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INDICATIONS:
Credelio Quattro is indicated for the prevention of heartworm disease caused by *Dirofilaria immitis* and for the treatment and control of roundworm (immature adult and adult *Toxocara canis* and adult *Toxascaris leonina*), hookworm (adult *Uncinaria stenocephala*), and tapeworm (*Dipylidium caninum, Taenia pisiformis,* and *Echinococcus granulosus*) infections. Credelio Quattro kills adult fleas and is indicated for the treatment and prevention of flea infestations (*Ctenocephalides felis*) and the treatment and control of tick infestations [*Amblyomma americanum* (lone star tick), *Dermacentor variabilis* (American dog tick), *Ixodes scapularis* (black-legged tick), and *Rhipicephalus sanguineus* (brown dog tick)] for one month in dogs and puppies 8 weeks of age and older and weighing 3.3 pounds or greater.

IMPORTANT SAFETY INFORMATION:
Lotilaner, an ingredient in Credelio Quattro, belongs to the isoxazoline class and has been associated with neurologic adverse reactions like tremors, ataxia, and seizures even in dogs without a history of seizures. Use with caution in dogs with a history of seizures or neurologic disorders. Dogs should be tested for existing heartworm infections before Credelio Quattro administration as it is not effective against adult *D. immitis*. The safe use in breeding, pregnant, or lactating dogs has not been evaluated. The most frequently reported adverse reactions in clinical trials were vomiting and diarrhea.
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FROM THE EDITOR

Keeping an Eye on the Trends

With new technology emerging seemingly every day, client demands shifting with the times, and product launches dominating the headlines, there is no shortage of *new* for practitioners to track. Toss in emerging parasitic diseases, the expanding range of vectors, and updated recommendations on diagnostics, and it can all feel overwhelming. This resource compiles a collection of peer-reviewed articles published in *Today's Veterinary Practice*. When planning this e-book, our editorial staff aimed to highlight the updates and emerging topics that will help keep practitioners up to date. The goal? To ease the burden of seeking out the new updates by bringing analysis from leading experts on some key topics in one place.

Author Bios

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Dr. Aicher is an assistant professor in the Gastrointestinal Laboratory at Texas A&M University. She earned her DVM degree from Texas A&M, followed by an internship at the University of Tennessee and a residency at North Carolina State University. She worked in private practice as an internist and medical director before returning to Texas A&M, where she teaches in the preclinical DVM curriculum, performs clinical duty on the small animal internal medicine service, and conducts research investigating liver and gastrointestinal disease of dogs. Her love for retrievers led her to develop a research subgroup known as The Drake Project, investigating *Heterobilharzia americana*.

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Dr. Burton is a medical affairs specialist for infectious diseases at IDEXX and has delivered numerous educational presentations at veterinary schools, conferences, and veterinary medical association meetings. Prior to joining IDEXX in 2015, he was an associate in a mixed animal practice and a practice owner. He continues to work with industry leaders to educate and inform the veterinary community, including developing research, educational tools, and publications. He currently serves on the Texas A&M College of Veterinary Medicine Development Council and is a member of the Texas A&M Veterinary Emergency Team.

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Dr. Duncan is an assistant professor at Oklahoma State University's College of Veterinary Medicine. She obtained her DVM degree from the University of Tennessee, and her National Center for Veterinary Parasitology clinical residency in parasitology and PhD degree were conducted at Oklahoma State University. In 2022, she became board certified in parasitology through the American College of Veterinary Microbiologists. Currently, she teaches veterinary parasitology at Oklahoma State University, where she also has an active research laboratory. Her publications and professional interests include ticks, vector-borne diseases, and internal parasites of domestic animals.

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Dr. Poellmann is a postdoctoral research associate in the Gastrointestinal Laboratory at Texas A&M University. She completed her DVM degree at the Ludwig Maximilian University of Munich, Germany. Following her graduation, Dr. Poellmann joined the Gastrointestinal Laboratory to further her academic and research career by pursuing a Dr. med. vet. degree. Her research focuses on studying *Heterobilharzia americana* infections in dogs, particularly in Texas and the southwestern United States.

