Lyme Disease and Pregnancy/Gestational Lyme

Mothers’ Quest for the Truth

By Sue Faber, RN, BScN
Co-Founder of LymeHope

November 3rd 2017.
• No conflicts of interest
• Registered Nurse, BScN
• Co-founder of LymeHope
Vertical/Transplacental Transmission of Disease

A vertically transmitted infection is caused by active pathogens such as bacteria or viruses that are *transmitted directly from the mother to an embryo, fetus or baby* during pregnancy or childbirth.

Congenital Disease:

The term *congenital infection* can be used if the vertically transmitted infection persists in the baby after childbirth – causing chronic postnatal infection.

Clinical evidence of infection may be seen at birth, soon afterward, or not until years later – this latency has been noted in both HIV and Syphilis.

https://en.wikipedia.org/wiki/Vertically_transmitted_infection
**Borrelia Duttoni** – Relapsing Fever transmitted by soft ticks– complications of pregnancy, miscarriage and documented neonatal death and congenital infection in sub-Saharan Africa.

**Borrelia Hermsii** – Tick Borne Relapsing fever (TBRF) transmitted by soft ticks– associated with pregnancy complications including spontaneous abortion, premature birth and neonatal infection, perinatal mortality.

**Syphilis (Treponema Pallidum)**, a spirochete – well known to be transmitted through pregnancy (prenatal screening for syphilis in Canada). Borrelia Burgdorferi (Lyme) is taxonomically related to syphilis - both spirochetes.

What about **Borrelia Burgdorferi** – Lyme Disease? Can Bb be transmitted in the womb causing congenital disease?

**Simple answer = YES**
‘TORCHES CLAP’

Table 1-4  Suggested Acronym for Microorganisms Responsible for Infection of the Fetus: TORCHES CLAP

| TO  | Toxoplasma gondii |
| R   | Rubella virus     |
| C   | Cytomegalovirus   |
| H   | Herpes simplex virus |
| E   | Enteroviruses     |
| S   | Syphilis (Treponema pallidum) |
| C   | Chickenpox (varicella-zoster virus) |
| L   | Lyme disease (Borrelia burgdorferi) |
| A   | AIDS (HIV)        |
| P   | Parvovirus B19    |

TO - Toxoplasma gondii  
R – Rubella virus  
C – Cytomegalovirus  
H – Herpes simplex virus  
E – Enteroviruses  
S – Syphilis  
C – Chickenpox  
L – Lyme Disease  
A – AIDS (HIV)  
P – Parvovirus B19

Maldonato, Y, Nizet, V, Klein, J, Remington, J, Wilson, C.  
Current concepts of Infections of the Fetus and Newborn Infant.  
Canada Diseases Weekly Report
June 4, 1988

What were the adverse pregnancy outcomes?
Gestational Lyme Disease Case Studies of 102 Live Births (Retrospective Analysis)

102 case studies of children with Gestational Lyme

• **Majority of mothers** diagnosed prior** to the child’s diagnosis**

• Progression of symptoms - many initial symptoms were present in infants but overlooked until progression in frequency and severity.

• Children were typically diagnosed between 1 and 5 years of age.

• **Diagnosis was clinical**: based on history and physical

• Lab results including EIA, WB, PCR, culture were used to support clinical diagnosis and/or to help describe the study population

• 66% of mothers had difficult pregnancy – false labor, nausea, vomiting, fevers, severe fatigue, history of miscarriage, inability to function

