Leishmaniasis is a major zoonotic disease caused by the intracellular protozoa *Leishmania*, transmitted primarily by the bite of infected female phlebotomine sand flies.\(^1\)\(^2\) It is widespread in tropical and subtropical areas and found in 99 countries and territories in the Mediterranean basin, Africa, central and southwestern Asia, and Central and South America, where sand flies are endemic.\(^1\)\(^3\) In humans, leishmaniasis is one of the world’s most significant emerging infectious diseases.\(^4\)

Leishmaniasis has not been considered a significant concern to veterinarians in the United States because the sand flies capable of transmitting the parasite have been documented in only a few isolated parts of the country and prevalence of leishmaniasis in dogs native to the United States has been relatively low. However, the number of cases of canine leishmaniasis has increased, from both importation of dogs from *Leishmania*-endemic areas and local acquisition (autochthonous cases); autochthonous cases have been reported in California,\(^5\) Georgia, Maryland, New York, North Dakota, Oklahoma, and Texas.\(^6\) Contributions to the rise of canine leishmaniasis in the United States include vertical transmission, international travel and animal transportation, and climate change.\(^7\) Like people, dogs and cats (along with other animals) can be infected by a variety of species of *Leishmania*; the main reservoir hosts, however, are dogs, susceptible to

**Abstract**

Leishmaniasis is a zoonotic disease that affects humans, dogs, and other mammals. Although endemic to certain areas of the world, it is not endemic to the United States, where prevalence among companion animals has been historically low. However, case numbers have been increasing in North America. Therefore, it is imperative that U.S. veterinarians be able to identify and treat leishmaniasis in their patients. This article reviews canine leishmaniasis and provides a case study of a dog with infection that was imported from China. The case study highlights the complexity of this zoonotic infection and the wide range of its clinical manifestations, from asymptomatic infection to potentially fatal disease. This review should raise awareness and provide guidance for U.S. veterinarians on how to recognize, diagnose, treat, and manage imported and autochthonous leishmaniasis in dogs.
Take-Home Points

- Dogs are the main reservoir hosts of *Leishmania* and can experience asymptomatic infection or leishmaniasis, which is the active form of the disease.
- *Leishmania* infection in dogs is more prevalent than leishmaniasis because infection often does not equate with clinical disease.
- Leishmaniasis is often difficult to diagnose due to the wide variety of clinical manifestations, ranging from mild and nonspecific to severe and potentially fatal disease with multiorgan involvement that can often be confused with other, more common, diagnoses.
- Leishmaniasis is rarely considered a differential diagnosis by U.S. veterinarians because of its historically low prevalence in the United States; however, *Leishmania* case numbers are increasing due to a variety of factors, including international travel, importation of pets from *Leishmania*-endemic countries, autochthonous cases, and climate change affecting the migration patterns of the phlebotomine sand fly vector.
- A correct diagnosis is crucial as leishmaniasis can be fatal if left untreated. Therefore, a patient’s workup should include the pet’s travel history.

Different forms of disease, ranging from self-limiting cutaneous lesions to severe visceral disease that can be fatal if untreated. As incidence of leishmaniasis diagnoses in dogs increases in the United States, veterinarians should be familiar with the clinical signs and consider it as a potential diagnosis for dogs in North America, especially ill dogs that have been to or bred to any dog from a *Leishmania*-endemic region.

This article describes a case of canine leishmaniasis in a 5-year-old spayed female Australian shepherd recently imported to the United States from a shelter in the Henan province of China.

**CASE REPORT**

History and Clinical Findings

**Initial Examination in China**

On initial examination in China, the patient appeared healthy, except for being underweight and having moderate to severe dental tartar. Basic veterinary care was provided, including core vaccinations and occasional doses of flea protection. She progressively lost weight, and lameness developed and worsened over the 9 months preceding her transport to the United States.

