Pregnancy and Lyme Disease

John Drulle, M.D. December 1990

When a pregnant woman is infected with Lyme disease, not only is she subject to its devastation, but her baby is too. At this time there is only a small amount of information available in the medical literature. I will review the major articles, and after describing our own experiences with Lyme and pregnancy, present what I feel is a rational approach to this issue.

The first case of transplacental passage of Borrelia burgdorferi was reported in 1985 in Wisconsin by Schlesinger. The woman was bitten during her first trimester and developed an EM rash with two satellite lesions. This was followed by typical Lyme symptoms. She did not receive medical treatment as Lyme was not diagnosed at the time. She delivered a male baby at 35 weeks. The baby died 39 hours later from congestive heart failure, and at autopsy there were several major defects of the heart. Spirochetes were found in the spleen, kidneys, bone marrow and the heart. The mother tested positive for Lyme. Here we can only speculate that the Lyme might have been responsible for the birth defects, as these same types of problems can occur in non-Lyme situations.

In 1987, Dr. Alan MacDonald reported a case of a woman infected with Lyme in her first trimester of pregnancy, which unfortunately was not diagnosed or treated. She had developed a circular red rash which was followed by painful swelling of her knee. These resolved spontaneously. The woman went into labor at term, and delivered a 2,500 gram stillborn baby. Autopsy revealed a ventricular septal defect, i.e. a hole in the wall of the heart which separates the two main pumping chambers. The Lyme bacterium was cultured from the baby's liver, and it was demonstrated in the brain, heart, adrenal gland and the placenta. The mother's blood tested positive for antibodies to the Lyme spirochete and negative for syphilis. Dr. MacDonald reported three other cases of fetal death in the second trimester, in which the Lyme spirochete was cultured from the livers. None of the mothers gave any history suggesting Lyme infection.

In 1986, Weber reported a case of Lyme infection in a newborn baby. The mother had been bitten by multiple ticks during her first trimester. She developed an EM rash several weeks later. She was treated with a "standard" course of oral penicillin for seven days, three times a day. The baby was delivered at term and appeared normal. During the next 23 hours the baby developed breathing problems and died. Autopsy showed brain hemorrhages. Spirochetes compatible with Borrelia burgdorferi, the Lyme spirochete, were demonstrated in the brain and the liver. Initial testing of the mother's blood was negative for antibodies to the Lyme spirochete; however, at a later date her frozen blood tested positive for IgM antibodies by the ELISA test.

Markowitz published a study of Lyme and pregnancy in 1986. He described nineteen patients who were infected during pregnancy. Five of these had adverse outcomes (one fetal death at 20 weeks, high bilirubin level in a four-week premature baby, webbed toes, blindness and developmental delay, and a newborn rash). Thirteen of the nineteen had received antibiotics. The authors concluded that there was no proof that Lyme was responsible for the adverse outcomes since all of them were dissimilar. However, there was a consensus that this was an abnormally high frequency of adverse outcomes, and that pregnant women with diagnosed Lyme should be treated immediately with penicillin.

Williams and colleagues conducted a study in a Lyme-endemic area in New York of umbilical cord blood. Of 255 infants tested, 10.2% had detectable antibody to the Lyme spirochete. Of 166 infants born in a non-endemic area, 2.4% had detectable antibodies. The rate of birth defects did not differ significantly between the two groups; however, the first group tended to be of lower birth weight and smaller for their gestational age, and tended to have more jaundice. The authors concluded that these differences were not significantly different. A glaring flaw in this study is that it only included live births. Since miscarriages, stillbirth and perinatal infant deaths were not included, the possibility of congenital defects possibly associated with Lyme and incompatible with life are not included. Therefore, the author's contention that no association exists between gestational Lyme and congenital defects should be viewed with skepticism.

Dr. Andrea Dlesk, of the Marshfield clinic in Wisconsin, studied 143 healthy pregnant women. Lyme serologic tests were obtained on the initial and postpartum visits. At the time the data were reported, 116 women had completed their pregnancies and 12 had miscarried, one of whom tested positive. Of the 104 women who did not miscarry, 13 women tested positive for Lyme. The conclusion was that healthy women who test positive for Lyme are at no increased risk for miscarriage. Again this study is flawed in that there are no autopsy data on the 12 miscarriages. It is quite possible that, in the 11 seronegative mothers who miscarried, seronegative Lyme was present and may have caused defective fetuses. Seronegative Lyme is a real entity and nay account for 25% of all cases of Lyme.

In 1988, Carlomango studied 49 women who had either a 1st or 2nd trimester spontaneous abortion. Six (6) of them (12.2%) tested positive, compared to 3 of 49 women who delivered at term. The authors concluded that there was no statistical significance between the two groups.

In 1988, Nadal surveyed 1,416 women and their 1,434 infants at delivery for presence of antibodies to the Lyme spirochete. Twelve women tested positive (only one had a history compatible with EM during pregnancy), six had a history of pre-existing Lyme and five had unremarkable histories. Of these twelve women, seven had remarkable outcomes:

1. Two had elevated bilirubinemia
2. One had muscle hypotonia (laxness)
3. One was post-term, small for age, and evidenced chronic placental insufficiency

4. One had transient macrocephaly (large head)

5. One had transient supraventricular extrasystoles ("skipped heart beats")

6. The infant born of the mother with EM had a VSD-hole in the heart connecting the two main pumping chambers.

Since none of these babies had positive blood tests for antibodies to Lyme, the conclusion was that the adverse outcomes were not due to Lyme. The major flaw in this conclusion is the assumption that congenital Lyme babies are seropositive. This has been refuted by the work of Dr. Alan MacDonald, and is analogous to the findings of seronegativity in congenital syphilis.

