Infections Acquired in the Garden

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ABSTRACT Gardening is a wonderful pastime, and the garden is a very peaceful place to enjoy one’s vacation. However, the garden may be a treacherous place for very young or compromised hosts when one takes into account the infectious potential residing in the soil, as well as the insect vectors on plants and animals. Even normal hosts may acquire a variety of infections from the soil, animals, or animal-related insect bites. The location of the garden, its natural animal and insect inhabitants, and the characteristics of the soil play a part in determining its infectious potential. The most important factor making the garden an infectious and dangerous place is the number and interaction of animals, whether they are pets or wild, that temporarily use the garden for part of their daily activities. The clinician should always ask about garden exposure, which will help in eliminating the diagnostic possibilities for the patient. The diagnostic approach is to use epidemiological principles in concert with clinical clues, which together should suggest a reasonable list of diagnostic possibilities. Organ involvement and specific laboratory tests help further narrow the differential diagnosis and determine the specific tests necessary to make a definitive diagnosis.

INTRODUCTION
Although there are many infections that one may acquire in the garden, some people have gardened for years without becoming infected, yet others may become ill after a rather limited time in the garden. Being in the garden presents a series of complex possibilities from an infectious disease standpoint, and the likelihood of acquiring an infectious disease while gardening depends upon many factors. Gardens are usually near the home and may be the closest that many people get to being in the great outdoors, especially in urban or suburban environments. The time spent in the garden is not nearly as important as the age and nature of the gardener, his friends, or his family.

Gardening may be a salutary experience for healthy individuals, but it is conceivably more dangerous to a patient with impaired immunity. For example, if a compromised host contracts coccidioidomycosis, histoplasmosis, or cat scratch disease, there is increased risk of dissemination. Elderly patients are fortunately relatively well off in terms of acquiring diseases in the garden. While elderly individuals can still acquire a variety of infections from the soil, animals, or animal-related insect bites, as a group they are not at increased risk for acquiring disorders solely on account of their age. Of course, if excavations are taking place or there is construction nearby, if local aquatic areas harbor Legionella species, and if the wind is right, then an elderly person would be more likely to acquire or have a more severe case of Legionnaires’ disease than his or her younger gardening counterparts.

Since many gardens are an extension of the home, children of various ages may frequent the garden or the land close to the house alone or with gardening adults. Many infectious diseases acquired in the garden are age specific; e.g., small children are more likely than adults to get Strongyloides stercoralis or hookworm infections in the appropriate locale. The garden not only is inhabited by plants and visited by humans but also may be a stopping point or refuge for birds and animals. Because
of its proximity to the house, household pets frequently wander freely throughout the backyard and the garden. Even if you do not have pets, it is not uncommon that pets from the neighborhood will spend various lengths of time and perform various bodily functions while passing through your garden. Therefore, if dogs, cats, or rodents are in the area, it is wise to consider that your garden and yard present the potential for contact with these animals or their excreta. Toxocara species organisms may be picked up by your dog or cat by ingestion and later transmitted to children via petting, for example. A stray neighborhood cat giving birth in or near your garden immediately sets the stage for the possibility of Q fever. The possibilities are almost endless. Birds may fly over, nest above, or be found sick or dead in the garden. Due to bird droppings in wood stacked for winter or in nests near the soil, the potential for histoplasmosis or blastomycosis exists.

Last, we come to the soil and plants themselves, which are, after all, the purpose of having a garden. What potential pathogens soil contains is largely a function of the animal life in the area as well as the location of the garden. For example, if the garden is located near moist, humid environments along riverbanks in the South, then blastomycosis becomes a diagnostic consideration. In contrast, if the garden is in the Southwest, then coccidioidomycosis and even plague, if an infected rodent is in the area, also become diagnostic possibilities. Rosebush thorn or sphagnum moss contact should immediately suggest the possibility of sporotrichosis. In the southeastern United States, where soil in moist areas may be contaminated with hookworm or Strongyloides larvae, these worms add to the potential diseases that can be acquired by contact of unprotected skin with the soil alone. The Ixodes species ticks that transmit Lyme disease, babesiosis, and ehrlichiosis in areas of endemicity may be found in the lawn adjacent to the garden, so Lyme disease, babesiosis, and/or ehrlichiosis may literally be acquired in your own backyard or garden. Therefore, the soil, by the nature of the organisms that normally reside in specific locations, e.g., spores of Coccidioides immitis or larvae of hookworm, presents infectious disease hazards that need to be reckoned with, as do the contributions made by various animals to the soil either by their presence or by contamination with their body fluids. One can easily appreciate the large array of infectious diseases that confront a person simply slipping out of the house and walking across the yard to do a little gardening.

