Tick-borne Diseases of Companion Animals: An Update
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Indiana Ticks and Changes in Distribution

Indiana is home to more than 15 different species of ticks. However, four species of ticks represent the most common and important vectors of tick-borne disease in companion animals. These are the Blacklegged tick (*Ixodes scapularis*), Lone Star tick (*Amblyomma americanum*), American dog tick (*Dermacentor variabilis*), and brown dog tick (*Rhipicephalus sanguineus*). Changes in climate and increased pet transportation have increased the prevalence of tick-borne diseases within the mid-eastern United States.[4,10]

The Importance of Tick Identification

If a tick is found on an animal, the client or clinician should attempt to at least identify the genus of the tick. Identification of a tick parasite aids in shortening the differential diagnosis list for many hemolytic and thrombocytopenic diseases. It is also important in monitoring the potential distribution of tick-borne diseases in both humans and animals.[2] Figure 1 provides a very basic algorithm for identification of the four most common ticks in Indiana. If a tick cannot be identified, it should be submitted to the Indiana Animal Disease Diagnostic Laboratory (ADDL). Contact the ADDL for additional information on parasite identification services ((765) 494-7440; addl@purdue.edu).

Tick Key Facts by Species

Blacklegged Tick *(Ixodes scapularis)*

- 3 host tick
  - -Small mammals, birds, and lizards (larvae and nymphs)
  - -White-tailed deer, humans, dogs, cats, and other larger mammals (adults)
- Identifying features:
  - -Palps of capitulum broadened at junctions of second and third segments
  - -Very long mouthparts
  - -Nonornamental scutum
  - -No festoons
  - -Anus and anterior anal groove
- Potential vector of:
  - -Lyme disease (*Borrelia burgdorferi)*
  - -Anaplasmosis (*Anaplasma phagocytophilum*)
  - -Babesiosis

Lone Star Tick (*Amblyomma americanum*)

- 3 host tick
  - -Small mammal, white-tailed deer, humans, dogs and other mammals (larvae, nymphs, and adults)
- Identifying features:
  - -Very long mouthparts
  - -Ornamental scutum
- Potential vector of:
  - -Rocky Mountain Spotted Fever (*Rickettsia rickettsii)*
  - -Ehrlichiosis (*Ehrlichiosis chaffeensis* and *Ehrlichiosis ewingii*)
  - -Cytauxzoonosis – pattern of distribution more closely related than ADT

American Dog Tick (*Dermacentor variabilis*)

- 3 host tick
  - -Small rodents (larvae and nymphs)
  - -Humans, dogs, horses, cattle, wildlife (adults)
- Identifying features:
  - -Rectangular basis capituli
  - -Festoons
  - -Ornamental scutum
- Potential vector of:
  - -Rocky Mountain Spotted Fever (*Rickettsia rickettsii)*
  - -Ehrliciosis (*E. canis* and *E. chaffeensis)*
  - -Cytauxzoonosis

Rhipicephalus sanguineus (Brown Dog Tick)

- 1 host tick
  - -All stages feed on dogs and sometimes humans
- Identifying features:
  - -Hexagonal basis capituli
  - -Eyes and festoons
  - -Nonornamental scutum
- Potential vector of:
  - -Rocky Mountain Spotted Fever (*Rickettsia rickettsii)*
  - -Ehrlichiosis (*Ehrlichia canis)*
  - -Babesiosis (Boozer)
  - -Anaplasmosis (*Anaplasma platys*)
- Can be found inside buildings and kennels

Tick-borne Diseases

Lyme Disease

Lyme disease is caused by the spirochete, *Borrelia burgdorferi.* Rodent and other small mammal populations in endemic areas serve as the reservoir for *B. burgdorferi*. Migratory birds may also play a key role in its increasing prevalence. Ticks are the primary mode of transmission of the organism to dogs and cats, although other bloodsucking insects have been suggested. Preventing bites from *Ixodes scapularis,* the primary tick vector, is the key to Lyme disease prevention.[6] It takes at least 50 hours of tick attachment for transmission of the organism.[8] After being inoculated into the host, *B. burgdorferi* has changes in outer-surface proteins (Osp) that allow it to adapt in response to host conditions.[9] The Osp is the target for current recombinant vaccines.