In 1989, Dr. Alan MacDonald reported his findings in autopsies performed following perinatal deaths at Southhampton Hospital between 1978 and 1988. It must be noted that routine pathology studies on tissues will not demonstrate the Lyme spirochete. Unless there is a high index of suspicion for Lyme disease, the special silver or immunologic stains which can identify the spirochete are not used. He also reports four cases where there was live birth and spirochetes were demonstrated in the placentas. In the group of perinatal deaths there was no history or eveidence of Lyme disease in the mothers. Their blood tests were negative in all but one case. Spirochetes compatible with Borrelia burgdorferi were identified in the vital organs and numerous developmental defects were observed. Dr. MacDonald's conclusions are:

1. Tissue inflammation is not seen in fetuses with transplacentally acquired infection with the Lyme spirochete.

2. Lyme disease acquired in utero may result in fetal death in utero, fetal death at term or infant death after birth. Babies may also survive in spite of the bacteria being isolated in the placenta.

3. In all but one of these cases where the Lyme organism was identified in the placenta or the fetal tissues, the maternal blood had no evidence of antibodies to the Lyme bacteria. In only two of the fourteen cases was there a maternal history compatible with Lyme disease, yet neither of the two were serologically confirmed.

This is the extent of the currently available information on Lyme and Pregnancy in the medical literature in 1990. Comparing the various studies have led us to arrive at the following conclusions:

1. Lyme disease is a serious threat to pregnant women in that it may cause fetal damage and death.

2. Pregnancy may mask symptoms of Lyme in the mother and may result in seronegativity.

3. Serologic screening of pregnant women in highly endemic areas is not recommended.

4. Pregnant women who test positive for Lyme antibodies, yet have no symptoms suggesting active Lyme, are probably at a lower risk of passing the infection across the placenta. It may be possible that the presence of antibody prevents the Borrelia burgdorferi from crossing the placenta.

5. Babies born with Lyme disease can be expected to have a negative blood test for Lyme antibodies. Few have positive test.

6. We desperately need a better test for detecting Lyme in pregnant women. It is clear that serologies are inadequate. Efforts should be directed at evaluating urine antigen and PCR testing in pregnancy and in neonates.

In our practice we have seen several dozen pregnant women with Lyme disease. I feel that a much more aggressive approach must be taken with them than with non-pregnant patients. It is preferable to err on the side of overtreatment than undertreatment, especially since the antibiotics we use have not been associated with birth defects or adverse effects on the developing fetus. These are general recommendations that we have developed over the last three years:

1. A pregnant woman who presents with a deer tick bite in an endemic area for Lyme disease is treated as if she had Stage 1 Lyme disease. We would treat with one to two months of oral antibiotics, such as Amoxicillin or Ceftin. (Tetracycline and Doxycycline are contraindicated in pregnancy.)

2. A pregnant woman with an EM rash should receive three to four weeks of intravenous Claforan, Rocephin or aqueous penicillin. We have evidence that even without constitutional symptoms the Lyme spirochete may have spread throughout the mother's body by the fifth day after an infected tick bite. As noted above, treatment failure with oral penicillin has been reported.

3. Pregnant women who are diagnosed as having Lyme by symptoms and blood tests, who do not have a clear history of a tick bite or EM rash, and have not yet been treated, should be treated with intravenous antibiotics. Here, since the length of infection is unknown, we must assume that the spirochetes have spread throughout the mother's body. It has generally been assumed that it is only possible to culture the Lyme spirochete from the blood only in the early stages of Lyme disease, so that a woman in the later stages of Lyme is safe from having blood-borne spirochetes reaching and crossing the placenta to the fetus. Yet unpublished data suggests that blood drawn from chronic Lyme patients during the afternoon, when they usually spike a mild fever, may yield spirochetes, using a specially modified BS Kelly culture Medium. Animal studies with chronically infected dogs show that when their immune systems are suppressed by injecting them with dexamethasone, a steroid similar to prednisone, it is possible to culture the Lyme spirochete from their blood the day after the injection. It may be possible that the state of pregnancy, which is also immunosuppressive, may induce the spirochete to enter the bloodstream and reach the placenta.

4. We recommend that pregnant women with active Lyme, or a history of treated Lyme, have monthly urine antigen tests for Lyme until the seventh month of pregnancy. There is some evidence that during the 3rd trimester, false positive urine tests may occur.

5. When the baby is delivered, we recommend that the placenta be examined for spirochetes. If spirochetes are demonstrated in the placenta, the baby should be treated with intravenous antibiotics.

I must again stress that these are guidelines that we use in our own practice. I realize that many physicians might criticize them fro being an over-reaction and too aggressive: however, I have seen a number of babies born with congenital Lyme, and am quite aware of the devastating effects it can cause. Following the recommendations I've outlined above, we have had normal outcomes in all the pregnant women whom we have treated.

Written by John Drulle, M.D. in December, 1990 and reprinted by the John Drulle, MD Memorial Lyme Fund, Inc. in 2006.