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Case Report

Beyond conduction defects: fetal myocarditis as a rare cardiac manifestation in anti-Ro/La positive lupus pregnancy

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ABSTRACT

Fetal myocarditis is an uncommon yet potentially fatal complication of maternal systemic lupus erythematosus (SLE), in contrast to the more recognized congenital heart block (CHB). We report a case of a fetal myocarditis in young female with lupus and anti-Ro/SSA and anti-La/SSB antibodies positivity. This case emphasizes the importance of early fetal monitoring in lupus pregnancies and highlights the complex role of maternal auto antibodies in fetal cardiac inflammation.

Keywords: Fetal myocarditis, SLE, MTP

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with multi system involvement, often exacerbated during pregnancy due to immune and hormonal changes. Pregnant women with SLE are at increased risk of maternal and fetal complications, including pre-eclampsia, preterm birth, intrauterine growth restriction (IUGR), neonatal lupus, and, in extreme cases, stillbirth. Among the most serious fetal complications is congenital heart block (CHB), occurring in approximately 2% of anti-Ro/SSA and anti-La/SSB positive pregnancies due to immune-mediated fibrosis of the atrioventricular (AV) node.[1]

The presence of maternal anti-Ro/La antibodies are strongly associated with adverse pregnancy outcomes such as fetal loss and CHB. In rare instances, these antibodies can also lead to fetal myocarditis—an inflammatory cardiac complication with potentially fatal outcomes. We present a rare case of fetal myocarditis in a pregnant woman with SLE and anti-Ro/La antibody positivity, with a history of recurrent pregnancy loss, culminating in medical termination of pregnancy (MTP).

CASE REPORT

A 28-year-old female, diagnosed with SLE in 2017, with manifestations including inflammatory polyarthritis, Raynaud's phenomenon, leukopenia, ANA positivity (IF +3 speckled pattern at 1:100 dilution), persistently low complement levels (C3 and C4), and anti-Smith antibody positivity, presented at 20 weeks of gestation during her second pregnancy. Her obstetric history was significant for a spontaneous abortion at 10 weeks of gestation during her first pregnancy. At that time, evaluation revealed positive anti-Ro/SSA antibodies with a titer of 145 RU/ml (normal <20). Her antiphospholipid antibody (aPL) profile was negative, including anti-cardiolipin IgG/IgM, beta-2 glycoprotein IgG/IgM, and lupus anticoagulant (LAC).

Her current pregnancy was also marked by persistently positive anti-Ro/SSA and anti-La/SSB antibodies. At 20 weeks of gestation, fetal echocardiography demonstrated supraventricular tachycardia, global hypokinesia, biventricular dysfunction, and mild pericardial effusion. These findings were consistent with fetal myocarditis. Additionally, there was moderate tricuspid and mitral regurgitation, along with a prominently echogenic aortic

valve. Notably, there was no evidence of conduction abnormalities or congenital heart block. Given the progressive cardiac dysfunction and deteriorating fetal status, the pregnancy was medically terminated at 23 weeks of gestation. Prior to conception, her SLE disease activity was well controlled on a maintenance regimen of low-dose corticosteroids (prednisolone 5 mg daily), hydroxychloroquine 200 mg/day, and tacrolimus 2 mg daily. She remained clinically stable throughout the pregnancy without any disease flare-ups.

Table 1: Timeline of clinical course.

Timeline	Clinical course
Pre-conception	SLE stable on low-dose steroids, HCQ, tacrolimus
10 weeks (1st pregnancy)	Spontaneous abortion; anti-Ro/La positive; aPL negative
20 weeks (2nd pregnancy)	Fetal echo: SVT, biventricular dysfunction, mild pericardial effusion. Moderate MR/TR, echogenic aortic valve; no CHB
22–23 weeks	Progression of fetal myocarditis with cardiac dysfunction; pregnancy terminated after multidisciplinary counseling

DISCUSSION

Maternal anti-Ro and anti-La antibodies can result in neonatal lupus erythematosus syndrome (NLES). These antibodies are known to be associated with CHB and fetal loss. The pathogenesis of cardiac involvement in anti-Ro/La antibody-positive pregnancies begins with the transplacental passage of maternal IgG autoantibodies, which occurs from the end of the first trimester, peaking in the second trimester.[1] These anti-Ro/SSA and anti-La/SSB antibodies specifically target fetal cardiac tissue, particularly the atrioventricular (AV) node and cardiomyocytes, leading to immune-mediated injury.[2] The Ro (52 kDa and 60 kDa) and La (48 kDa) antigens, typically intracellular, become abnormally expressed on the surface of apoptotic fetal cardiomyocytes, where they become accessible to circulating maternal antibodies.[2] This binding results in immune complex formation and activation of the classical complement cascade, especially C3 and C4 consumption, initiating cytolytic and pro-fibrotic inflammation.[2] The resulting injury leads to fibrosis and calcification of the AV node, culminating in irreversible third-degree CHB in approximately 2% of anti-Ro-positive pregnancies.[2] Inflammatory cytokines such as TNF-α, IL-6, and interferon-α further exacerbate myocardial injury by promoting fibroblast activation and myocardial fibrosis. In some cases, especially in the absence of CHB, persistent autoimmune damage may lead to endocardial fibroelastosis (EFE)—a pathological thickening of the ventricular endocardium associated with diastolic dysfunction, heart failure, or hydrops fetalis.[3] The pathogenesis of fetal myocarditis in SLE pregnancies is believed to be mediated by direct immune-mediated myocardial inflammation due to maternal anti-Ro/SSA and anti-La/SSB antibodies, leading to lymphocytic infiltration and myocardial injury.[4] However, while CHB is widely recognized, fetal myocarditis is a much rarer cardiac complication in maternal SLE and remains under reported in the literature.

Fetal myocarditis differs from CHB in that it represents an active inflammatory process affecting the myocardium, rather than a fibrotic conduction abnormality. The distinction between CHB and myocarditis is clinically important, as CHB is primarily a conduction abnormality leading to bradycardia, whereas myocarditis presents with tachycardia, systolic dysfunction, ventricular dilation, and pericardial effusion like in our case

In our patient, fetal echocardiography at 20 weeks of gestation revealed biventricular dysfunction, global hypokinesia, and pericardial effusion, findings consistent with fetal myocarditis rather than conduction abnormalities. Additionally, moderate tricuspid and mitral regurgitation and echogenic aortic valve suggesting inflammatory involvement of the myocardium. These findings align with previous reports where maternal anti-Ro antibodies have been implicated in both myocarditis and conduction defects, reinforcing the idea that autoimmune-mediated fetal cardiac injury extends beyond CHB. Reports suggest that maternal usage of flourinated corticosteroids and intravenous immunoglobulin (IVIG) may mitigate inflammation in such cases, but data on effective treatment strategies remain limited with no conclusive evidence.

In our patient, early fetal echocardiography enabled the detection of myocarditis before irreversible damage occurred. Despite hydroxychloroquine and close fetal surveillance, pregnancy ended in second trimester loss. The presence of severe fetal myocarditis likely contributed to this adverse pregnancy outcome.

CONCLUSION

This case highlights the significant risks posed by anti-Ro/La antibodies in patients with SLE, emphasizing their potential to cause fetal loss and, in rare instances, fetal myocarditis—a serious and under recognized complication. It reinforces the critical importance of early detection through serial fetal echocardiography for timely diagnosis and optimal perinatal management.

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