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Percentage</th>
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</thead>
<tbody>
<tr>
<td>Fatigue, lack of stamina</td>
<td>72%</td>
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<tr>
<td>Joint pain</td>
<td>69%</td>
</tr>
<tr>
<td>Low grade fevers</td>
<td>59%</td>
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<tr>
<td>Hyperactivity, lack of concentration</td>
<td>56%</td>
</tr>
<tr>
<td>Jointed sensitivity</td>
<td>55%</td>
</tr>
<tr>
<td>Irritability and mood swings</td>
<td>54%</td>
</tr>
<tr>
<td>Headaches</td>
<td>50%</td>
</tr>
<tr>
<td>Pale and sickly – dark eye circles</td>
<td>42%</td>
</tr>
<tr>
<td>Photophobia (sensitive to light)</td>
<td>43%</td>
</tr>
<tr>
<td>Poor memory</td>
<td>39%</td>
</tr>
<tr>
<td>Hyperacuity (sensitive to noise)</td>
<td>36%</td>
</tr>
<tr>
<td>Vertigo</td>
<td>30%</td>
</tr>
<tr>
<td>Diarrhea and constipation</td>
<td>32%</td>
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<tr>
<td>Abdominal pain</td>
<td>29%</td>
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<tr>
<td>GERD</td>
<td>27%</td>
</tr>
<tr>
<td>Night sweats</td>
<td>23%</td>
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<tr>
<td>Nausea</td>
<td>23%</td>
</tr>
<tr>
<td>Cardiac manifestations – palpitations, PVC, Mitral VP, heart murmur</td>
<td>23%</td>
</tr>
<tr>
<td>Generalized muscle pain or spasms</td>
<td>23%</td>
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<tr>
<td>Anger and rage</td>
<td>23%</td>
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<tr>
<td>Anxiety</td>
<td>21%</td>
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<tr>
<td>Speech delay</td>
<td>21%</td>
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<tr>
<td>Reading and writing delay</td>
<td>19%</td>
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<tr>
<td>Developmental delays</td>
<td>18%</td>
</tr>
<tr>
<td>Photophobia (sensitive to light)</td>
<td>14%</td>
</tr>
<tr>
<td>Auditory/visual processing problems</td>
<td>13%</td>
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<tr>
<td>Aggression or violence</td>
<td>13%</td>
</tr>
<tr>
<td>Depression</td>
<td>13%</td>
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<tr>
<td>Word selection problems</td>
<td>12%</td>
</tr>
<tr>
<td>Tic disorders</td>
<td>14%</td>
</tr>
<tr>
<td>OCD</td>
<td>11%</td>
</tr>
<tr>
<td>Seizure disorder</td>
<td>11%</td>
</tr>
<tr>
<td>Involuntary movements</td>
<td>9%</td>
</tr>
<tr>
<td>Motion sickness</td>
<td>9%</td>
</tr>
<tr>
<td>Autism</td>
<td>9%</td>
</tr>
<tr>
<td>Dyslexia</td>
<td>8%</td>
</tr>
<tr>
<td>Suicidal thoughts</td>
<td>7%</td>
</tr>
<tr>
<td>Hypotonia at birth (floppy, poor muscle tone)</td>
<td>7%</td>
</tr>
</tbody>
</table>

The insidious nature of gestational LD can present a complicated diagnosis due to:

- the delay of presentation
- the multi-systemic often transient nature of symptoms that can vary in severity and change with progression of disease
- unreliability of standard diagnostic tests.
and specific for the confirmation of Lyme disease. Patients with other infections either previous or acute infections caused by spirochetes, viruses or bacteria, may have circulating antibodies that cross-react with Borrelia antigens, thus producing positive results. Therefore, positive test results provide supportive evidenced to clinicians, but not sole evidence, for the diagnosis of Lyme disease.

Regarding your concerns on congenital transmission of Lyme disease, our Health Department Information Specialist conducted a literature search on this topic. Although a significant amount of research was conducted, no scientific evidence to support congenital Lyme disease transmission in the scientific literature was found. The literature search is attached for your information.

If you still have health concerns for either you or your daughter, please continue to seek medical advice from your health care provider.

Regards,

Halton Region Public Health Department, Ontario, January 25, 2017

‘No scientific evidence to support congenital Lyme disease transmission in the scientific literature was found.’
1. It is clear that Bb can be transmitted in the blood of infected pregnant women across the placenta into the fetus
2. Transmission resulted in congenital infections and fetal demise
3. Spirochetes were recovered from infant’s tissues including brain, spleen and kidney
4. Cardiac abnormalities seen
5. Inflammation changes not pronounced but possibly due to immature immune system.

LYME DISEASE IN MATERNAL INFECTIONS

It is clear that *B. burgdorferi* can be transmitted in the blood of infected pregnant women across the placenta into the fetus. This has now been documented with resultant congenital infections\(^2\text{8}\) and fetal demise.\(^2\text{9}\) Spirochetes can be recovered or seen in the infant’s tissues including the brain, spleen and kidney. The chorionic villi of the placenta show an increase in Hofbauer cells as in luetic placentitis. Inflammatory changes of fetal or neonatal changes are not as pronounced as in the adult, but cardiac abnormalities, including intracardiac septal defects, have been seen.\(^2\text{8,29}\) It is not known why inflammatory cells are so sparse from maternal transmission, but it is possible that an immature immune system plays a role.
Maternal-Fetal Transmission of the Lyme Disease Spirochete, *Borrelia burgdorferi*

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Hennepin County Medical Center and the University of Minnesota Medical School, Minneapolis, Minnesota; Yale University School of Medicine, New Haven, Connecticut.