Diagnosis of Lyme disease is made primarily on history, clinical signs with a concurrent C6 peptide ELISA SNAP or quantitative test, and response to antibiotic therapy. A serological titer alone does not indicate current infection. In fact, over 95% of infected dogs are asymptomatic.[6]

The classic signs of Lyme disease in dogs are non-erosive, secondary immune-mediated polyarthritis, anorexia, lymphadenopathy and fever.[3, 6, 8] Many dogs with high *B. burgdorferi* antibody titers also develop a lymphoplasmacytic, interstitial nephritis or immune-mediated glomerulonephritis.[6] The clinical signs of Lyme disease in cats have not been described in the literature. However, cats can be infected with *B. burgdorferi.* Infection in dogs has been associated

with myocarditis, which clinically manifests as third or high-grade second-degree atrioventricular block. Histopathologically, there are focal areas of myocardial necrosis and both mono- and polynuclear leukocyte infiltration. Cardiovascular clinical signs usually do not resolve with treatment. Rarely, dogs with Lyme disease may develop cerebral-associated neurological signs such as changes in behavior and seizures. In the case of neurological signs, early antibiotic treatment may be effective.[8] Treatment of Lyme disease includes supportive care and administration of a tetracycline antibiotic.

Prevention of Lyme disease is concentrated on tick control, client education, and monitoring for clinical signs. Administration of the bacterin or rOspA Lyme vaccine is still controversial and not recommended in moderate-risk states, such as Indiana. However, the ultimate decision for vaccination should be made on a case-by-case basis between the client and veterinarian.

In humans, symptoms of *B. burgdorferi* exposure are acute flu-like signs, erythema migrans rash, arthritis and regional lymphadenopathy. Less common symptoms may include cardiac, neurologic or dermatologic changes.[9]

Rocky Mountain Spotted Fever

Rocky Mountain Spotted Fever (RMSF) is caused by *Rickettsia rickettsii. R. rickettsii* can infect both dogs and cats. The primary tick vector is *Dermatocenter variabilis* but *Amblyomma americanum,* and *Rhipicephalus sanguineous* have also been implicated in its spread. The organism is maintained in small rodent reservoirs. It is spread transovarially, therefore, nymphs and larvae do not need to feed to become infected.[8] Clinical signs of RMSF are related to the effects of a secondary vasculitis, caused by replication of the organism in epithelial cells. These signs include malaise, petechial hemorrhages of the retina and skin, gravity-dependent edema, and cardiovascular and nervous system dysfunction.[7] Less commonly, RMSF can cause a non-erosive, secondary immune-mediated polyarthritis.[8] Diagnosis of RMSF is based upon clinical signs, serologic tests and response to treatment. Treatment and prevention is similar to Lyme disease and other tick-borne bacterial diseases.

Ehrlichiosis

Ehrlichia canis, Ehrlichia chaffeensis, and *Ehrlichia ewingii* have all been identified as the cause of canine and feline ehrlichiosis. *Rhipicephalus sanguineus* and *Amblyomma americanum* are the primary vectors of *Ehrlichia spp.* The cellular tropism differs between *E. canis* and *E. chaffeensis* and *E. ewingii*. *E. canis* and *E. chaffeensis* cause monocytrophic ehrlichiosis and *E. ewingii* canine granulocytrophic ehrlichiosis. Severity of clinical signs varies greatly depending on the species of *Ehrlichia*, co-infection with other organisms, and animal's immune system. Clinical signs of *E. erwingii* are less severe than *E. canis.*[8]

The incubation period for canine monocytrophic ehrlichiosis (*E. canis)* from bite to infection of monocytes is 1-3 weeks. *E. canis* can be transmitted by male ticks and does not require females for rapid spread since transovarian transmission is uncommon. The subclinical stage of disease may last for many years after infection with some animals never entering the clinical phase. The most common signs of ehrlichiosis are similar to those of RMSF and include fever, petechiation, thrombocytopenia and related sequelae, anemia, neurological signs, and ocular lesions.

Similar signs have been found in feline monocytropic ehrlichiosis. However, culture or isolation of *Ehrlichia sp.* has not bet reported.[8] *E. chaffeensis* is the cause of monocytrophic ehrlichiosis in humans.

Infection with *E. erwingii* produces similar signs to *E. canis* but is more likely to cause suppurative polyarthritis.[5, 7]

Diagnosis of ehrlichiosis is based upon PCR analysis and presence of clinical signs. The antibody titer for *Ehrlichia* spp. (especially *E. canis*) many vary depending on clinical stage. Positive antibody tests (including point-of-care SNAP tests) should be rechecked or confirmed by PCR to prevent false positive results. The decision to treat a seropositive yet clinically negative dog should be made in conjunction with each owner, as there are pros and cons to treatment. Treatment is the same as for other tick-borne illnesses and should be continued until negative PCR tests are obtained at 2 and 10 weeks post-treatment.[8]