Routine laboratory tests (TABLE 1), including several complete blood counts (CBCs), most notably indicated initial hemoconcentration, which progressed over 9 months to mild nonregenerative anemia. Initial biochemistry results indicated mild hypoalbuminemia and hyperglobulinemia, which progressed to significant hypoalbuminemia and hyperglobulinemia, and creatinine and blood urea nitrogen (BUN) progressed from normal ranges to elevated values. No urinalysis was performed. A fecal flotation result was negative.

One month before transport, progressively worsening lameness was noted. Radiographs showed marked soft tissue swelling around the right elbow with normal appearing carpi and long bones. No cause was identified. Immediately before transport, non–weight-bearing

| **TABLE 1** Pretreatment Laboratory Results, China |
|------------------------------|------------------|------------------|------------------|
| ANALYTE                      | REFERENCE RANGES | BASELINE RESULTS | RESULTS 9 MONTHS LATER |
| Red blood cells, M/μL        | 5.65–8.87        | 11.32            | 4.77              |
| Hematocrit, %                | 37–61            | 69.7             | 32.1              |
| Hemoglobin, g/dL             | 13–21            | 24.3             | 10.8              |
| Globulin, g/dL               | 2.5–4.5          | 4.5              | 6.1               |
| Albumin, g/dL                | 2.3–4            | 2.6              | 1.9               |
| Creatinine, mg/dL            | 0.5–1.5          | 1.4              | 2.3               |
| Blood urea nitrogen, mg/dL   | 7–27             | 25               | 31                |
lameness developed on the left forelimb. Radiographs showed increased soft tissue swelling of the right elbow and mild right carpal soft tissue swelling (left carpus not seen). A chondroitin supplement was started. Because of the dog’s clinical decline, she was taken to a veterinarian immediately after arrival in the United States.

**Initial Examination in the United States**

During initial examination (FIGURE 1), the patient was quiet, alert, and slightly dehydrated. Her mucous membranes were pale pink. She had a temperature of 39.9 °C (103.8 °F). She had mild alopecia and scaling around both eyes and on the bridge of the nasal planum, small crusts on the tip of the nares, and bilateral blepharitis. The anterior carpi and caudal tarsi had mild, diffuse alopecia and large white scales. There was large flaky white crusting on all paw pads and diffuse sarcopenia (FIGURE 2). The patient was unwilling to rise without assistance, but once standing, she was able to walk with lameness noted in multiple limbs. Swelling and moderate palpable effusions were noted on the right elbow, left carpus, and both tarsi. Submandibular, prescapular, and popliteal lymph nodes were mildly enlarged and firm. Fundic and neurologic examinations were unremarkable.

Doppler blood pressure was normal (140 mm Hg). Clinical pathology revealed further elevated creatinine and BUN; elevated phosphorous; worsening hypoalbuminemia, hyperglobulinemia, and nonregenerative anemia (normochromic and normocytic); and no reticulocyte response (TABLE 2). Urinalysis revealed no active sediment or bacteria but indicated significant proteinuria, elevated specific gravity, and elevated urine protein:creatinine ratio. Testing results for heartworm infection, Lyme disease, *Ehrlichia* species, and *Anaplasma* species were negative.

**Differential Diagnoses and Further Diagnostics**

Based on the problem list (BOX 1), differential diagnoses were:

- chronic systemic immune-mediated disease
  - immune-mediated versus infectious polyarthritis
  - fungal or infectious vector-borne diseases, especially those endemic to the patient’s region or origin in China
  - neoplasia (e.g., multiple myeloma, lymphoma)

Further testing revealed the following:

- Serum protein electrophoresis demonstrated polyclonal gammopathy, ruling out multiple myeloma and supporting a diagnosis of a chronic inflammatory process.
- Skin biopsy samples from the nose and periocular skin were stained routinely with Grocott methenamine silver for fungal disease. Findings indicated dermatitis (lymphoplasmacytic, histiocytic, and neutrophilic), and lesions were multifocal to coalescing, moderate, and chronic-active with acanthosis. No organisms were seen.
- Cytology and aerobic culture of joint fluid aspirated from both carpi revealed sterile suppurative inflammation of unknown origin. No organisms were seen, and cultures were negative.