### The Diagnostic Approach

In trying to analyze diagnostic possibilities for someone who has become ill and has spent time in the garden, it is necessary to consider the diagnosis from three different perspectives. First, one should consider the potential nature of contact, either passive or active, that the individual has had with sources of infection. If there has been extensive soil contact, then sporotrichosis is a diagnostic possibility. If piles of stacked or old moldy wood have been moved in association with gardening, then blastomycosis and histoplasmosis become additional possibilities. Nearby excavations with aerosolization of soil and water may suggest the possibility of Legionnaires’ disease. If the location of the patient is one where Lyme disease (Table 1), babesiosis, or ehrlichiosis is endemic, then these diagnoses should be considered with the appropriate clinical presentation. Similarly, as mentioned in the introduction, specific locations suggest specific soil organisms, e.g., hookworms, *C. immitis, Histoplasma capsulatum*, etc.

Additionally, potential animal contact needs to be considered from a variety of standpoints. The person’s own pets and their interaction with insect vectors and other animals in the area should be carefully ascertained and considered. In addition, one needs to consider the pets in the neighborhood as well as any wild animals interacting with the gardener or the gardener’s pet(s). One should inquire specifically about dead birds or other animals that the gardener may have found and buried in the garden. Specific inquiry should be made about the potential for contact with rodents or rabbits in the wild or runaway pets in the area. Only rarely is a disease actively transmitted from an animal to a human, and the situations are usually found to be straightforward if the proper question is asked. As mentioned

### Table 1: Clinical features of Lyme disease

<table>
<thead>
<tr>
<th>Stage</th>
<th>Dermatologic</th>
<th>Neurologic</th>
<th>Rheumatologic</th>
<th>Cardiac</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Erythema migrans</td>
<td>Possible headache, myalgias</td>
<td>Arthralgias</td>
<td>Carditis</td>
</tr>
<tr>
<td>2</td>
<td>Multiple and/or recurrent erythema migrans</td>
<td>Meningoencephalitis, peripheral neuritis</td>
<td>Arthralgias</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Acrodermatitis chronica atrophicans</td>
<td>Chronic arthritis</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
previously, some infectious diseases may be acquired actively or passively; for example, sporotrichosis may be acquired by simple handling of sphagnum moss or may be actively acquired as the result of a puncture wound from the thorn of a rose. The epidemiological associations with infectious diseases acquired from plants, soil, or animal vectors are presented in Table 2.

The next step in the diagnostic process is to determine the pattern of organ involvement by the infectious disease process to limit diagnostic possibilities and suggest specific disease entities. For example, if the patient presents with lymphadenopathy and a history of garden contact, then diagnostic possibilities are narrowed to toxoplasmosis, cat scratch disease, sporotrichosis, and occasionally Lyme disease. Cat contact increases the likelihood that these lesions are due to cat scratch disease or toxoplasmosis, whereas nodular lymphangitis suggests sporotrichosis. Obviously, there are many causes of adenopathy that have nothing to do with gardening or being in the garden, and the clinician must always be careful not to fail to consider the usual causes of lymph node involvement. However, if the adenopathy is likely associated with gardening, then diagnostic possibilities are greatly reduced. If there are other associated findings, this also helps to limit diagnostic possibilities. It is a good diagnostic principle for infectious diseases as well as in internal medicine to combine diagnostic findings, even if nonspecific, to increase diagnostic specificity. For example, if the patient with a history of gardening and axillary adenopathy also has a mild, nonexudative pharyngitis and a few atypical lymphocytes, then the likelihood of acquired toxoplasmosis is enhanced. Similarly, the likelihood of Lyme disease being present in a patient with headache is enhanced if the patient has a facial nerve palsy. The more variables that one can combine, the easier it is to arrive at a presumptive diagnosis. For example, if a patient presents with an ill-defined infiltrate on chest X-ray, abdominal pain, and a cough, accompanied by mental confusion and some diarrhea, then the chances of that individual having Legionnaires’ disease are high. These would not be the findings for other atypical pneumonias, i.e., Q fever, psittacosis, or Mycoplasma pneumoniae pneumonia.

The diagnosis of worms producing cough or pneumonitis during their pulmonary-migration phase may be a challenging diagnostic problem. Once again, by looking for associated features, one can increase diagnostic specificity and limit the differential diagnosis. For example, if after soil contact, the patient develops a nonspecific pulmonary infiltrate with eosinophilia, then strongyloidiasis becomes a likely explanation. Mental confusion, especially in a young child, with persistent eosinophilia, may suggest visceral larva migrans, especially if there has been a history of cat or dog contact.