LYME DISEASE usually begins with a characteristic skin lesion, erythema chronicum migrans, accompanied by "influenza-like" or "meningitis-like" symptoms (1). Some patients later develop cardiac abnormalities such as atrioventricular heart block or myopericarditis, neurologic complications, or intermittent attacks of arthritis (1). The causative agent, the Lyme disease spirochete *Borrelia burgdorferi* (2), is transmitted by *Ixodes dammini* or related ixodid ticks (3). Antibiotic treatment with tetracycline or penicillin is usually curative (4).

We report the case of a woman who developed Lyme disease during the first trimester of pregnancy. She did not receive antibiotic therapy. Her infant, born at 35 weeks gestational age, died of congenital heart disease during the first week of life. Histologic examination of autopsy material showed the Lyme disease spirochete in the spleen, kidneys, and bone marrow.

A 28-year-old mother of two healthy children became pregnant for the third time in September 1983. Soon thereafter, she participated in outdoor activities in an area of northwestern Wisconsin known to be endemic for Lyme disease (3). On 7 November 1983, she noted an expanding annular skin lesion in the left popliteal region reaching a size of 20 × 30 cm. She also developed two secondary skin lesions, headache, stiff neck, arthralgias, malaise, and insidious lymphadenopathy. All symp-
**Maternal-Fetal Transmission of Lyme Disease Spirochete - Schlesinger et al, 1985**

- 28 yr old woman with Lyme disease - first trimester of pregnancy/EM rash – camping in Wisconsin
- **NO** antibiotic therapy
- Lyme serology IFA positive postpartum in mom.
- Infant born at 35 weeks with respiratory distress **died after 39 hours**
- **Autopsy** of infant showed **widespread** cardiac abnormalities
- **No evidence of inflammation** in tissues/organs
- **Lyme disease Spirochetes** (Bb) were identified in **spleen, renal tubules/kidney and bone marrow**.
- Dr Alan Macdonald later demonstrated Bb in the myocardium by **IHC - immunohistochemicals technique**.

**Stillbirth Following maternal Lyme Disease Macdonald et al, 1987**

- 24 year old mother with Lyme disease -first trimester of pregnancy bitten/EM rash – hiking in Utah
- **No** antibiotic therapy
- Stillborn delivered at term
- Lyme serology (IFA/ELISA) on postpartum maternal blood was positive at 2 of 3 laboratories
- **Autopsy** revealed heart defect
- **No significant inflammation** in tissue
- **Spirochetes cultured** from fetal liver and identified in **heart, adrenal gland, placenta and mid brain** using histological techniques
- ‘**Overwhelming spirochetosis** in the fetus’
Schlesinger Autopsy –
Bb in fetal Heart tissue

Bb Spirochete in Fetal Kidney

**Author conclusions and recommendations:**

**Schlesinger et al**

- **Pregnant women** who acquire Lyme disease should be **treated promptly** with antibiotics.
- At childbirth, **placenta should be examined** for histologic abnormalities as well as spirochetes.
- Lyme spirochete **may spread transplacentally** to organs of the fetus.
- If infant is ill, **diagnosis of congenital Lyme should be considered.**

**Macdonald et al**

- **Clinical diagnosis** of probable Lyme must be the ‘gold standard of diagnosis’ because Lyme serologic studies may be non-diagnosistic.
- Even classical symptoms (EM rash) may be overlooked by the patient and her physician.
- **Pathologists urged to search for spirochetes in tissues of stillborn fetuses who have malformations in cardiovascular system.**

*Two cases of Transplacental Transmission = Association with Fetal death and Cardiac Malformation.*

Different anomalies were found in each case; therefore a cause and effect relationship **cannot be determined** between the spirochete in tissue and a specific lesion.
- Nineteen women infected with Lyme during pregnancy
- **Five 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
- Consensus that this was an **abnormally high frequency of adverse outcomes**, and that pregnant women with diagnosed Lyme disease should be treated immediately with antibiotics.
- Authors concluded that there was no proof that Lyme disease was responsible for the **heterogeneous range** of adverse outcomes.

Bb could not be implicated as the exclusive causative agent in any adverse outcomes.
Case Report #3 Culture Positive, Seronegative Transplacental Lyme Borreliosis 1987.