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Dr. Scimeca is an assistant professor at Oklahoma State University College of Veterinary Medicine and the parasitology diagnostics department head at the Oklahoma Animal Disease Diagnostic Laboratory, Oklahoma State University. Dr. Scimeca dedicates the majority of her time to diagnostics. She enjoys working in research, focusing mainly on protozoans, tick-borne pathogens, host immune response to parasitic diseases, and development of new parasitology diagnostic tests. Dr. Scimeca also teaches fourth-year veterinary students at the College of Veterinary Medicine during their clinical diagnostic rotation.

Diagnosing and Managing *Anaplasma* Infection in Dogs

***Kathryn Duncan**, DVM, PhD, DACVM (Parasitology), Oklahoma State University College of Veterinary Medicine, Stillwater, Oklahoma*

***K. Wade Burton**, DVM, IDEXX Laboratories*

Signs of canine anaplasmosis may manifest following infection with either *Anaplasma phagocytophilum* or *Anaplasma platys*. However, many dogs with circulating antibodies to *Anaplasma* species do not exhibit obvious signs of disease. Both agents of anaplasmosis are transmitted by ticks, and their distribution corresponds with the range of their vector.

As tick-borne diseases continue to spread and vector-borne disease diagnostics continue to improve, veterinarians are diagnosing more dogs with *Anaplasma* infections. In managing seropositive dogs, consistent messaging for clinic staff and pet owners regarding diagnostic and control recommendations is critical for widespread awareness and success of preventive measures.

ANAPLASMOSIS IN DOGS

A phagocytophilum and *A platys* are well-known agents of anaplasmosis in dogs (**TABLE 1**). Both are obligate intracellular bacteria found globally and transmitted by ticks commonly found on dogs in North America. Prior infection with *Anaplasma* species does not appear to offer full protective immunity, meaning dogs are susceptible to reinfection.[2]

TABLE 1 Important Characteristics of the Agents of Anaplasmosis in Dogs in North America

	ANAPLASMA PHAGOCYTOPHILUM	*ANAPLASMA PLATYS*
Disease name in dogs	Canine granulocytic anaplasmosis	Infectious canine cyclic thrombocytopenia
Clinical signs[a]	Lethargy, fever, anorexia, lymphadenopathy, joint swelling and stiffness, lameness	Fever, lymphadenopathy, petechiae, ecchymoses, pale mucous membranes
Laboratory findings	Thrombocytopenia, mild anemia, neutrophilic polyarthritis	Thrombocytopenia, leukocytosis
Distribution in United States	Northeastern, upper midwestern, and western regions	Southern regions
Tick vector	*Ixodes scapularis* and *Ixodes pacificus* (primarily)	*Rhipicephalus sanguineus* (suspected)
Main reservoir hosts	White-footed mice, redwood chipmunks, woodrats	Dogs
Public health concerns	Zoonotic	Potentially zoonotic
Differential diagnoses	Other vector-borne diseases (e.g., Lyme disease, ehrlichiosis), immune-mediated polyarthritis, hemolytic anemia	Other vector-borne diseases (e.g., ehrlichiosis), immune-mediated thrombocytopenia

[a]*Most seropositive dogs (60%) have no clinical signs.*[1]

A phagocytophilum resides and multiplies primarily in neutrophils before disseminating throughout the body. Seropositive dogs are found in the northeastern, upper midwestern, and western United States. *Ixodes scapularis* (**FIGURE 1A**) is the primary vector in the Northeast and Midwest, while *Ixodes pacificus* is the primary vector in the West. The majority of *I scapularis* adults seek hosts in the cooler months of the year, and this unique characteristic is worth mentioning to pet owners.[3] In nature, *A phagocytophilum* is maintained in various vertebrate hosts, such as rodents, and after feeding on a reservoir host, ticks remain infected after each molt.[2]