**BOX 1 Problem List**

- Azotemia
- Effusion of multiple joints
- Fever
- Hyperglobulinemia
- Hypoalbuminemia
- Lymphadenomegaly
- Marked lameness
- Multifocal dermatopathy
- Nonregenerative anemia
- Proteinuria/protein-losing nephropathy
- Sarcopenia
- Weight loss

**FIGURE 1.** Patient at initial examination in the United States.
FIGURE 2. Patient’s clinical signs. (A) Mild alopecia, scaling noted around both eyes, and blepharitis; (B) mild, diffuse alopecia and large white scales on bridge of nasal planum; (C) right caudal tarsus; (D) large flaky white crusting on paw pad; (E) and anterior carpi.
Lymph node aspirates showed reactive lymphoid hyperplasia of unknown origin; therefore, lymphoma was considered to be very unlikely. No organisms were seen.

Antinuclear antibody test result was negative, making immune-mediated disease slightly less likely.

Polymerase chain reactions (PCRs) for vector-borne infectious disease tests were negative for *Mycoplasma haemocanis; Candidatus Mycoplasma haematoparvum; Babesia, Anaplasma, Ehrlichia, Rickettsia, Hepatozoon*, and *Leishmania* species; *Neorickettsia risticii*; and canine *Bartonella*. *Leishmania* antibody immunofluorescence antibody titer was positive (1:1600).

**Definitive Diagnosis**
A diagnosis of leishmaniasis was based on history, clinical signs, and clinicopathologic abnormalities consistent with leishmaniasis, as well as the high titer and, ultimately, response to treatment. Although not necessary for a definitive diagnosis, and not done in this case study, identifying the organism in tissue (FIGURE 3) supports the diagnosis.

**Treatment**
Considering the severity of the joint effusions and inflammatory component, treatment was started immediately pending diagnostic results. Treatment included gabapentin (20 mg/kg PO q8h) for discomfort, doxycycline (10 mg/kg PO q24h) to cover possible tick-borne infection and for its anti-inflammatory properties, and intra-articular injections of triamcinolone (3 mg) into both tarsi. Because of the marked proteinuria, the patient was given oral clopidogrel (1.5 mg/kg PO q24h) as an antithrombotic and oral telmisartan (1 mg/kg PO q24h) to lessen renal protein losses.

In response to the joint injections and supportive care, the patient’s lameness, fever, and azotemia resolved within 48 hours. After receiving the positive immunofluorescence antibody titer confirming a diagnosis of leishmaniasis, allopurinol (10 mg/kg PO q12h indefinitely) and meglumine antimoniate (100 mg/kg SC, divided into 2 equal doses q12h for 6 weeks) were prescribed.9

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**TABLE 2 Pretreatment Laboratory Results, United States**

<table>
<thead>
<tr>
<th>ANALYTE</th>
<th>REFERENCE RANGES</th>
<th>RESULTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BLOOD</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Red blood cells, M/μL</td>
<td>5.65–8.87</td>
<td>3.74</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>37–61</td>
<td>22.9</td>
</tr>
<tr>
<td>Hemoglobin, g/dL</td>
<td>13–21</td>
<td>7.9</td>
</tr>
<tr>
<td>Globulin, g/dL</td>
<td>2.5–4.5</td>
<td>5.5</td>
</tr>
<tr>
<td>Albumin, g/dL</td>
<td>2.3–4</td>
<td>1.9</td>
</tr>
<tr>
<td>Creatinine, mg/dL</td>
<td>0.5–1.5</td>
<td>2.8</td>
</tr>
<tr>
<td>Blood urea nitrogen, mg/dL</td>
<td>7–27</td>
<td>48</td>
</tr>
<tr>
<td>Phosphorous, mg/dL</td>
<td>2.5–6.8</td>
<td>7.9</td>
</tr>
<tr>
<td>Reticulocytes, K/μL</td>
<td>10–110</td>
<td>8.6</td>
</tr>
<tr>
<td><strong>URINE</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Specific gravity</td>
<td>1.015–1.05</td>
<td>1.032</td>
</tr>
<tr>
<td>Protein</td>
<td>Negative</td>
<td>3+</td>
</tr>
<tr>
<td>Protein:creatinine ratio</td>
<td>&lt;0.5</td>
<td>4.2</td>
</tr>
</tbody>
</table>