- Mother from California (low-endemic region)
- **No** antibiotic therapy, no clear onset for Lyme
- Neonatal distress born by C-section
- Infant initially appeared healthy and discharged
- Readmitted at 8 days with profound lethargy and progressive multisystem failure, died.
- Upon autopsy Borrelia Burgdorferi was cultured from a frontal cerebral cortex.
- Silver stain of brain and heart confirmatory of tissue infection.
- Mother and infant were **seronegative** for LB by ELISA at Yale

Case # 4 Borrelia Burgdorferi in Newborn despite oral antibiotics for Lyme in Pregnancy – 1988, Germany

- 37 year old woman bitten by ticks in Germany in 1984 first trimester of pregnancy, - late July developed EM rash.

- Treated with a 7 day course of oral penicillin

- Negative serology IgG and IgM antibody by IFA for mother – first in August and repeated in October.

- Delivered a healthy baby after healthy pregnancy.

- 23 hours after birth the child had respiratory distress and died

- Bb found in the brain and liver of deceased neonate.

- No inflammation seen in any organ examined including heart, liver brain and kidney
Comprehensive review of 14 cases of adverse fetal and neonatal outcomes of gestational borreliosis.

Autopsy and clinical studies have associated gestational Lyme borreliosis with various medical problems including:

- fetal death
- hydrocephalus
- cardiovascular anomalies
- neonatal respiratory distress
- hyperbilirubinemia
- intrauterine growth restriction,
- cortical blindness
- sudden infant death syndrome
- maternal toxemia of pregnancy.

‘It is my expectation that the spectrum of gestational Lyme borreliosis will expand into many of the clinical domains of prenatal syphilis.’
3 Observations from cases of Fetal Borreliosis

1: Tissue inflammation is absent in fetuses with transplacentally acquired Bb infection

2: Gestational Lyme Borreliosis may be associated with fetal death in utero, fetal death at term, or infant death after birth

3: Maternal blood is seronegative for specific antibodies against Bb in cases where the spirochete can be demonstrated in the fetus or placenta
Bb spirochetes found in autopsy of infant brain age 4 months Sudden Infant Death Syndrome (SIDS)

**Serosurveys**

**Nadal et al (1989):** Serosurvey from 1986-1987 in Switzerland. Seropositivity of 0.85% (12 of 1416) in maternal sera. One mom had a first trimester tick bite and Lyme Borreliosis and her infant was born with congenital ventricular septal defect. Other adverse outcomes reported but babies serology negative. *authors conclude that adverse outcomes not due to Lyme – but possible flaw in this research is the assumption that babies born with congenital Lyme disease are seropositive.*

**Isukova et al (1994):** Serosurvey of 1039 pregnant women in Russia between 1992 and 1994. Seropositivity in 5.5% (57 of 1039). Data indicated that LB is a serious risk factor for miscarriage and perinatal death but provided no information on individual outcomes.
• Interesting differences/observations in seropositive population pregnancy outcomes.
• Higher incidence of low birth weight, birth size small for gestational age and APGAR score less than 7 in seropositive women. (small sample size).

• Authors noted that the incidence of cardiac defects was two times higher born to mothers in high versus low endemic areas.
• Authors noted there was an association of minor malformations with a history of maternal tick bite less than 3 years before conception.

• Maternal B Burgdorferi exposure was 5-10 times higher in endemic vs non-endemic cohort
• Infants from endemic area had 13% incidence of congenital cardiac defects and murmurs compared to 5% in non-endemic cohort.
• Authors note that late developmental sequelae would not be detected by this study owing to absence of long term follow-up.
Maternal Lyme Borreliosis and Pregnancy Outcome – Lakos, Solymosi, 2009 – Hungary – A retrospective review from 95 women with Lyme Borreliosis during pregnancy over 22 yr

A statistically significant association between untreated LB and adverse outcomes

Placentas and offspring were not tested for Bb by PCR or culture in the study and therefore it could not be concluded that adverse outcomes were a result of Bb infection of fetus or placenta.