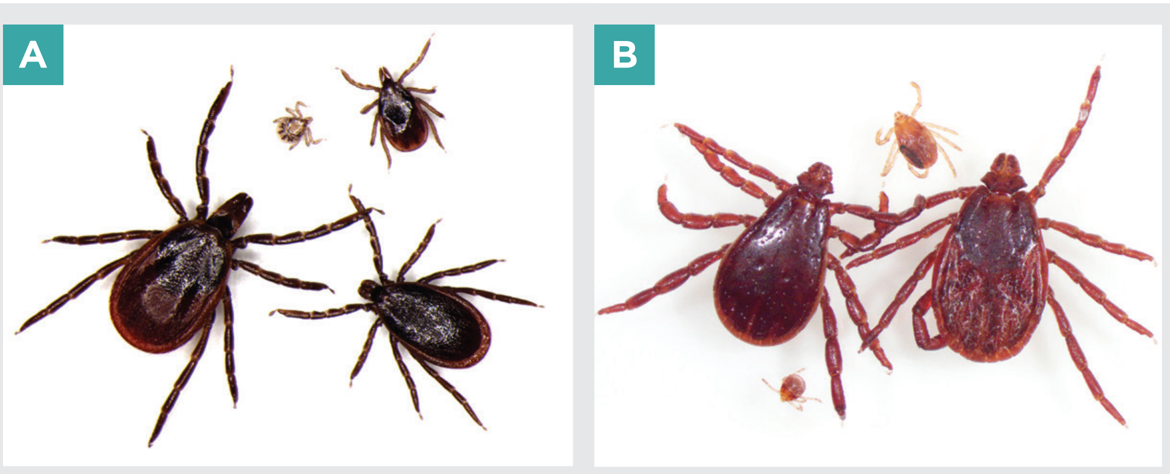

FIGURE 1. Tick vectors of the causative agents of canine anaplasmosis: **(A)** *Ixodes* species for *Anaplasma phagocytophilum* and **(B)** *Rhipicephalus sanguineus* (suspected) for *Anaplasma platys*.

A platys resides and multiplies in platelets, from which it disseminates. In the United States, most infections are diagnosed in southern regions where the suspected tick vector, the brown dog tick (*Rhipicephalus sanguineus*), thrives.[2] This tick species (**FIGURE 1B**) feeds on dogs at every life stage, and high tick burdens are not uncommon. Because this tick can infest kennels and homes, year-round activity and pathogen transmission is possible. Dogs are the primary reservoir for *A platys*, and brown dog ticks, once infected, can maintain infection between molts, allowing efficient pathogen transmission between dogs.[1]

Subclinical infections are common, with up to 60% of seropositive dogs not showing obvious signs of infection.[1] If present, disease occurs soon after infection with *A phagocytophilum*; however, persistent manifestations were noted experimentally and chronic infection with *A platys* is not uncommon.[2] With *A platys*, disease is often mild with recurrent thrombocytopenia every 10 to 14 days, which is attributed to phagocytosis of infected platelets. Other signs may include bleeding tendencies (e.g., epistaxis). Dogs with *A phagocytophilum* infection might be lethargic, anorexic, or feverish. Thrombocytopenia can be seen in >90% of patients and signs suggestive of polyarthritis, such as reluctance to move, could be present.[1,2]

TRENDS IN DIAGNOSTIC TESTING

Canine seropositivity to tick-transmitted *Anaplasma* species has increased over the past few years, and some veterinarians in endemic areas may be detecting *Anaplasma* antibodies more often than any other tick-borne disease agent. When reviewing canine test results from IDEXX between 2013 and 2019, researchers demonstrated the continued expansion of *A phagocytophilum* across the United States.[4] More recently in 2021, the Companion Animal Parasite Council documented that 1 out of every 50 tested dogs was seropositive to *Anaplasma* species, whereas in 2022, 1 in 30 dogs tested positive.[5,6] These escalating trends continued through 2023, and the reports may be explained, in part, by improved diagnostics and awareness. Several point-of-care enzyme-linked immunosorbent assays (ELISAs) exist, with a range of documented sensitivities and specificities; 1 ELISA reports sensitivity and specificity as high as 94.1% (95% confidence interval [CI]: 86.8% to 98.1%) and 98.4% (95% CI: 96.6% to 99.3%), respectively.[7]

Serology remains a popular method of detecting infections with *Anaplasma* species. Generally, it takes 1 to 2 weeks after infection before antibodies can be detected, and because there are varying levels of cross-reactivity, a positive result is reported at the genus level.[8] Other diagnostics to consider include blood smears for visualization of morulae in neutrophils (*A phagocytophilum*) or platelets (*A platys*) (**FIGURE 2**). Neutrophils infected with *A phagocytophilum* are more likely to be seen during the acute phase in sick dogs, and the evaluation of blood smears yields moderate to high sensitivity in those cases.[2] However, the lack of morulae on a blood film should not rule out an infection as parasitemia may be low in chronic phases.