### Table 2: Epidemiological considerations for infections from the garden

<table>
<thead>
<tr>
<th>Focus or vectors</th>
<th>Infectious disease or organisms&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Passively acquired</th>
<th>Actively acquired</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Soil and plants</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sporotrichosis</td>
<td>Sporotrichosis</td>
<td></td>
<td></td>
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<tr>
<td>Blastomycosis</td>
<td>Legionnaires’ disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Histoplasmosis</td>
<td>Nocardiosis</td>
<td></td>
<td></td>
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<tr>
<td>Strongyloidosis</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Hookworm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nocardiosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Animals</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cats</strong></td>
<td>Toxoplasmosis</td>
<td>Cat scratch disease</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Q fever</td>
<td>Pasteurella multocida</td>
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<tr>
<td></td>
<td>Tularemia</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>CLM (Ancylostoma spp.)</td>
<td>P. multocida</td>
<td></td>
</tr>
<tr>
<td></td>
<td>VLM (Toxocara cati)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Strongyloides</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Campylobacter spp.</td>
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<td></td>
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<tr>
<td></td>
<td>Giardiasis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yersinia pestis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Salmonella enterica</td>
<td></td>
<td></td>
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<tr>
<td><strong>Dogs</strong></td>
<td>Group A streptococci</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>VLM (Toxocara canis)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CLM (Ancylostoma spp.)</td>
<td></td>
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<tr>
<td></td>
<td>Leptospirosis</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Brucellosis</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Cryptosporidium spp.</td>
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<td></td>
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<tr>
<td></td>
<td>Dirofilaria immitis</td>
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<td></td>
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<tr>
<td></td>
<td>S. enterica</td>
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<td></td>
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<tr>
<td></td>
<td>Giardiasis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Campylobacter spp.</td>
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<td></td>
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<tr>
<td></td>
<td>RMSF (via tick bite)</td>
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<td></td>
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<tr>
<td></td>
<td>Listeria spp.</td>
<td></td>
<td></td>
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<tr>
<td><strong>Birds</strong></td>
<td>Blastomycosis</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Histoplasmosis</td>
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<tr>
<td></td>
<td>Cryptococcosis</td>
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<td></td>
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<tr>
<td></td>
<td>Q fever</td>
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<td></td>
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<tr>
<td><strong>Rabbits</strong></td>
<td>Tularemia</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Brucellosis</td>
<td></td>
<td></td>
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<tr>
<td><strong>Rats</strong></td>
<td>Leptospirosis</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Rat bite fever</td>
<td></td>
<td></td>
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<tr>
<td><strong>Other rodents</strong></td>
<td>Relapsing fever</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>(via tick bite)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Leptospirosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(via tick bite)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Leptospirosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>S. enterica</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>LCM (hamsters)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Ticks</strong></td>
<td>Babesiosis</td>
<td>Babesiosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lyme disease</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Ehrlichiosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(Anaplasma/Ehrlichia)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>Abbreviations: CLM, cutaneous larva migrans; VLM, visceral larva migrans; RMSF, Rocky Mountain spotted fever; LCM, lymphocytic choriomeningitis virus.
The differential diagnosis of infectious diseases by organ involvement is presented in Fig. 1. The clinician should remember that other diseases may produce similar end organ dysfunction and clinical manifestations, but gardening is an important epidemiological factor to consider in assessing the patient’s problem.

Laboratory tests represent the last approach in making the diagnosis. With all of the diseases potentially acquired by working in the garden, the clinician needs to establish a working diagnosis as described above and then arrive at a definitive diagnosis by ordering the appropriate specific tests. Aside from the specific laboratory tests needed to make a diagnosis, the clinician needs to have some clues that suggest the proper tests to be ordered for the individual patient. Therefore, nonspecific tests are most helpful when applied in the appropriate clinical context and combined with epidemiological and/or characteristic clinical manifestations. For example, anemia in a small child from a rural area of the southeastern United States should prompt a search for hookworm or Strongyloides. The liver is involved in many infectious disease processes, and therefore the finding of abnormal liver function tests is an important clue to a range of infectious diseases. With respect to the gardening population, an increased bilirubin count in a patient with pneumonitis may suggest Legionnaires’ disease, and conjunctival suffusion should suggest leptospirosis. Mild increases in alkaline phosphatase or serum transaminases may occur with dissimilar diseases, such as toxoplasmosis and Rocky Mountain spotted fever (Table 3). If the patient has an atypical pneumonia, i.e., an ill-defined infiltrate and mild to moderately abnormal liver function tests, then diagnostic possibilities are quickly narrowed to Legionnaires’ disease, psittacosis, and Q fever. Once again, it is important not to interpret diagnostic tests in a vacuum but rather to combine them with other findings in the history or physical diagnosis that quickly limits the diagnostic possibilities and provides the rationale for the working diagnosis (Fig. 2).

