Recurrent Fetal Wastage in a Woman with Anti-Ro (SS-A) Antibody: Successful Therapy with Corticosteroids

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Case Reports

This is the case of a 30-year-old woman with recurrent fetal loss at 25–27 weeks of pregnancy, who was found to have strongly positive anti-Ro (SS-A) antibody and a normal anatomical, hormonal, chromosomal and infection work-up. Treatment with corticosteroids early in the fourth pregnancy resulted in disappearance of anti-Ro (SS-A) antibody and successful delivery of a healthy baby.

Case History

The patient was a 30-year-old normotensive, non-diabetic woman with a history of recurrent fetal loss. Her first pregnancy in 1984 had resulted in intrauterine death at 26 weeks and induced delivery of a 630 g macerated fetus. There followed two additional pregnancies in 1985 and 1986 that similarly resulted in induced deliveries at 25 and 26.5 weeks of macerated fetuses of 330 g and 720 g of body weight respectively. After her third induced delivery, she underwent clinical and laboratory evaluation for possible immunological causes of recurrent fetal wastage. Her physical examination was normal, with no clinical evidence of connective tissue disease. The following laboratory studies were carried out and found to be normal:

1. SMAC (biochemical screening).
2. Oral glucose tolerance test.
4. Thyroid function studies (free T3, free T4, TSH).
5. TORCH profile.
6. Chromosomal studies (both on the patient and her husband).
8. Rheumatoid factor (RF), Antinuclear antibody (ANA), anticardiolipin antibody and VDRL were negative.

In August 1986, anti-Ro (SS-A) was found to be strongly positive and anti-La (SS-B) was also positive. The patient was then advised to return to our clinic when she became pregnant. In January 1987, she returned to the clinic in the 9th week of her fourth pregnancy. The vital signs and physical examination were found to be normal. Infectious disease work-up, including tests for mycoplasma and toxoplasma were negative. Her blood group was O Rh positive. Ultrasound examination of the uterus done in April 1987 revealed twin pregnancy with active fetal cardiac motion. Ultrasound measurement of both fetuses corresponded to 15.5 weeks of gestation. At the time anti-Ro (SS-A) and anti-La (SS-B) were still positive. The patient was then placed on treatment with oral prednisilone 30 mg/day (subsequently increased in June 1987 to 50 mg/day). Repeat tests for anti-Ro (SS-A) and anti-La (SS-B) antibodies done in June, July and August 1987 were negative.

On the 28th week of her pregnancy, repeat ultrasound examination revealed one viable, apparently normal fetus, and a second, dead, fetus. A Caesarian section was performed. The first baby was a boy weighing 1630 g with an APGAR score of 1, 5 and 8. The second baby was a 170 g fetus compressus. Pathological examination revealed a diamnionic, monochorionic placenta weighing 515 g. An anti-Ro (SS-A) antibody test in the infant was negative. Repeated tests for anti-Ro (SS-A) antibody on the patient as well as the infant remained negative for 4 months postpartum. After delivery the prednisilone dosage was gradually tapered off over a period of 3 months before the drug was stopped. Both the patient and the infant have remained well at 9 months follow-up.

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Discussion

This patient had no hypertension, diabetes mellitus or infection, and no anatomical, chromosomal or hormonal abnormalities that could possibly account for the recurrent intrauterine deaths in her previous three consecutive pregnancies. An unfavourable pregnancy outcome has been reported in association with maternal autoimmune disease manifested by the presence of ANA, anti-Ro (SS-A), antiribonucleoprotein (RNP), antiphospholipid antibodies (lupus anticoagulant and anticardiolipin antibodies). These antibodies may persist for long periods of time and may even precede the onset of clinical symptoms by several years. Our patient had negative ANA and antiphospholipid antibodies, negative VDRL, normal coagulation studies, but positive anti-Ro (SS-A) and anti-La (SS-B) antibodies.

Anti-Ro (SS-A) antibodies are found in two-thirds of patients with subacute cutaneous lupus erythematosus; 60% of patients with ANA-negative systemic lupus erythematosus; 30–40% of systemic lupus erythematosus patients; and 60–70% of patients with Sjogren's syndrome. Anti-Ro (SS-A) antibody is associated with isolated congenital complete heart block, the most serious manifestation of the neonatal lupus syndrome. Postmortem studies of neonates with anti-Ro (SS-A) antibodies have demonstrated inflammation, and scarring of, and immunoglobulin deposition on, the cardiac conducting system.1 Ro antigen is also demonstrable in fetal cardiac tissue.2 This neonatal lupus syndrome also includes transient lupus dermatitis, hepatosplenomegaly and haematological abnormalities. These result from transplacental passage of maternal IgG anti-Ro (SS-A), which may result in fetal death.3,4

The almost universal presence of the anti-Ro (SS-A) antibody in the affected infants and their mothers raises the possibility of the potential pathogenic role of this antibody system. From six Ro-positive pregnancies that were studied prospectively by Lockshin,7 one resulted in a child with neonatal lupus syndrome. In a series reported by Watson8 on ten anti-Ro (SS-A) positive neonatal lupus mothers, the rate of fetal wastage was 30%. It is also recognized that increased rate of fetal wastage may occur even before the clinical detection of systemic lupus erythematosus.9 This may well be the case with our patient.

Corticosteroids have been used to treat prophylactically patients with repeated fetal loss and lupus anticoagulant and/or antibody to cardiolipin to prevent new fetal loss.7 It is also known that many autoantibodies including ANA, anti-DNA and anticardiolipin may disappear following the use of corticosteroids and other immunosuppressive therapies.

In summary, this case demonstrates the successful use of corticosteroids in preventing fetal loss due to anti-Ro (SS-A) antibody.

References