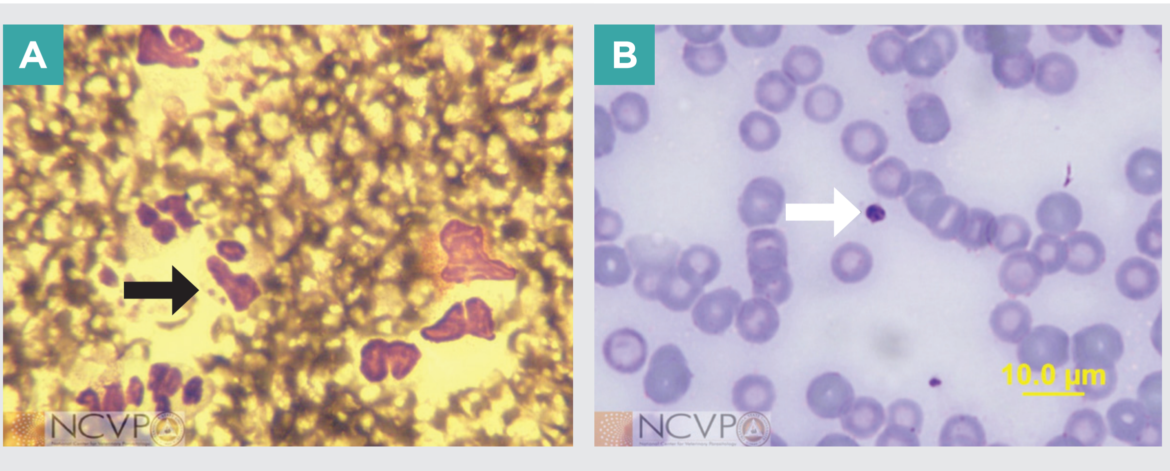

FIGURE 2. (A) Neutrophils infected with morulae of *Anaplasma phagocytophilum* **(black arrow)** and **(B)** platelets infected with morulae of *Anaplasma platys* **(white arrow)**.

Sending blood or tissue samples to a laboratory for polymerase chain reaction (PCR) testing can also be considered. In chronic cases, PCR testing of biopsy samples from spleen, lymph node, or bone marrow may yield a greater sensitivity than peripheral blood; however, whole blood collected during acute illness appears to have acceptably high sensitivity.[2] PCR results can take several days to return to the clinic, and, as with blood films, a negative result does not rule out infection with low parasitemia, particularly in chronic cases. For these reasons, a multimodal approach to diagnosing anaplasmosis is recommended and samples should be collected as soon as clinical illness presents.

MANAGING SEROLOGIC RESULTS

To assist veterinarians with managing seropositive dogs, some diagnostic companies have collaborated with veterinary specialists to develop clinical reference guides.[9] These guides can help veterinarians develop diagnostic and management plans and aid client communication.

Positive Result With Clinical Signs

If a dog is exhibiting clinical signs of anaplasmosis and a serologic test result is positive, next considerations should include a complete blood count (CBC) and blood film evaluation of fresh whole blood. If thrombocytopenia or anemia—with or without neutrophilia and monocytosis—is present, the results agree with a diagnosis of anaplasmosis. Appropriate treatment with doxycycline, or other tetracycline, should be strongly considered, particularly if no other cause for these abnormalities is found.[2] The diagnosis would be strengthened if morulae are found in neutrophils (*A phagocytophilum*) or platelets (*A platys*).

In diagnosed cases, a recheck CBC should be considered approximately 7 days later to assess progression of disease or response to treatment. Alternatively, if no morulae are detected on blood film and the CBC is within normal limits, the positive serologic test may be an incidental finding and the noticeable clinical signs could be due to another cause; therefore, other testing (e.g., PCR) should be considered. In these situations, it is important to remember that many dogs can be seropositive without obvious clinical signs. Additionally, the signs suggestive of tick-borne diseases (e.g., lethargy, fever) overlap with those of many other infectious diseases.