**SPECIFIC INFECTIOUS DISEASES**

**Sporotrichosis**

The classic fungus associated with the soil is *Sporothrix schenckii*. *S. schenckii* is a dimorphic fungus which on culture produces conidia arranged in a “daisy” cluster on top of a conidiophore. In tissue, the organism assumes an oval or cigar-shaped yeast form. The organism may be introduced into the skin via a minor abrasion, such as a thorn or splinter, resulting in the development of a suppurative lymphangitis of the skin and subcutaneous tissues, although rarely, hematogenous dissemination to the lungs, bones, and joints does occur. Alcoholics seem particularly prone to developing disseminated sporotrichosis, so this diagnostic point should be kept in mind when assessing patients who work in gardens and consume alcohol. The skin lesions of sporotrichosis usually begin as a small, gradually enlarging papular nodule which may become pustular and eventually ulcerates. Spread is distal to proximal along the lymphatics, and the lesions are characteristically not painful. While other diseases, such as tularemia, may resemble sporotrichosis, the indolent course of the illness along the lymphatics, with bridges of normal skin between painless lesions, is highly suggestive of sporo-

**FIGURE 1** Blood smear showing *Babesia* spp. rings with basophilic stippling within the erythrocytes. *Babesia* organisms resemble *Plasmodium falciparum*, but *Babesia* parasites present several distinguishing features. They vary more in shape and in size, and they do not produce pigment. (Source: CDC/Dr. Mae Melvin, CDC-PHIL ID# 2223) doi:10.1128/microbiolspec.IOL5-0020-2015.f1
trichosis. Diagnosis of sporotrichosis is made by culturing the mycelial form of the organism from the affected tissue; however, repeat cultures may need to be performed. Direct examination of tissue for the presence of the yeast form may be helpful, but the organisms are generally rare. Serologic testing is generally not useful in the diagnosis of sporotrichosis.

**Babesiosis**

Babesiosis is a zoonotic, intraerythrocytic, systemic infection that is transmitted in endemic areas by several tick vectors. While babesiosis is present in North America, Europe, and Asia, the areas in North America that are endemic (northeastern United States, Minnesota, Wisconsin, and some areas in California) mirror the distribution of the *Ixodes dammini* (*Ixodes scapularis*) tick vector. The species that cause human babesiosis vary depending on the location (e.g., U.S. infections principally caused by *Babesia microti*, infection in Europe caused by *Babesia divergens/Babesia bovis*). The parasites are transmitted from their animal reservoir (the white-footed mouse and white-tailed deer) to humans via nymphs and adults, respectively, of the *I. dammini* tick. Often the patient will be unaware of the tick bite and may not report this to the clinician. Transmission may also occur through administration of contaminated blood products.

Clinically, babesiosis typically presents as a malaria-like illness characterized by fever with relative bradycardia, chills, fatigue, headache, and myalgias. Rash is absent, and there are few if any localizing signs. Splenomegaly may be the only finding. Most patients infected with *B. microti* are either asymptomatic or have a relatively mild course of illness, but certain populations are at greater risk of severe, even life-threatening infection, e.g., asplenic individuals, immunosuppressed

**TABLE 3** Differential diagnosis of Rocky Mountain spotted fever

<table>
<thead>
<tr>
<th>Signs and/or symptoms</th>
<th>Rocky Mountain spotted fever</th>
<th>Meningococcal meningitis</th>
<th>Dengue fever</th>
<th>Leptospirosis</th>
<th>Atypical measles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mental confusion</td>
<td>±</td>
<td>±</td>
<td>±</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td>Headache</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>±</td>
</tr>
<tr>
<td>Photophobia</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Myalgia, arthralgia</td>
<td>+++</td>
<td>++</td>
<td>+++</td>
<td>+++</td>
<td>±</td>
</tr>
<tr>
<td>Nausea, vomiting</td>
<td>±</td>
<td>−</td>
<td>−</td>
<td>±</td>
<td>−</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>±</td>
<td>−</td>
<td>−</td>
<td>±</td>
<td>−</td>
</tr>
<tr>
<td>Rash</td>
<td>Maculopapular, petechial (ankles/wrists)</td>
<td>“Palpable” petechiae (diffuse)</td>
<td>Petechial (truncal)</td>
<td>Maculopapular (truncal)</td>
<td>Urticarial, maculopapular (truncal)</td>
</tr>
<tr>
<td>Jaundice</td>
<td>±</td>
<td>−</td>
<td>−</td>
<td>±</td>
<td>−</td>
</tr>
<tr>
<td>Splenomegaly</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>±</td>
<td>−</td>
</tr>
<tr>
<td>Periorbital edema</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>−</td>
</tr>
<tr>
<td>Conjunctival suffusion</td>
<td>±</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>−</td>
</tr>
<tr>
<td>Abnormal LFTs</td>
<td>±</td>
<td>−</td>
<td>−</td>
<td>+++</td>
<td>−</td>
</tr>
<tr>
<td>Eosinophilia</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+++</td>
</tr>
<tr>
<td>Infiltrates on chest film</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
</tr>
</tbody>
</table>

*Abbreviations: LFTs, liver function tests; +, present; −, absent; ++, frequent manifestation; ++++, very frequent manifestation; ±, present or absent.

