pregnancy can be considered as SLE activity and might be associated with a worse outcome, fortunately if new onset SLE accoutred during the third trimester of pregnancy, the outcome is better. The flare of SLE during pregnancy increases rate of foetal and obstetric complications (Table 2).[4]

Table 2: Complications during pregnancy caused by SLE.

Groups	Complications
Pregnancy lost	Spontaneous abortion
	Intrauterine foetal death
Foetal complications	Premature birth
	Intrauterine growth restriction (IUGR)
	Premature and precocious rupture of membrane (PPROM)
Obstetric complication	Pre-eclampsia
	Eclampsia

IUGR is one the most common foetal complications in lupus pregnancies. Risk factors include hypertension, active lupus and the presence of anti-phospholipid antibodies (aPLs) Placental insufficiency is common in SLE and intrauterine growth restriction is associated with decidual vasculopathy and thrombi, chronic villitis and decreased placental weight. APLs cause the venous and arterial trombosis which leads to decidual vasculopathy and coagulopathy and have effect on gestational outcome. The patient's aPLs were negative.[6-9]

Neither disease activity nor antibodies can be a risk factor for foetal complications. Anti-Ro/SSA and anti-La/SSB are associated with neonatal lupus syndrome.[3] The most common cardiac manifestation is congenital cardiac block (CHB). CHB affects about 2% of children born to primigravid women with anti-Ro antibodies.[3,10] Despite positive patient's anti-Ro and anti-La the baby was born healthy.

As a screening for SLE an antinuclear antibodies (ANA) test can be used. The ANA test was positive for this patient. The ANA test has high sensitivity - 95 %. Positive ANA test without clinical symptoms features is not a diagnostic criterion. Anti-double stain antibodies (anti-dsDNA) is rarely used for SLE screening, because the sensitivity is only 70%, however anti-dsDNA has high specificity – 99 % .On the first admission the patient had negative anti-dsDNA test.[11-13]

The patient had low C3 level, which is one of the diagnostic criteria of SLE. During pregnancy compliment C3 and C4 level, increased due to oestrogen induced synthesis in the liver. Therefore, the normal C3 and/or C4 level cannot exclude the possibility that the disease is active in a pregnant woman with SLE. On the other hand, the low compliment level cannot always be associated with SLE activity in case of toxaemia or other hepatic disease during pregnancy.[1,14]

The signs and symptoms of lupus pleuritis are non-specific reasons and the diagnosis is based on exclusion of other reason, for example, pneumonia, heart failure, malignancy etc. Microbiologic and cytological analysis of pleural fluid should be done. In case of lupus pleuritis, the fluid is exudative, contains SLE cells and usually neutrophilic leukocytes. In our case, cytological analysis of pleural effusion showed neutrophilic leukocytes without SLE cells. According to the cytological analysis the pneumonia was suspected. A sensitive and specific, respectively, 91.67 % and 83.33 %, biomarker for lupus pleuritis could be the pleural fluid ANA at a titter of ≥ 1:160. At the beginning SLE was not the provisional diagnosis, the ANA titter was not preformed. Although, it does not give additional diagnostic information about autoantibodies in serum and the test is not needed.[15,16]

The choice of treatment for SLE is limited to a few safe drugs during pregnancy. The prednisolone and HCQ were used for the treatment in this case. The lowest steroid dose should be prescribed, because a high dose of steroid during pregnancy may lead to diabetes, hypertension, pre-eclampsia, and PPROMS.[3] However, in case of flare, 'pulse' corticosteroids can be used.[1,3,17] HCQ is safe and can reduce the risk of flare, congenital heart blockage and neonatal lupus syndrome.[3] HCQ should be continued in all SLE pregnancies, discontinuation may result in flare.[3]

CONCLUSION

New onset lupus during pregnancy should be considered as a high risk pregnancy. A multidisciplinary approach to the treatment and management of lupus can decrease maternal and foetal morbidity and mortality.

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