*FIGURE 3. Leishmania infantum* amastigotes in a macrophage from a splenic aspirate of a clinically infected dog. Scale bar indicates 10 microns.
Outcome and Future Treatment

All clinical signs resolved within 2 to 4 weeks of starting allopurinol and meglumine antimoniate treatment, and after 8 months, the proteinuria resolved (FIGURE 4); however, the Leishmania titer remain elevated (1:1024).

Treatment with allopurinol will continue until titers significantly decline or become negative. If clinical signs return after the initial course of treatment, allopurinol therapy alone, or together with a course of miltefosine, will be restarted. Blood work, urine, and Leishmania immunofluorescence antibody titer will be monitored annually for the rest of the patient’s life. If no clinical signs recur, she will enjoy her life free of medication.

DISCUSSION

Leishmania are parasitic species that infect a variety of hosts, leading to the disease leishmaniasis. In the United States, several species have been associated with disease. Canine leishmaniasis, the visceral form of Leishmania species infection and the topic of this review, is caused predominantly by Leishmania infantum. Leishmaniasis (active disease) does not develop in all Leishmania-infected dogs, and some dogs will remain asymptomatic for their lifetime. In other dogs, the parasite can cause visceral, cutaneous, or mucosal leishmaniasis, and clinical disease can appear months to several years after exposure. All infected dogs, however, remain lifetime reservoirs. Disease progression depends on numerous factors, including the species of Leishmania, host immunocompetence, concomitant disease, and breed.

Globally, the main route of Leishmania transmission to humans and animals is through the bite of infected female phlebotomine sand flies. In the United States, according to the literature to date, a predominant means of Leishmania species is vertical transmission (transplacental or transmammary). Horizontal transmission (via blood transfusions and dog bites) has also been reported.

The first reported outbreak of canine leishmaniasis in the United States was in 1999, in a foxhound kennel in New York. By 2005, seropositive foxhounds had been reported from 60 kennels in 22 U.S. states and 2 Canadian provinces. Disease spread was believed to be limited to vertical transmission between these foxhounds, mostly via breeding and blood transfusions. A Centers for Disease Control and Prevention investigation indicated that the infected foxhounds in 1999 originated from southern France, were imported into Great Britain, and were subsequently brought to the United States, suggesting that the origin of the initial outbreak was from imported infected dogs. According to data for U.S. cases between 2006 and 2019, other hypothesized modes of transmission include horizontal transmission through direct dog-to-dog contact, unidentified sand flies, or another species of arthropod.

Although the current literature supports the finding that vertical transmission is the main cause of canine leishmaniasis in the United States, the large number of dogs imported annually into the United States from Leishmania-endemic countries raises concerns about unreported and undiagnosed cases, especially when we
now know that these dogs, if infected, can spread *Leishmania* species by vertical and horizontal transmission.

Each year, an estimated 1 million dogs are imported into the United States for adoption or by commercial breeders. Currently, there is no requirement for dogs to be screened for *Leishmania* before being imported into the United States. Therefore, it is imperative that U.S. veterinarians be aware of their patients’ travel history and that newly imported dogs be screened as soon as possible. Because progression of the disease depends on the type of immune response developed by infected dogs, it can take months to years for detectable antibodies or clinical disease to develop. Long-term follow-up testing is therefore recommended. Prompt identification and initiation of treatment for an infected dog are critical for improving the dog’s prognosis, and sterilization should be performed to reduce the risk for vertical transmission.