*The size of the spleen increases by 50%.

**FIGURE 2** Lateral view of a female blacklegged, or deer tick, *Ixodes scapularis*, with its abdomen engorged with a host blood meal. *I. scapularis* transmits Lyme disease, a disease caused by a spiral-shaped bacterial microbe, *Borrelia burgdorferi*. This disease is known in Europe, Africa, Asia, and in almost all the United States. It is especially common in the Northeast, in Minnesota, and in northern California. This larval tick is no bigger than the size of the period at the end of this sentence. (Source: CDC/ Dr. Gary Alpert, Urban Pests-Integrated Pest Management [IPM], CDC-PHIL ID# 15993) [doi:10.1128/microbiolspec.IOL5-0020-2015.f2](https://doi.org/10.1128/microbiolspec.IOL5-0020-2015.f2)
patients, and elderly patients. While the degree of parasitemia is often <5% in normal hosts, asplenic and immunocompromised individuals may have higher degrees of parasitemia (>5 to 10%). Nonspecific laboratory abnormalities may include normal white blood cell count or slight leukopenia, relative lymphopenia, atypical lymphocytes, anemia, and thrombocytopenia. Elevated erythrocyte sedimentation rate (ESR), lactate dehydrogenase (LDH), total bilirubin, and serum ferritin levels may be diagnostically helpful. A definitive diagnosis of babesiosis is made by identifying the intraerythrocytic parasites on peripheral blood smears, sometimes forming the characteristic “Maltese cross” inclusions. Alternatively, positive PCR or elevation of IgM titer is also diagnostic.

The preferred treatment of Babesia is atovaquone in combination with azithromycin. In cases of severe babesiosis or in those who cannot tolerate this regimen, quinine and clindamycin may be used. Typical therapy duration is 7 days, but this may be extended in patients who fail to clear their infection or are asplenic. Exchange transfusion may be lifesaving in severe cases of babesiosis.

Ehrlichiosis (Ehrlichia/Anaplasma)

Human ehrlichiosis is caused by two organisms. Anaplasmosis, also called human granulocytic anaplasmosis (HGA) (formerly human granulocytic ehrlichiosis), is caused by the intracytoplasmic, small Gram-negative organism Anaplasma phagocytophilum that infects the granulocyte white blood cells. Anaplasmosis is transmitted by the I. scapularis tick, primarily found in the northeastern United States as well as areas of Wisconsin, Minnesota, and sections of California. The distribution is identical to that of Babesia and Lyme disease because they share the same tick vector. Coinfection may occur but is rare. Human monocytic ehrlichiosis (HGE), caused by Ehrlichia chaffeensis or sometimes Ehrlichia ewingii (primarily in immunocompromised patients), infects the monocyte white blood cells and is spread by the A. americanum tick (Lone star tick) found in the southeastern and central United States.

The clinical presentation of HGE and anaplasma are clinically indistinguishable, and the epidemiology and laboratory testing are what differentiates the two. Ehrlichia/Anaplasma typically presents with the acute or subacute onset of fever, headache, malaise, and myalgias. The presentation may resemble that of Rocky Mountain spotted fever, but unlike that disease, rash is rarely present. Splenomegaly, with occasional hepatomegaly, may be present. Laboratory studies usually demonstrate leukopenia, relative lymphopenia, and thrombocytopenia. Unlike babesiosis, anemia is not typically present, and ESR is minimally elevated if at all. Liver function tests and serum ferritin levels may also be elevated.

Although not always present, and more common in anaplasmosis than in HGE, visualization of the characteristic mulberry-shaped morulae in the cytoplasm of the infected white blood cell is diagnostic. Alternatively, positive PCR or elevated indirect fluorescent antibody titers between acute and convalescent serum against human granulocytic ehrlichiosis (HGA) or human monocytic ehrlichiosis (HME) may be used to confirm the diagnosis.

Treatment for both HGA and HME consists of a 1–2 week course of doxycycline. In cases where doxycycline cannot be used, a quinolone, rifampin, or chloramphenicol may be substituted.

Blastomycosis

Blastomyces dermatitidis is the dimorphic fungus responsible for the development of blastomycosis. This organism has proven difficult to isolate from environmental sources; however, exposure to organically rich, warm, moist soil appears to be a risk factor for the development of infection. Blastomycosis is endemic in the southeastern and midwestern United States and has been classically associated with the Ohio and Mississippi River Valley regions. The fungus enters via the lungs and can result in asymptomatic disease, acute infection that mimics a bacterial pneumonia, or chronic pulmonary infection which may be confused clinically with tuberculosis. B. dermatitidis often disseminates hematogenously, with the skin being the most frequent site of extrapulmonary infection. The skin lesions are characteristically verrucous or ulcerative in nature.

