Lyme disease in Canada: Focus on children

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Abstract
Lyme disease, the most common tick-borne infection in Canada and much of the United States, is caused by the bacteria Borrelia burgdorferi. Peak incidence for Lyme disease is among children five to nine years of age and older adults (55 to 59 years of age). The bacteria are transmitted through the bite of infected black-legged ticks of the Ixodes species. The primary hosts of black-legged ticks are mice and other rodents, small mammals, birds (which are reservoirs for B burgdorferi) and white-tailed deer (Figure 1). Although dogs can contract LD and carry ticks into homes and yards, there is no evidence that they spread the infection directly to people. [2]

Peak incidence for LD is among children five to nine years of age and older adults (55 to 59 years of age), and many cases likely go unreported.[1] No relationship between treated maternal LD and abnormal pregnancies or disease in infants has been documented.[3] Although there is a theoretical risk, no case of infection has been linked to blood transfusion.[4]

Ticks cannot jump or fly. Instead, they climb and wait on tall grasses or shrubs for a potential host to brush against them. They then transfer to the host and seek an attachment site.[5] Immature ticks (nymphs) are responsible for most human LD infections because their very small size hinders detection.[1]

If a tick is found attached to or feeding on a child, remove it as soon as possible. Ticks can attach and feed for five days or longer (Figure 2). Removing a tick within 24 h to 36 h of its starting to feed is likely to prevent LD.[6]

Key Words: Black-legged tick; Borrelia burgdorferi; Erythema migrans; Post-treatment Lyme disease syndrome

Lyme disease (LD), a serious disease, is the most common tick-borne infection in Canada and the Northeastern to Midwestern United States, with cases also occurring (with less frequency) on the west coast. LD is caused by the bacteria spirochete, Borrelia burgdorferi, transmitted to humans through the bite of infected black-legged ticks: Ixodes scapularis in Eastern and Central Canada and Ixodes pacificus in British Columbia.[1]

The primary hosts (carriers) of black-legged ticks are mice and other small rodents, small mammals, birds (which are a reservoir for B burgdorferi) and white-tailed deer (Figure 1). Although dogs can contract LD and carry ticks into homes and yards, there is no evidence that they spread the infection directly to people. [2]
What are the clinical manifestations of LD?
Clinical manifestations are divided into early localized, early disseminated and late disease.

**Early localized disease**: Erythema migrans (EM) – a rash at the site of a recent tick bite – is the most common presentation in children and adults (Figures 3 and 4). EM typically develops seven to 14 days (range three to 30 days) after a tick bite. EM begins as an erythematous macule or papule that rapidly expands centrifugally, sometimes with central clearing. The lesions may be round or oval, flat or slightly raised, and are typically ≥5 cm in diameter. EM is usually painless and not pruritic. However, fever, malaise, headache, mild neck stiffness, myalgia and arthralgia often accompany EM.

Early LD can occur without rash, and rash may not be detected by all patients in which it occurs. Without treatment, EM resolves spontaneously over a four-week period, on average.

**Early disseminated disease**: Approximately 20% of children with LD first present to a healthcare provider with multiple, rather than single, EM lesions. This rash usually occurs several weeks after the tick bite and consists of secondary annular, erythematous lesions similar to but typically smaller than the primary lesion. These lesions reflect spirochetemia with cutaneous dissemination.

Other manifestations of early disseminated disease (with or without rash) include acute neurological signs, such as facial nerve palsy, papilledema and lymphocytic meningitis. Children with facial palsy should be assessed for meningitis, especially if neck stiffness or severe headache occurs. Lyme carditis, resulting in heart block, is rare in children.

**Late disease**: Children treated with antimicrobial agents in the early stage of LD very rarely develop late disease. The most common late-stage symptoms are pauciarticular arthritis affecting large joints, especially the knees, which may manifest weeks to months (mean four months) after the tick bite. Arthritis can occur without a history of earlier stages of illness. Periph-
eral neuropathy and central nervous system manifestations can also occur, although rarely during late disease in children.[4]

![Clinical manifestations of confirmed Lyme disease cases (United States, 2001 to 2010). The most common presentation is the erythema migrans rash. Other symptoms are less common. Some individuals experienced >1 symptom. Reproduced from reference 8 with permission from the United States Centers for Disease Control and Prevention (Atlanta, USA)](image-url)

**Figure 3**

**How is the diagnosis of LD made?**

**Early localized disease:** In general, the diagnosis of LD is principally clinical, supported by a history of potential tick exposure in an area where it is known or suspected that black-legged ticks have been established. However, because tick populations are expanding, it is possible that LD can be acquired outside of currently identified areas. Such a possibility should be considered when assessing patients. Patients with clear symptoms of early LD should be diagnosed and treated without laboratory confirmation,[1][4][10][11] because antibodies against *B. burgdorferi* are often not detectable by serodiagnostic testing within the first four weeks after infection.[4][12][13] All clinical manifestations of possible LD, except EM, require laboratory confirmation.[13]