Given the rising autochthonous cases in the United States, in addition to screening for *Leishmania* in imported dogs, U.S. veterinarians should be able to recognize canine leishmaniasis in their patients, which can be extremely challenging as the clinical symptoms are nonspecific and common to many other chronic inflammatory diseases. Leishmaniasis results in damage to multiple organs and can vary from subclinical to life-threatening visceral disease. Factors such as immunosuppression or concomitant disease can cause subclinical infection to progress very quickly to clinical disease.

The most frequent manifestation of canine leishmaniasis is skin lesions (e.g., dermatitis; hyperkeratosis of nasal planum, paws, or haired skin; paw pad fissures) and may accompany other clinical signs or clinicopathologic abnormalities. However, the main presenting clinical signs can be unrelated to cutaneous lesions, including generalized (e.g., fever, weight loss, anorexia, lethargy, vomiting, diarrhea), ocular (e.g., uveitis, keratoconjunctivitis), systemic (e.g., glomerulonephritis, hepatomegaly and/or splenomegaly), musculoskeletal (e.g., polyarthritis, lameness), and, less commonly, neurologic disorders. The most frequent laboratory abnormalities are nonregenerative anemia; leukopenia/leukocytosis; hyperproteinemia; hypoalbuminemia; polyclonal hypergammaglobulinemia; reduced albumin/globulin ratio; and in severe cases, hematuria, proteinuria, and isosthenuria. Death often results from chronic renal failure secondary to immune complex glomerulonephritis. Leishmaniasis should be considered for all patients with these clinical signs and especially for dogs that were imported from, or traveled to, a country where leishmaniasis is prevalent.

Diagnosis of canine leishmaniasis requires a clinicopathologic diagnostic component (e.g., CBC, biochemistry, urinalysis) together with 1) serologic detection of *Leishmania*-specific antibodies (immunoglobulin G) by either enzyme-linked immunosorbent assay (ELISA) or immunofluorescence antibody test (IFAT) quantitative serologic techniques, and/or 2) detection of *Leishmania* amastigotes by cytologic or histopathologic examination of affected

### TABLE 3 Management Scenarios for Dogs with Leishmaniasis

<table>
<thead>
<tr>
<th>FINDINGS</th>
<th>INTERPRETATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate to high antibody levels together with clinical signs and clinicopathologic abnormalities consistent with leishmaniasis, with or without positive PCR or organism identified by cytology/histopathology</td>
<td>Considered active disease and treatment is warranted⁹</td>
</tr>
<tr>
<td>Medium to high antibody levels, negative PCR, no clinicopathologic abnormalities, no organism identified by cytology/histopathology, and clinically asymptomatic</td>
<td>Considered asymptomatic infection. Controversy exists regarding treatment options. These dogs can be treated or solely monitored for early detection of clinical signs and/or laboratory abnormalities suggesting presence of active disease. Some suggest using immunomodulators (e.g., domperidone) to potentially boost cellular immune response and delay clinical disease. Dogs can become symptomatic at any time, even years, after infection. After a dog becomes symptomatic, leishmanicidal treatment should be started.</td>
</tr>
<tr>
<td>Low antibody levels; negative PCR; and no clinical signs, clinicopathologic abnormalities, or organism identified by cytology/histopathology</td>
<td>Considered exposed to or infected with <em>Leishmania</em>, but no active disease. For these patients, treatment is not recommended; however, they should be carefully monitored for early detection of any clinical signs and/or laboratory abnormalities suggesting the presence of active disease as they can become symptomatic at any time, even years, after infection.⁹</td>
</tr>
</tbody>
</table>

*PCR = polymerase chain reaction*
tissues. PCR screening of peripheral blood for *Leishmania* DNA is not recommended because the sensitivity is low in healthy and sick infected dogs, thus resulting in false-negative results. PCR screening of tissue (e.g., lymph node, spleen, skin, conjunctiva, bone marrow), however, enables quantification of the *Leishmania* parasite load in the tissues of infected dogs, which is useful for diagnosis and treatment.

Confirming a diagnosis of canine leishmaniasis is complicated because serologic tests do not differentiate between clinical disease and asymptomatic infection and do not distinguish between exposure and current infections. There are several possible management scenarios based on organism identification and serologic test results (TABLE 3).