Negative Result With Clinical Signs

If a dog is exhibiting clinical signs suggestive of anaplasmosis but a serologic test result is negative, the dog may be displaying acute disease prior to development of antibodies. When disease is recognized in dogs, it is shortly (approximately 1 to 2 weeks) after infection and may slightly precede production of antibodies, which can become detectable as early as 8 days after infection.[1,8] In these cases, a second serologic test may be warranted in 2 to 4 weeks, and PCR testing should be strongly considered since it is more likely to detect acute infections.[2,8]

Since the window between clinical disease and antibody production is small, another cause may be responsible for the clinical signs observed. Performing additional diagnostic tests, such as a CBC, serum chemistry panel, blood film, urinalysis with urine protein to creatinine ratio, or tick-borne PCR panel, should be considered to explore other possibilities for the clinical signs.

Positive Result Without Clinical Signs

If a dog is not exhibiting clinical signs of anaplasmosis but a serologic test result is positive, a CBC and blood film are warranted. If morulae are seen on a blood film or thrombocytopenia is present on the CBC, then the dog may have anaplasmosis and treatment should be considered along with recheck testing of the CBC a week later. If no improvement is noted on recheck, other causes for the abnormalities should be investigated.

If a clinically normal, seropositive dog has a CBC within normal limits, the positive serologic result likely indicates a prior infection that remained undiagnosed or subclinical. In these cases, a recheck CBC can be performed in 7 days to ensure no clinical abnormalities develop, and treatment is not warranted if the CBC stays within normal limits.[2] Antibodies to *Anaplasma* species can be long-lived (i.e., several months or years), and chronically infected dogs may have high titers; however, if the pathogen is actively evading the immune system, a chronically infected dog may be seronegative at times.[2,8]

Owners of seropositive but apparently healthy dogs should be advised on the signs of anaplasmosis and educated on proper tick control and removal and the value of annual tick-borne disease testing.

Negative Result Without Clinical Signs

If no clinical signs of anaplasmosis are present and an annual serologic test result is negative, infection with *Anaplasma* species is unlikely. However, the many benefits of tick prevention and annual testing for common tick-borne diseases should be reviewed with the pet owner. There is enough convincing evidence to suggest that tick-borne diseases will continue to spread to new areas; thus, the infection risk is rarely zero.[10]

SUMMARY

Current trends in canine tick-borne diseases suggest anaplasmosis is a growing threat to dogs. As diagnostics improve and awareness of routine tick-borne disease testing increases, more dogs will be identified as seropositive to *Anaplasma* species. Not all seropositive dogs will display clinical signs, and the need for treatment is likely unnecessary in most cases. However, performing a CBC to assess platelet counts in seropositive dogs is necessary given the potential severity of disease; when thrombocytopenia is present, treatment should be considered. Additionally, PCR tests may provide more diagnostic evidence, but the results should be combined with serology and clinical history for treatment decisions. Evidence of infection by detection of antibodies presents the opportunity to educate clients on the need for annual tick-borne disease screening and strict tick control.

Rarer and Emerging Canine Tapeworms

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Several species of tapeworms can live as adults in the small intestines of dogs. In the United States, infections with tapeworms such as *Dipylidium caninum* and *Taenia pisiformis* are very common, while infections with tapeworms in the genera *Mesocestoides*, *Echinococcus*, *Dibothriocephalus*, and *Spirometra* are less common. This chapter will focus on identifying less common tapeworms that present an emerging threat.

RARER TAPEWORMS AND EMERGING INFECTIONS

Echinococcus

Known for many years to exist in wild canids,[1,2] *Echinococcus multilocularis* has been recently recorded in dogs in the contiguous United States.[3] Adult worms are less than 1 cm in length and consist of 3 to 5 proglottids (**FIGURE 1**). Terminal proglottids are shed and contain infective taeniid-type eggs. Typically, rodents serve as intermediate hosts, but humans can accidentally acquire the infection through ingestion of the eggs. Alveolar echinococcosis results in proliferative invasion of tissues of the intermediate host that can lead to death.