Table 1
Adverse outcomes in 20 pregnancies

<table>
<thead>
<tr>
<th>Adverse outcome</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous abortion</td>
<td>6</td>
</tr>
<tr>
<td>Stillbirth</td>
<td>1</td>
</tr>
<tr>
<td>Premature birth</td>
<td>1</td>
</tr>
<tr>
<td>Small for dates</td>
<td>1</td>
</tr>
<tr>
<td>Cavernous hemangioma</td>
<td>4</td>
</tr>
<tr>
<td>Neonatal jaundice requiring exchange transfusion</td>
<td>2ᵃ</td>
</tr>
<tr>
<td>Dysplasia coxae</td>
<td>2</td>
</tr>
<tr>
<td>Pyloric stenosis</td>
<td>1ᵇ</td>
</tr>
<tr>
<td>Papulovesicular eruption at birth</td>
<td>1</td>
</tr>
<tr>
<td>Cerebral bleeding</td>
<td>1</td>
</tr>
<tr>
<td>Muscular hypotonicity</td>
<td>1</td>
</tr>
<tr>
<td>Hypospadias</td>
<td>1ᶜ</td>
</tr>
<tr>
<td>Skeletal anomaly</td>
<td>1</td>
</tr>
</tbody>
</table>
Case # 5

**Wife acquired tickbite in 1968** resulting in early EM rash and later chronic symptoms including meningitis, depression, chronic arthritis.

Her husband had **no memory of primary infection** but remembers recurrent flu like symptoms a year after infection of his wife, he developed **progressive symptoms** including ventricular arrhythmias.

In 1969 a son was born **suffered from multiple symptoms** including weakness, recurrent fevers and irritability and depression.

All three patients **tested positive for Bb by immunoblotting** (WB) at clinic for Bb associated disorders.

Case report represents the only report in literature of a **whole family infected and ill** with Lyme disease for a long period of time.

Raises questions of **sexual and transplacental** transmission.
Infant presents to Pediatric Dermatology with multiple annular erythematous patches, fever and lymphadenopathy which had started at 3 weeks of age and were relapsing/remitting. No history of tick bite.

32 yr mother no recall of any tick bite and no symptoms during pregnancy but had taken part in outdoor activities in area known to be endemic for LB. Postpartum serum antibody to Bb was elevated – indicative of maternal exposure to LB

Initial infant serology 9 months negative. 13 month seroconversion by WB – IgG.

B- Burgdorferi was isolated and detected by PCR from skin biopsy samples

Despite repeated courses of oral antibiotic therapy, lesions recurred multiple times over the following 3 years and child was retreated each time (this suggests persistence of Bb infection). By age 4, no further lesions documented.

Authors suggest a congenital borreliosis and cutaneous manifestations of congenital spirochetosis

Detection of Borrelia Burgdorferi in breastmilk

Breast milk from two untreated lactating women with EM rash was tested and **Bb DNA found by PCR**. In one of these patients Bb was also cultured from a skin biopsy.

No reports of Bb transmission via breastmilk in humans. – however one might consider the **Precautionary Principle** – when there isn’t proof beyond reasonable doubt and yet reason and rationale come into play.

Review of 263 cases of Gestational Lyme Borreliosis and compilation of adverse outcomes

- 888 citations

- Most comprehensive, extensive and thoughtful review of Lyme Borreliosis in the Fetus and Newborn Infant
‘Although a **homogeneous congenital Lyme borreliosis syndrome** has not yet emerged, there are **several features that are common** among the 66 adverse outcomes of pregnancies complicated by gestational Lyme Borreliosis’

- Miscarriage during the first 20 weeks gestation with high frequency of fetal cardiac abnormality, stillbirth, perinatal death

- **Severe early congenital infection** with neonatal sepsis and meningioencephalitis and high frequency of cardiac abnormality

- **Mild early congenital infection** with growth retardation and mild cardiac abnormality

- **Later onset chronic progressive infection**

  - **Late congenital infection** with
    1. growth retardation,
    2. developmental delay,
    3. neurologic,
    4. cutaneous,
    5. dental and
    6. skeletal involvement.

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- **Mild early congenital infection** – usually in the first two weeks of life
- **Severe early congenital infection** usually first week of life
- **Later onset chronic progressive infection** – usually noted between 2 weeks and under two years.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Mild Early</th>
<th>Severe Early</th>
<th>Late</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset</td>
<td>Usually first 2 weeks of life</td>
<td>Usually first week of life</td>
<td>Usually &gt; 2 wks and &lt; 2 yrs of age</td>
</tr>
<tr>
<td>Maternal gestational Lyme borreliosis</td>
<td>Usually first or second trimester</td>
<td>Usually first or second trimester</td>
<td>Usually second or third trimester</td>
</tr>
<tr>
<td>Signs and symptoms</td>
<td>Mild suspected sepsis or meningoencephalitis, Hyperbilirubinemia, Adenopathy, Rash, Intrauterine growth retardation, Miscellaneous anomalies (eg, genitourinary [GU], skeletal, cardiac)</td>
<td>Severe suspected sepsis or meningoencephalitis, Respiratory distress, Perinatal death, Intrauterine growth retardation, Fever, Rash</td>
<td>Subacute illness, Developmental delay/meningoencephalitis, Growth retardation/failure to thrive, Prematurity, Fever, Adenopathy, Rash</td>
</tr>
<tr>
<td>Prematurity?</td>
<td>&lt; 4 weeks</td>
<td>&lt; 5 weeks</td>
<td>--</td>
</tr>
</tbody>
</table>
Dr Tessa Gardner – Review of Pathologic Findings.