When treatment is warranted, the regimens described worldwide as most effective include a combination of either subcutaneous meglumine antimoniate or oral miltefosine (leishmanicidal agents) together with allopurinol to reduce the parasite load. Simultaneous use of immunostimulants has also been recommended.

When unable to source leishmanicidal agents, allopurinol alone is an acceptable alternative.

In the United States, there are currently no Food and Drug Administration (FDA)–approved drugs for the treatment of leishmaniasis in any non-human species, which makes sourcing medications challenging. Compounding the difficulty, the liquid form of miltefosine (sold under the brand name Milteforan [Virbac, us.virbac.com/home]) and meglumine antimoniate are not available in the United States and must be obtained according to FDA requirements for importing veterinary drugs. There is, however, a capsule form of miltefosine (50 mg capsules) sold under the brand name Impavido that has been FDA-approved in the United States for treatment of leishmaniasis in humans over the age of 12 years. This drug has been successfully used off-label to treat canine and feline leishmaniasis and can be ordered directly from the manufacturer by a licensed veterinarian. Impavido is supplied in capsule form, which can facilitate compounding for precise dosing. The dose has been extrapolated from the recommendations for the

| TABLE 4 Leishmaniasis Treatment Protocols for U.S. Veterinarians |
|-----------------|-----------------|-----------------|-----------------|-----------------|
| **DRUG**        | **DOSE**        | **SIDE EFFECTS**| **AVAILABILITY**| **NOTES**       |
| Miltefosine     | 2 mg/kg PO q24h| Anorexia, Diarrhea, Vomiting | Readily available in the United States | Capsule form facilitates dose adjustment via compounding |
| (Impavido)      | for 4 weeks    |                  |                  | Recommended to be given in combination with allopurinol |
| Allopurinol     | 10 mg/kg PO q12h| Xanthine urolithiasis, Renal mineralization | Readily available in the United States | To minimize risk for xanthine urolithiasis, a low-purine diet, such as Royal Canin Urinary UC Low Purine (royalcanin.com), is recommended. |
|                 | for at least 6-12 months |                  |                  | 50% dose reduction is recommended for patients with renal dysfunction |
| Meglumine antimoniate | 75-100 mg/kg SC q24h; for large volumes, 40-75 mg/kg SC q12h for 4-6 weeks | Potential nephrotoxicity, Pain and inflammation at injection sites, Abscessation/cellulitis | Not available in the United States, but can be imported in compliance with FDA requirements | Initial dose should be reduced for 1-3 days to monitor for any adverse reactions |
| Domperidone     | 0.5 mg/kg PO q24h for 1 month | Galactorrhea, Polyuria | Available in a gel form for horses in the United States (Equidone; Dechra, dechra-us.com) | None |

*Guidelines based on LeishVet.org recommendations and tailored for U.S. veterinarians. FDA = Food and Drug Administration*
BOX 2 Practical Tips

- Know the patient’s travel history. Leishmaniasis should always be considered when evaluating a dog that was imported from, or traveled to, a country where *Leishmania* is prevalent.
- Consider leishmaniasis for dogs with any of these hallmark clinical signs: fever, weight loss, anorexia, various dermatologic (e.g., dermatitis; hyperkeratosis of nasal planum, paws or haired skin; paw pad fissures); ocular (e.g., uveitis, keratoconjunctivitis); systemic (e.g., glomerulonephritis, hepatomegaly, splenomegaly); or musculoskeletal (e.g., polyarthritis) disorders.
- Consider leishmaniasis for dogs with the following abnormal blood work: unexplained nonregenerative anemia, hypoaalbuminemia, hyperglobulinemia (polyclonal gammopathy), and proteinuria, even if the patient has no known international travel history.
- Consider leishmaniasis for any dog with skin scales, crusts, or ulcerations, especially with nonhealing or progressive skin lesions on the nose and ears.
- Diagnose canine leishmaniasis by serologic testing and/or identification of *Leishmania* amastigotes on cytologic or histopathologic examination of affected tissues. Polymerase chain reaction detection of *Leishmania* DNA in blood is not recommended as a screening test because the sensitivity of this blood test is low for both healthy and sick *Leishmania*-infected dogs.
- Monitor dogs exposed to *Leishmania* for life as they can go for months to years without significant titers or manifestation of disease.
- Do not treat *Leishmania* infection in a clinically healthy dog, but monitor the dog for life for clinical signs and/or laboratory abnormalities suggesting presence of disease. For best response to treatment, start therapy for sick dogs as soon as possible.
- Treat clinical leishmaniasis (considered as high antibody titers and/or compatible clinical manifestations and laboratory abnormalities) in any dogs. Although treatment recommendations are generally accepted globally, availability of medications in the United States dictates the treatment options available to U.S. veterinarians. Medications readily available in the United States are Impavido (miltefosine) and allopurinol.