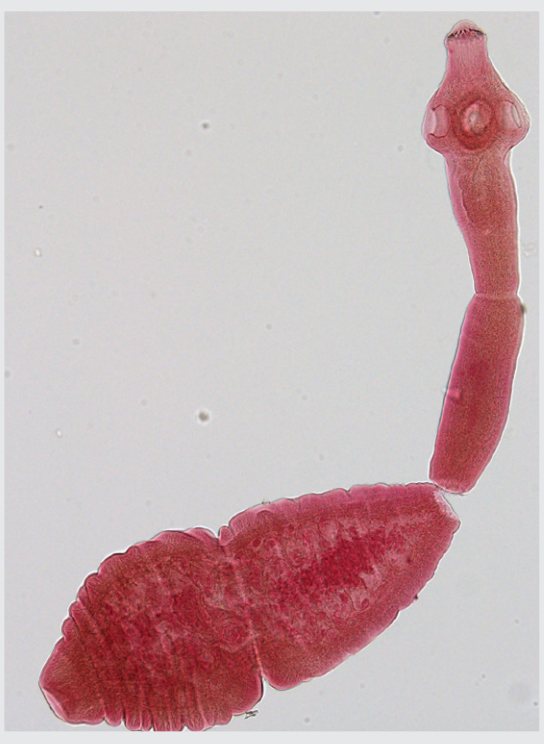

FIGURE 1. Adult worm of *Echinococcus* species.

Coproantigen enzyme-linked immunosorbent assays (ELISAs) and copro-PCR tests are useful diagnostic tests to help differentiate *Echinococcus* from *Taenia*.[4,5] Praziquantel is FDA approved for use in dogs against *Echinococcus* adults. Since taeniid-type eggs cannot be differentiated, veterinarians must be aware of the exposure risk they pose to clients, clinic staff, and the general public and advocate for the safe handling of dog feces.

Mesocestoides

Mesocestoides species may use dogs as definitive hosts or as second intermediate hosts. Adult worms live in the small intestine. Terminal proglottids are shed and contain a characteristic, medially located parauterine organ (**FIGURE 2**). Eggs do not possess the thick shell seen in taeniid-type eggs, are colorless with a hexacanth embryo, and are 30 to 40 microns in size. *Mesocestoide*s species use an insect as the first intermediate host and a vertebrate as the second intermediate host. Dogs may become accidental second intermediate hosts and suffer from peritoneal and/or pleural infections with larval stages of the parasite (tetrathyridia).

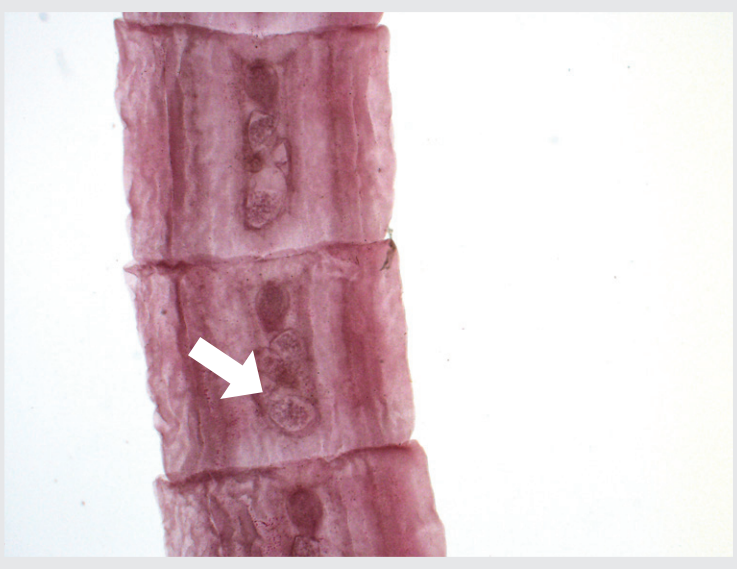

FIGURE 2. Nonterminal segment of *Mesocestoides* species stained with acetocarmine, which renders the proglottid pink. **Arrow** points to the parauterine organ, which is larger, rounded, and well developed in terminal gravid segments.

Treatment of the larval infection in dogs requires prolonged use of high doses of fenbendazole. Treatment of adult infections should be treatable with either praziquantel or epsiprantel.