Osteomyelitis due to B. dermatitidis occurs as well. Genitourinary tract involvement manifests as prostatitis and/or epididymo-orchitis in males, but involvement of the female genitourinary tract is rare. Central nervous system infection resulting in meningitis or a brain abscess is seen most commonly in immunocompromised individuals, especially in people with AIDS.

The diagnosis of blastomycosis is confirmed by isolating the organism in culture or from a biopsy specimen, where the fungus appears in its yeast phase. The organism may also be observed on potassium hydroxide (KOH) preparations of clinical specimens such as sputum, pus, or prostatic secretions. Serologic testing remains unreliable and should be used only in conjunction with isolation of the organism.
**Legionnaires’ Disease**

Legionnaires’ disease can be acquired in the garden only if the organism is in soil that is being excavated nearby and there is airborne spread of the organism in the garden area. Legionnaires’ disease is varied in its distribution; some areas have a relatively high incidence, while the disease is unheard of in other locations. Legionnaires’ disease is most common in the late spring and early fall, especially during periods of increased precipitation. The course may be subacute or fulminant, and it typically presents as pneumonia. Legionnaires’ disease should be considered in the diagnosis of all community-acquired pneumonias, and characteristic diagnostic features should be looked for to arrive at a working diagnosis. The clue to all of the atypical pneumonias lies in their extrapulmonary manifestations, since they are all systemic infections. With Legionnaires’ disease, extrapulmonary manifestations commonly include changes in mental status, nonspecific abdominal pain, or diarrhea. In contrast to *Mycoplasma pneumonia*, Legionnaires’ disease is not associated with otitis or pharyngitis. If the patient has a temperature in excess of 102°F, does not have an arrhythmia or a pacemaker, and is not on beta-blockers, diltiazem, or verapamil, then a pulse-temperature deficit provides the single most important clue to the diagnosis. Relative bradycardia is present in virtually all patients with Legionnaires’ disease presenting with a temperature over 102°F, and if the pulse is charted with a temperature, a pulse-temperature deficit is readily seen by simple inspection. However, if one wants to calculate if there is relative bradycardia present, then one takes the temperature in degrees Fahrenheit, takes the last digit, decreases it by 1, multiplies that number by 10, and adds that number to 100. For example, if the temperature is 105°F, the 5 is reduced to 4 and multiplied by 10 to get 40, and this is added to 100 to get 140. Therefore, a pulse of <130 in a patient with a 105°F temperature indicates a pulse-temperature deficit even if the patient is “tachying along” at 120 beats/min.

Chest X-rays do not have specific findings, but they usually “behave” in a typical way. Legionnaires’ disease on chest X-ray is characterized by a rapidly progressive asymmetrical infiltrate(s). While not all *Legionella* infiltrates behave in this fashion, this is nevertheless the most typical radiographic manifestation. In terms of laboratory tests, a decreased serum phosphorus level, when present, is a helpful finding. A decreased serum sodium level appears to be more commonly associated with Legionnaires’ disease than with other pneumonias, but it is not specific for *Legionella* infections. A decrease in sodium on the basis of the syndrome of inappropriate secretion of antidiuretic hormone may occur with any pulmonary process, whether it is infectious, inflammatory, or neoplastic. In contrast, a depressed serum phosphorus level is uniquely associated with Legionnaires’ disease. An elevated bilirubin with an atypical pneumonia limits diagnostic possibilities to pneumococcal pneumonia and Legionnaires’ disease. The serum transaminases are almost always mildly elevated in patients with Legionnaires’ disease. This is another important laboratory clue to the presence of an atypical pneumonia, since only Legionnaires’ disease, Q fever, and psittacosis are frequently associated with abnormal liver function tests, in contrast to *Mycoplasma pneumonia*. Therefore, a working diagnosis can readily be obtained by combining the aforementioned features, while a definitive diagnosis depends upon identifying the organism with direct fluorescent-antibody assay of sputum or pleural fluid, urinary antigen testing, or indirect fluorescent-antibody assay serologic methods. The organism may also be cultured directly from sputum or appropriate samples of lung or pleural fluid. The differential diagnostic features of Legionnaires’ disease are presented in Table 4.