Babesiosis

Babesia canis and *Banesia gibsonii* are the primary apicomplexan protozoal etiological agents of babesiosis in dogs. After feeding for at least 48 hours, ticks release sporozoites into circulation. The sporozoites attach to erythrocytes and are phagocytized into cells where they replicate. *Babesia spp.* can be transmitted through vertical, blood-borne and bite wounds. Greyhound and pit bull dogs are the dog breeds commonly associated with babesiosis.[1] Clinical disease ranges from subclinical to acute fatal infections. Fever, depression, anorexia, splenomegaly, thrombocytopenia, and hemolytic anemia have all been associated with infection with *Babesia spp.*[4] Due to the similarity of signs between babesiosis and immune-mediated hemolytic anemia, infection with *Babesia spp.* must be ruled out before administering immunosuppressive agents. Differentiation between *B. canis* and *B. gibsoni* is based upon identification of the organisms on blood smear. *B. canis* is bigger and usually found in pairs. As with other tick-borne diseases, presence of a serologic antibody titer is suggestive of previous exposure to *Babesia spp.* but not necessarily concurrent disease. Rising titers or a positive PCR test along with clinical signs is the most sensitive and specific way to diagnose babesiosis. Treatment is similar to that of other tick-borne illnesses.[8]

Cytauxzoonosis

Cytauxzoon felis is the protozoan agent of cytauxzoonosis first described in Missouri in 1979.[4] Since then the range of *Cytauxzoon felis* has expanded and has been reported in Indiana. Bobcats and feral cats are the reservoir hosts and *Dermacentor variabilis* is the primary tick vector of *C. felis* to domestic cats. Both clinical and subclinical infection of cats is possible with most of the pathology characterized by the organism's tropism for mononuclear phagocytes. The clinical signs of cytauxzoonosis are nonspecfic signs, anemia, icterus, dyspnea, leukopenia, thrombocytopenia, and splenomegaly. Diagnosis is based upon visualization of the organism in erythrocytes. PCR tests are also available for *C. felis*. Infection with *C. felis* has historically been associated with a poor prognosis and numerous antibiotics have been administered in an attempt to clear the disease. However, an increased likelihood of survival has been most strongly associated with the implementation of early supportive care.[4] It is important to communicate to clients that preventing exposure and prompt removal of ticks are the best defense against *C. felis* infection.

Anaplasmosis

Anaplasma phagocytophilum and *Anaplasma platys* are both associated with canine anaplasmosis. *Ixodes scapularis* is the only recognized vector of *A. phagocytophilum* in Indiana. *A. platys* is transmitted by *Rhipicephalus sanguineous*. Clinical signs of infection with *A. phagocytophilum* include nonspecific signs, anemia, splenomegaly, lymphadenopathy, and thrombocytopenia with subsequent bleeding disorders. *A. phagocytophilum* can also cause a nonerosive, secondary immune-mediated polyarthritis.[3] Infection with *A. platys* produces a recurrent thrombocytopenia, which cycles every 2 weeks with the production of new platelets. Ocular signs are also often present. Cats are thought to acquire *Anaplasma spp.* but experimental infection of cats has not been successful. Diagnosis and treatment is similar as for the other tick-borne illnesses.[5]

Tick Prevention and Information for Clients

Tick-borne disease prevention begins with avoidance of tick habitats. Clients should be advised to remove or trim unnecessary vegetation on their property to help reduce both human and animal exposure to ticks. Additional information on indoor and outdoor environmental tick control can be found through the Purdue Extension Office (1-888-EXT-INFO).[10] Even with avoidance of tick habitats, clients should be advised to use acaricides year-round.

References:

1. Boozer AL, Macintire DK. Canine babesiosis. *Vet Clin Small Anim*. 2003(33):885-904.
2. Bowman, DD. Georgis' Parasitology for Veterinarians. 9th ed. Saunders. 2009.
3. Ettinger and Feldman. Textbook of Veterinary Internal Medicine. 7th ed. 2010.
4. Holman PJ, Snowden KF. Canine Hepatozoonosis and Babesiosis, and Feline Cytauxzoonosis. Vet Clin Small Anim 2009(39):1035-1053.
5. Little SE. Ehrlichiosis and Anaplasmosis in Dogs and Cats. *Vet Clin Small Anim* 2010(40) 1121–1140.
6. Littman MP, Goldstein RE, Labato MA, Lappin MR, Moore GE. ACVIM Small Animal Consensus Statement on Lyme Disease in Dogs: Diagnosis, Treatment and Prevention. *J Vet Intern Med* 2006(20): 422-434.
7. Mikszewski JS, Vite CH, Central Nervous System Dysfunction Associated with Rocky Mountain Spotted Fever Infection in Five Dogs. 2005 (41):259-266.
8. Nelson and Couto. Small Animal Internal Medicine. 4th ed.
9. Tilly K, Rosa PA, Stewart PE. Biology of Infection with *Borrelia burgdorferi*. *Infect Dis Clin N Am.* 2008 (22): 217-234.
10. Williams, RE. Ticks – Biology and their Control. E-71-W. Revised 2010. Purdue Extension, Purdue University Department of Entomology.