- **13** - pathologic or culture findings in gestational Lyme disease *placentas or decidua*
- **19** - neonatal pathologic findings in congenital Lyme borreliosis
- **2** - skin biopsies in congenital Lyme borreliosis
- **2** - brain pathologic and culture findings in SIDS of suspected Lyme borreliial etiology
- **11** - Major cardiac malformations in fetuses or infants with congenital Lyme borreliosis
- **7** - Neuropathology in fetuses or infants with fatal congenital Lyme borreliosis
- **5** - Musculoskeletal abnormalities found in term or near term infants with congenital Lyme borreliosis
- **5** - Renal histopathology reported in fetuses or infants with fatal congenital Lyme borreliosis.

- Spirochetes have been found by culture, silver stain or Bb specific IFA in autopsied organs of congenitally infected fetuses and neonates in the Liver, spleen, kidney, heart, bone marrow, brain

- Although relatively few cases of congenital Lyme borreliosis have been studied pathologically, comparisons with congenital syphilis may be appropriate

‘In order for infants with congenital Lyme borreliosis to be recognized/diagnosed, it is essential for clinicians caring for newborns and infants to become familiar with the various manifestations of Lyme borreliosis in the adult, as well as in the congenitally infected infant.’

‘The lack of inflammatory findings even when spirochetes were present has been remarkable and could be related to the immunopathogenetic features of B. Burgdorferi infection in which the spirochete is able to spread and persist in tissues without eliciting a prominent host immune response.’ - Dr Tessa Gardner – pg 555.

‘Because of bound host derived enzymes, the spirochete is invisible to, and able to evade the host immune response, in a mechanism refereed to as stealth pathogenesis. This may explain the paradox of the ability of B Burgdorferi to persist in skin and other tissues for long periods of time with only minimal mononuclear cell infiltration despite eliciting a strong immune response that, in vitro, is capable of killing it.’

Pg 553

‘Serology does not appear to be a sensitive method of diagnosis and reliance on seropositivity leads to misdiagnosis of the majority of congenitally infected infants.’

**Tetrogenicity:** ‘It is uncertain whether Bb is teratogenic, although there is an indication that there may be an increased risk of congenital cardiac malformations after first and early second trimester gestational Lyme borreliosis’.

**Adverse events:** ‘It is possible that Bb gestational infection with transplacental dissemination could cause fetal pathology simply by causing Lyme borreliosis with the same manifestations (cutaneous, musculoskeletal, neurologic, neuropsychiatric, neurocognitive and urologic) that it produces in children and adult patients which would explain some of the adverse events reported.’

How do you know if an infant is infected with Lyme?

- Negative serology (ELISA and WB) does not indicate lack of infection in the infant.
- Most diagnoses on fetal/neonatal deaths were made on histology samples.
- **PCR on the cord or infant blood.**
- **Elispot** – cellular response in T cells or I-spot
- **Urine** – urinary OSP A test
- Dr. Jones also suggests **PCR testing placentas, foreskin remnants** from circumcised male infants.
- **Bb culture on cord blood**
- **Placenta examined** for histologic abnormalities/spirochetes
- **Prenatal screening tool/Questionnaire (Horowitz Questionnaire)** can be used by OB and midwives and Fam MD’s
NEXT STEPS – Research, Study and Solutions

- Retrospective questionnaires/surveys

- Evaluate short and long-term outcomes (infants, stillborns, miscarriages) of pregnancies complicated by Lyme disease.

- Large-scale long-term prospective follow-up study of mother-baby pairs to determine maternal cofactors related to maternal-infant transmission; early diagnostic methods to identify Borrelia-infected infants; and maternal and infant outcomes, including occurrence of possible early and late sequelae of congenital Lyme borreliosis

- B. Burgdorferi specific evaluation of any fetal or neo-natal demise
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