**Hookworm**

Hookworm disease is caused by two intestinal nematodes, *Ancylostoma duodenale* and *Necator americanus*. The environmental conditions of the southeastern United States, with its warmth, high humidity, and heavy rainfall, are ideal for the life cycles of these nematodes. In contaminated soil, the eggs hatch in approximately 24 h and become rhabditiform larvae. The rhabditiform larvae incubate in the warm, moist soil for 5 to 10 days, developing into the infectious filariform larvae. This form can survive for up to 1 month in the soil. Human infection occurs when the filariform larvae penetrate exposed skin, usually through bare feet. When *N. americanus* is involved, there is often a local skin reaction consisting of erythema and a pruritic papular or vesicular eruption near the entry site. This is less commonly seen with *A. duodenale*. These larvae enter the venous circulation, where they are carried to the lungs. Pulmonary complaints such as cough, wheezing, or pulmonary infiltrates can be seen at this time. The filariform larvae then migrate into the pharynx, where they are swallowed. They mature into adult worms in the small intestine, where they attach and feed on the blood of the host and liberate more eggs. Symptoms at this time generally consist of nonspecific abdominal complaints such as pain, bloating, nausea, or symptoms...
attributable to anemia. Laboratory findings may consist of a hypochromic, microcytic anemia, the degree of which is a function of the worm burden and consequent blood loss, eosinophilia, and hypoproteinuria. Diagnosis of hookworm disease is made by finding the characteristic oval eggs in a direct stool smear. Stool studies may be negative during early infections, and infections with light worm burdens require stool concentration techniques. Fresh stools should be examined immediately since eggs may hatch into rhabditiform larvae resembling the rhabditiform larvae of *Strongyloides*.

### S. stercoralis

Strongyloidiasis, or threadworm infection, results from infection with the nematode *S. stercoralis*. It is less commonly encountered than hookworm infection, and the threadworm is unique among the nematodes in its ability to cause autoinfection due to its peculiar triphasic life cycle. The rhabditiform larvae of *Strongyloides* penetrate the skin in a manner analogous to that of the hookworm. A pruritic maculopapular eruption or larva currens, which is a migrating serpiginous linear rash, may be seen at that time. The rhabditiform larvae are carried by the venous circulation to the lungs. In a healthy host, this pulmonary migration is usually asymptomatic, but in an immunocompromised host, cough, wheezing, dyspnea, and fleeting pulmonary infiltrates accompanied by peripheral eosinophilia may provide a clue to the diagnosis. The rhabditiform larvae ascend and are swallowed to complete their life cycle within the small intestine of the host, where the presence of the nematode may cause abdominal pain, diarrhea, and weight loss. Autoinfection occurs when, while still in the intestine, the rhabditiform larvae develop into infectious rhabditiform larvae.

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**TABLE 4 Diagnostic features of atypical pneumonias**

<table>
<thead>
<tr>
<th>Manifestation</th>
<th>Mycoplasma pneumonia</th>
<th>Legionnaires’ disease</th>
<th>Psittacosis</th>
<th>Q fever</th>
<th>Tularemia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mental confusion</td>
<td>–</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Headache</td>
<td>±</td>
<td>+</td>
<td>±</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td>Meningismus</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<tr>
<td>Myalgia</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<tr>
<td>Ear pain</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Pleuritic pain</td>
<td>–</td>
<td>–</td>
<td>±</td>
<td>±</td>
<td>+</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>–</td>
<td>–</td>
<td>±</td>
<td>±</td>
<td>±</td>
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<tr>
<td>Diarrhea</td>
<td>±</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<tr>
<td>Hoarseness</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<tr>
<td><strong>Signs</strong></td>
<td></td>
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</tr>
<tr>
<td>Rash</td>
<td>± (erythema multiforme)</td>
<td>–</td>
<td>+ (Horder’s spots)</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Raynaud’s phenomenon</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Nonexudative pharyngitis</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<tr>
<td>Hemoptysis</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<tr>
<td>Lobar consolidation</td>
<td>–</td>
<td>–</td>
<td>±</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td>Cardiac involvement</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Splenomegaly</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td>Relative bradycardia</td>
<td>–</td>
<td>–</td>
<td>±</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td><strong>Chest film findings</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Infiltrates</td>
<td>Patchy</td>
<td>Patchy/consolidation</td>
<td>Patchy/consolidation</td>
<td>Ovoid infiltrates</td>
<td>Patchy/consolidation</td>
</tr>
<tr>
<td>Bilateral hilar adenopathy</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>Pleural effusion</td>
<td>± (small)</td>
<td>±</td>
<td>±</td>
<td>–</td>
<td>+ (bloody)</td>
</tr>
<tr>
<td><strong>Laboratory findings</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leukocytosis</td>
<td>±</td>
<td>±</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Hypophosphatemia</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Elevated AST/ALT</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td>Cold agglutinins</td>
<td>+</td>
<td>–</td>
<td>±</td>
<td>±</td>
<td>–</td>
</tr>
<tr>
<td>Microscopic hematuria</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>±</td>
<td>±</td>
</tr>
</tbody>
</table>


*Abbreviations: AST/ALT, aspartate aminotransferase/alanine aminotransferase; +, present; –, absent; ±, variously present or absent.*
larvae, which in turn penetrate the colonic mucosa or perirectal skin, reinfecting the host. In immunocompromised individuals, a hyperinfection syndrome may be seen, which occurs when there is widespread dissemination of the filariform larvae via the bloodstream. Secondary bacterial infections are common in this condition due to large numbers of filariform larvae migrating from the intestine. Mortality associated with hyperinfection syndrome is quite high.