**Early disseminated and late disease:** Two-tiered serological testing, including an ELISA screening test followed by a confirmatory Western blot test is used to supplement clinical suspicion of disseminated or late LD (Figure 5). Two-tiered testing is necessary because the ELISA may yield false-positive results from antibodies directed against other spirochetes, viral infections or autoimmune diseases.[1] Table 1 provides information related to the performance characteristics of serological assays in different clinical presentations of LD.[6][14]

Supplemental tests can detect *Borrelia* species that cause LD outside of North America. Therefore, travel history should be documented.[1]

Some individuals treated with antimicrobials for early LD never develop antibodies against *B. burgdorferi*. They are cured.[4][13]

Most individuals with early disseminated disease and almost all individuals with late disease have antibodies against *B. burgdorferi*. Once such antibodies develop, they persist for years. A decline in antibody levels is not useful to assess treatment response.[1][5] Serological test results for LD should be interpreted along with careful consideration of the clinical setting and quality of the testing laboratory.[1][15] Tests of joint fluid for antibody to *B. burgdorferi* and urinary antigen detection have no role in diagnosis.[3] In suspected Lyme meningitis, testing for intrathecal immunoglobulin M or immunoglobulin G antibodies may be helpful.[1][5][14]

![Erythema migrans rash showing the classic ‘bull’s eye’ form. Reproduced from reference 1 © All rights reserved. With permission from the Minister of Health, 2014](image-url)

**Figure 4**
Figure 5) Two-tiered serological testing for Lyme disease. Source: Dr L Robbin Lindsay, Research Scientist, Field Studies (for the Public Health Agency of Canada’s Lyme Disease Surveillance Group). EM Erythema migrans; Ig Immunoglobulin

**TABLE 1**

Performance characteristics of serological assays in Lyme disease

<table>
<thead>
<tr>
<th>Test</th>
<th>EM, acute</th>
<th>EM, convalescent*</th>
<th>Neurological involvement</th>
<th>Arthritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole-cell ELISA</td>
<td>33–49</td>
<td>75–86</td>
<td>79 (IgG only)</td>
<td>100 (IgG only)</td>
</tr>
<tr>
<td>IgM WB</td>
<td>43–44</td>
<td>75–84</td>
<td>80</td>
<td>16</td>
</tr>
<tr>
<td>IgG WB</td>
<td>0–13</td>
<td>15–21</td>
<td>64–72</td>
<td>96–100</td>
</tr>
<tr>
<td>Two-tier testing</td>
<td>29–40</td>
<td>29–78</td>
<td>87</td>
<td>97</td>
</tr>
</tbody>
</table>

Reproduced from reference 14 with permission from the American Society for Microbiology. *Sera obtained after antibiotic treatment; percent reactivity refers to the frequency that the different serological assays will be positive depending of the stage of the Lyme disease infection. EM Erythema migrans; Ig Immunoglobulin; WB Western blot

**TABLE 2**

Antibiotic therapy for children and youth with Lyme disease

<table>
<thead>
<tr>
<th>Drug, route</th>
<th>Dosage</th>
<th>Maximum per day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Doxycycline (≥8 years of age)</td>
<td>4 mg/kg per day, in two divided doses</td>
<td>200 mg</td>
</tr>
<tr>
<td>Amoxicillin (&lt;8 years OR unable to tolerate doxycycline)</td>
<td>50 mg/kg/day, in three divided doses</td>
<td>1.5 g</td>
</tr>
<tr>
<td>Cefuroxime</td>
<td>30 mg/kg per day, in two divided doses</td>
<td>1 g</td>
</tr>
<tr>
<td>Intravenous</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ceftriaxone</td>
<td>50–75 mg/kg once daily</td>
<td>2 g</td>
</tr>
<tr>
<td>Penicillin G</td>
<td>200,000–400,000 units/kg/4 h</td>
<td>18–24 million units</td>
</tr>
</tbody>
</table>

Data adapted from references 4 and 10. For patients allergic to penicillin, the alternative drug is cefuroxime. Macrolides (azithromycin, clarithromycin and erythromycin) have lower efficacy. Patients treated with macrolides should be closely observed to ensure resolution of clinical manifestations.[1][10][11]

**TABLE 3**

Route/duration of antibiotic therapy for Lyme disease

<table>
<thead>
<tr>
<th>Clinical stage</th>
<th>Route</th>
<th>Duration, days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Erythema migrans</td>
<td>Oral</td>
<td>14–21</td>
</tr>
<tr>
<td>Early disseminated and late disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Isolated facial palsy (Bell’s palsy)</td>
<td>Oral</td>
<td>14–21</td>
</tr>
<tr>
<td>Multiple erythema migrans</td>
<td>Oral</td>
<td>21</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Oral</td>
<td>28</td>
</tr>
<tr>
<td>Recurrent or persistent arthritis</td>
<td>Oral or IV</td>
<td>28</td>
</tr>
<tr>
<td>Heart block or carditis</td>
<td>IV</td>
<td>14–21</td>
</tr>
<tr>
<td>Meningitis</td>
<td>IV</td>
<td>14 (range 10–28)</td>
</tr>
<tr>
<td>Encephalitis/late neurological disease (encephalopathy, peripheral neuropathy)</td>
<td>IV</td>
<td>14–28</td>
</tr>
</tbody>
</table>