Dibothriocephalus and *Spirometra*

Dibothriocephalus (formerly *Diphyllobothrium*) and *Spirometra* tapeworms, which are associated with aquatic intermediate hosts, can infect dogs, humans, and other mammals. In the United States, *Dibothriocephalus* is found in the Great Lakes region, while *Spirometra* is found in eastern and Gulf Coast states. *Dibothriocephalus* is acquired when dogs and humans ingest larval tapeworm stages (plerocercoids) found in fish tissue. Adult *Spirometra* are acquired when hosts ingest larval tapeworm stages found in the tissues of frogs, snakes, birds, or mammals. Adult tapeworms develop in the small intestines and may be passed out as complete worms in the vomitus or feces. Single eggs, not proglottids, are released and are detectable in fecal sedimentations. Infection is tolerated with minimal gastrointestinal signs of diarrhea, vomiting, weight loss and, in the case of *Dibothriocephalus*, pernicious anemia.

Although not FDA approved, praziquantel has been successfully used in treatment. Repeated treatments at higher doses may be necessary to achieve complete cure. Additionally, dogs may experience sparganosis should they become intermediate hosts of *Spirometra* by ingesting copepods with larval stages (procercoids) or through the entry of larvae via open wounds. These may cause nonpainful swellings or even death depending on the organ involved.

Diagnosis and Treatment of *Heterobilharzia americana* Infection in Dogs

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Canine schistosomiasis, caused by the trematode *Heterobilharzia americana*, is an infectious disease that is currently expanding its known range across the United States. Historically considered to be confined to the Gulf Coast and southern Atlantic states, *H americana* has recently established new endemic areas in the Southwest,[1,2] a finding that inspired prospective surveillance work currently in progress at the authors' diagnostic laboratory. The preliminary findings support unexpectedly high prevalence in areas of southern California and western Arizona that border the Colorado River. While many infected dogs remain asymptomatic, some progress to severe clinical complications, leading to multiorgan failure and fatal outcomes.

H americana is not detected by routine fecal screenings, and no preventives yet exist to reliably protect dogs from contracting the parasite in freshwater environments. Raising awareness within the dog-owning and veterinary medical communities is essential for early detection and effective management of the disease. This article shares insights from the authors' ongoing research to help equip veterinarians with the necessary tools for accurate diagnosis and successful treatment of canine schistosomiasis.

LIFE CYCLE AND ROUTES OF TRANSMISSION

H americana can infect a wide range of mammals.[3-6] It is frequently found in raccoons, nutria, and dogs.[7,8] The ability of this blood fluke to establish itself in an area depends on the presence of an intermediate snail host of the Lymnaeidae family that is capable of supporting the parasite's life cycle.[1,2,8] Upon excretion in the feces of an infected animal, fluke eggs hatch and release free-swimming miracidia when immersed in water. Miracidia penetrate the intermediate snail host to carry out asexual development into infectious cercariae, which are then released from the snail.

Dogs are at risk of contracting *H americana* when they wade or swim in cercariae-infested fresh water. A diagram of the life cycle of *H americana* is shown in **FIGURE 1**. Infection occurs when cercariae penetrate a dog's intact skin. Inside the dermis, the cercariae transform into a new juvenile state that enters dermal vessels and migrates to the lungs in approximately 5 to 9 days before reaching the liver between days 7 and 45 postinfection. Here, the parasites mature into adult male and female flukes. Adults settle in the mesenteric vasculature, mate, and produce eggs.[8] The eggs disseminate through the bloodstream to visceral organs or penetrate the intestinal wall to enter the lumen, from which they are subsequently excreted in feces approximately 84 days after infection.[9]