Allopurinol is widely available in the United States and can be used off-label to treat canine leishmaniasis. In addition to medical treatment, while dogs are receiving allopurinol, diets should be transitioned to low-purine dog food to minimize the risk for the potential side effect of xanthine urolithiasis.

Medical therapy does not cure canine leishmaniasis but can result in improved clinical signs, normalization of blood work, seronegative IFAT or ELISA results, and decreased parasite burden, thereby reducing the chances of transmission. No therapy has been documented to completely eliminate the parasite; therefore, relapses can occur, requiring repeated treatment. Follow-up clinical evaluation including CBC, biochemistry, and urinalysis should be done every 3 to 4 months the first year of treatment and then every 3 to 6 months thereafter, depending on the clinical picture. Repeat serologic testing with a quantitative assay is recommended 6 months after starting therapy and then every 6 to 12 months. Lifetime monitoring of antibody-positive dogs is indicated and should include regular examinations, laboratory screening, and quantitative titer testing.

Because no treatment has been shown to completely eradicate the *Leishmania* parasite, dogs treated for canine leishmaniasis remain lifetime reservoirs of infection. Therefore, use of repellent insecticides, such as synthetic pyrethroids, can help prevent *Leishmania* transmission to other animals and/or humans. It is also recommended that *Leishmania*-positive dogs be sterilized and not used as blood donors.

**SUMMARY**

Leishmaniasis is one of the most common zoonoses worldwide and a serious public health concern, yet it is often overlooked and neglected in dogs in the United States. With the number of cases of canine leishmaniasis rising in the United States, it is imperative that veterinarians become familiar with the clinical signs as well as proper diagnosis, treatment, and management. In the past decade, several vaccines have been developed and used to prevent or aid in treatment of disease in *Leishmania*-endemic areas. Developing a program for screening dogs imported into the United...
States from *Leishmania*-endemic regions and ensuring that U.S. veterinarians have the information necessary to screen, identify, and treat patients with leishmaniasis is critical for preventing the spread of *Leishmania* in the United States (BOX 2). TVP

References


Diane Levitan

Dr. Levitan is an associate professor of small animal medicine at Long Island University College of Veterinary Medicine. With more than 30 years of practice, she has built several veterinary hospitals, charities, and novel businesses. She has introduced many concepts into the field of veterinary medicine, such as the first hospital where families could stay overnight with their pets and hyperbaric oxygen therapy for animals. She has a passion for teaching and mentoring and founded Chirp Vet Mentors. Through charitable work, she has become an advocate for dogs with leishmaniasis.

Jacqueline Finnegan

Ms. Finnegan is a former neonatal intensive care nurse and healthcare attorney. A prolific legal author, she maintained a legal column for the American College of Radiology and has written articles in several other legal publications. She now dedicates her time to raising her 3 children, volunteering for numerous animal welfare charities, and using her legal and writing skills to help raise awareness about animal welfare issues. Through her charitable work with international animal rescue groups, she has been involved in the screening, treatment, and monitoring of dogs infected with *Leishmania* both overseas and in North America.