Data adapted from reference 4. IV intravenous

**How is LD treated?**

Treatment of LD should follow the clinical practice guidelines by the Infectious Diseases Society of America[1][10][11][16] and the American Academy of Pediatrics (Tables 2 and 3).[4]

Arthritis frequency has decreased in the United States, probably because of improved recognition and earlier treatment of patients with early LD. Up to one-third of LD patients with arthritis experience residual
synovitis and joint swelling, which almost always resolve without repeating the antibiotic course. For patients who have persistent or recurrent joint swelling after a recommended course of oral antibiotic therapy, some experts recommend retreatment with another four-week course of oral antibiotics or with a course of parenteral ceftriaxone.\[10\] For cases with ongoing arthritis, consultation with an expert is recommended.\[4\] Consider hospitalization and constant monitoring for a child with heart block and syncope that may rapidly worsen enough to require a pacemaker.\[10\]

The Jarisch-Herxheimer reaction (fever, headache, myalgia and an aggravated clinical picture lasting <24 h) can occur when therapy is initiated. Nonsteroidal anti-inflammatory agents should be started and the antimicrobial agent continued.\[4\]

Approximately 10% to 20% of cases experience lingering symptoms of fatigue and joint and muscle aching that last longer than six months. The clinical term for this condition is ‘post-treatment Lyme disease syndrome’ (PTLDS).\[17\] The exact cause of PTLDS is not yet known. Most medical experts believe that lingering symptoms are the result of residual damage to tissues and the immune system.\[17\][18] Recent evidence suggests that persistent infection with B burgdorferi occurs only rarely after appropriate treatment.\[19\] Long-course antibiotic treatments do not provide long-term improvement in PTLDS cases.\[10\][13][17]

A

B

Figure 6) How to remove a tick. Reproduced with permission from the United States Centers for Disease Control and Prevention (Atlanta, USA)

How to remove a tick

- Use fine-tipped tweezers to grasp the tick close to the skin surface (Figure 6A).

- Pull upward with steady, even pressure (Figure 6B). Try not to twist or jerk, which can cause the mouthpart of the tick to break off and remain in the skin. If this happens and you are unable to remove the mouthpart easily with clean tweezers, leave it alone and let the skin heal.

- Clean the bite area and your hands with rubbing alcohol, an iodine scrub, or soap and water.\[21\]

The Public Health Agency of Canada advises people to:

- Keep any ticks they remove themselves in a resealable plastic bag or pill vial and note the location and date of the bite.

- Watch for symptoms and see a health care professional immediately should symptoms appear.

- Take the tick with them to their medical appointment, to verify species and test as needed.\[1\]

How can LD be prevented?

Physicians should be aware of the epidemiology of tick-borne LD in their area,\[1\][2][7] and recommend some basic precautions for families living, hiking or camping in rural or wooded areas where they may be exposed to ticks.\[1][2][3]

- Where play spaces adjoin wooded areas, landscaping can reduce contact with ticks.\[3\] A pictogram from the Centers for Disease Control and Prevention is available at: http://www.cdc.gov/lyme/prev/in_the_yard.html.

- Apply 20% to 30% DEET or icaridin repellents. Repellents can be applied to clothing as well as to exposed skin. Always read and follow label directions.\[1][20\]

- Do a ‘full body’ check every day for ticks. Promptly remove ticks found on yourself, children and pets. Shower or bathe within two hours of being outdoors to wash off unattached ticks.\[1\]

For more information on how to prevent tick bites, refer to a recent practice point from the Canadian Paediatric Society at: http://www.cps.ca/en/documents/position/preventing-mosquito-and-tick-bites.

Postexposure antibiotic therapy

Consensus on postexposure prophylaxis for LD is lacking at this time. Some experts recommend giving doxycycline as a single dose of 200 mg for children and youth ≥8 years of age after a tick bite (for individuals weighing <45 kg, 4 mg/kg to a maximum of 200 mg). Prophylaxis can be started within 72 h of remov-
ing a tick, even if it has been attached for ≥36 h.\textsuperscript{[1][4][7] [10]} Data are insufficient to recommend amoxicillin pro-
phylaxis in younger children.\textsuperscript{[1][4][10][11]}

In Canada, such prophylaxis should be considered in
‘known endemic areas’ (see Table 1 and Figure 1 in
reference 1). Physicians should bear in mind that the
ture prevalence of B burgdorferi is often unknown and
that the geographical range of infected ticks is expand-
ing in some areas.\textsuperscript{[1]} The Public Health Agency of
Canada continues to monitor the distribution and
prevalence of infected ticks as well as cases of LD.\textsuperscript{[1][7]}

A vaccine to prevent LD in humans is not available at
the present time.\textsuperscript{[1][4]}

\textbf{Selected resource:}

healthycanadians.gc.ca/video/lyme-eng.php (Ac-
Address July 22, 2014).

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