The diagnosis of strongyloidiasis is made by demonstrating the rhabditiform larvae in concentrated stool specimens or duodenal fluid. Stool concentration techniques may increase yield. Peripheral eosinophilia is generally present in immunocompetent (but not immunocompromised) individuals, and total immunoglobulin E may be elevated. The filariform larvae may be present in the stool, urine, sputum, or bronchoalveolar lavage fluid of immunocompromised people.

**Nocardiosis**

*Nocardia* species are soil-borne aerobic actinomycetes that can cause localized or disseminated infection in humans. *Nocardia asteroides* is most frequently implicated in human illness, followed by *Nocardia brasiliensis* or *Nocardia otitidiscaviarum*, although infections with other species have been reported. These organisms are recovered throughout the world from warm, moist soil as well as from other environmental sources. Clinical infection with *Nocardia* occurs most commonly in immunocompromised individuals, such as transplant recipients or people with collagen vascular disease, lymphoreticular malignancies, or chronic steroid use. People with pulmonary alveolar proteinosis or HIV are also at risk for nocardiosis. The organism enters the body via inhalation into the lungs, although the portal is occasionally the gastrointestinal tract or the skin through traumatic implantation.

Pulmonary infection is characterized by the formation of multiple suppurative abscesses. The clinical symptoms of pulmonary nocardiosis are similar to those of tuberculosis, with fever, malaise, cough, weight loss, and night sweats. Sinus tract formation from the lungs can occur. Radiographically, the infiltrates of patients with nocardiosis may present as consolidated, alveolar, or reticular infiltrates. Cavitation and pleural involvement are common, and empyema occurs in about one-quarter of patients. Pulmonary *Nocardia* infection can have a protracted course, but it may also remit spontaneously or have an acute self-limited course. Hematogenous spread of *Nocardia* from the lungs to distant sites can occur. Concurrent pulmonary symptoms may be absent at the time of presentation. The central nervous system is a common site of dissemination. The clinical picture is generally that of a brain abscess or tumor, with fever, headache, nausea, vomiting, and focal neurological deficits. There is frequent dissemination of the pathogen to the eyes, kidneys, heart, bones, and subcutaneous tissues. Dissemination can occur in the absence of pulmonary involvement. Cutaneous or subcutaneous manifestations are seen after traumatic inoculation through the skin. When subcutaneous abscesses form, they are generally discrete, firm, nonindurated nodules, which, in contrast to those of actinomycosis, do not form draining sinuses. One exception to this is when *Nocardia* species are the causative agents in maduromycosis; in these cases, draining of sinus tracts occurs.

The diagnosis of nocardiosis is made by isolation of the organism from a clinical specimen. Gram staining should be performed on pus or sputum. When *Nocardia* organisms are present, they appear as weakly Gram-positive, branching, filamentous rods, often looking “beaded.” Many species of *Nocardia* are acid fast. *Nocardia* species grow well on standard laboratory media; however, growth may take longer than 48 h when the organisms are present in mixed culture. They grow poorly on routinely used fungal media. No useful serologic tests are available at present.

**CONCLUSIONS**

Gardening is a wonderful pastime, and the garden is a very peaceful place to enjoy one’s vacation. However, the garden may be a treacherous place for very young or immunocompromised hosts when one takes into account the infectious potential residing in the soil, as well as the insect vectors on plants and animals. The location of the garden and the characteristics of the soil play a part in determining its infectious potential. The most important factor making the garden an infectious and dangerous place is the number and interaction of animals, whether they are pets or wild, that temporarily use the garden for part of their daily activities. The clinician should always ask about garden exposure, which helps in eliminating the diagnostic possibilities for the patient. The diagnostic approach is to utilize epidemiologic principles in concert with clinical clues, which together should suggest a reasonable list of diagnostic possibilities. Organ involvement and specific laboratory tests help further narrow the differential diagnosis and determine the specific tests necessary to make a definitive diagnosis.
PRACTICAL TIPS

- Alcoholics are particularly prone to developing disseminated sporotrichosis. Sporotrichosis should be considered in people who regularly consume alcohol and work in a garden.
- Suspect babesiosis in patients from areas endemic to the *I. scapularis* tick presenting with a malaria-like illness with fever, chills, headache, fatigue, and malaise with anemia or an elevated lactate dehydrogenase or total bilirubin.
- Ehrlichia/anaplasma infections may mimic the clinical presentation of Rocky Mountain spotted fever but usually do not have a rash.
- Patients whose gardens are near water or outdoor construction sites and who develop rapidly progressive asymmetrical infiltrates on chest X-rays should be suspected of having Legionnaires' disease.
- Strongyloidiasis with peripheral eosinophilia occurs in immunocompetent patients, but the eosinophilia may be absent in immunocompromised hosts.

RECOMMENDED READINGS