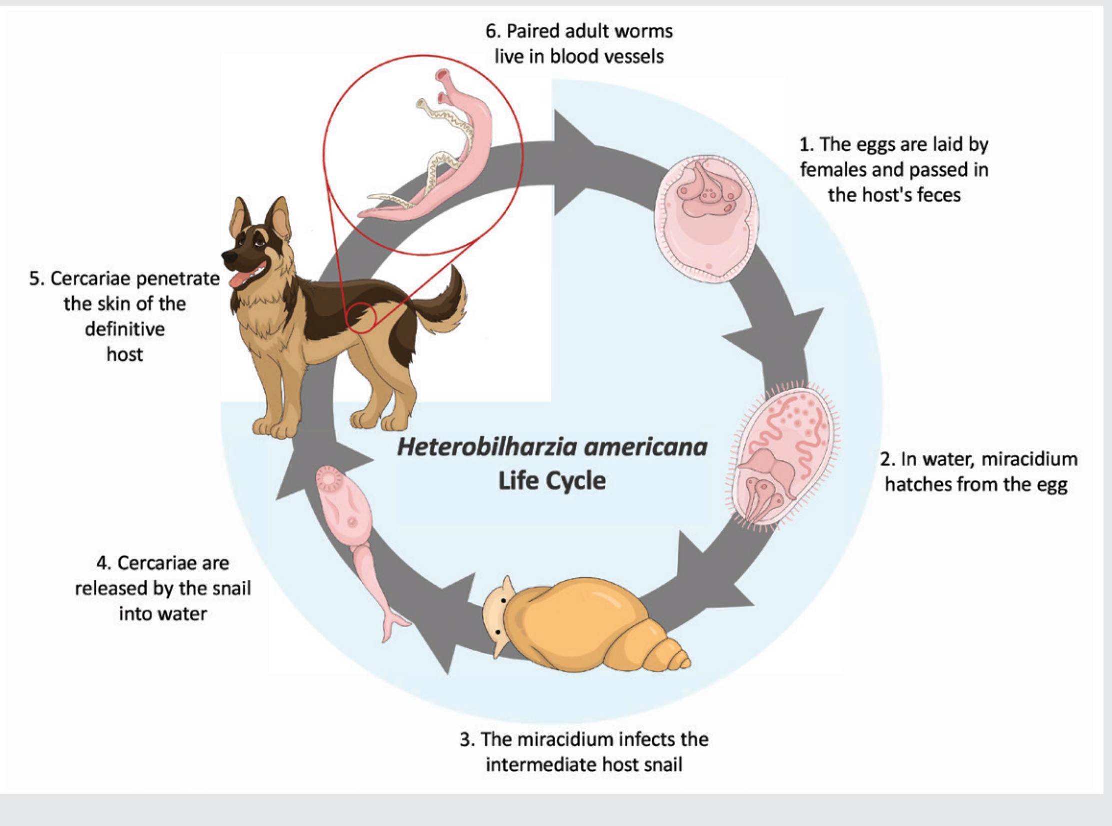

FIGURE 1. Life cycle of *Heterobilharzia americana.*

Even in long-recognized endemic areas, the prevalence of *H americana* remains unknown. This led to prospective prevalence research in Texas that is currently in progress. The observation from a recent study confirming the presence of *H americana* in a snail host in California[2] led to an additional targeted prevalence investigation in states located in the southwestern United States and with access to the Colorado River. Preliminary results of these studies strongly suggest that *H americana* is a parasitic infection of importance in dogs frequenting fresh water in these geographic regions. However, samples from dogs from all over the country have tested positive for *H americana* in the authors' diagnostic laboratory.

CLINICAL MANIFESTATIONS

Canine schistosomiasis can remain asymptomatic or manifest with a range of clinical signs and abnormalities. Schistosomiasis should be considered in dogs presenting with signs of persistent intestinal and liver disease. Frequently observed clinical signs include weight loss, anorexia, lethargy, diarrhea, hematochezia, vomiting, polyuria, and polydipsia.[10,11]

Disease progression is driven by the deposition of fluke eggs in visceral organs, leading to granulomatous inflammatory responses, mineralization, and fibrosis of affected tissues. Eggs can be located in the intestines, liver, pancreas, lymph nodes, and lungs, disrupting organ function and leading to corresponding hematologic and biochemical abnormalities.[9]

Among the key hematologic abnormalities are anemia, which may be regenerative or nonregenerative; lymphopenia; eosinophilia or eosinopenia; and thrombocytopenia. Biochemical changes to look for include decreased albumin and elevated globulins. Dogs frequently present with hypercalcemia, although hypocalcemia has also been observed in dogs with schistosomiasis.[10,11]

To assess the risk of past exposure to *H americana*, the authors encourage clinicians to routinely inquire about their patients' swimming history. Once the infection is confirmed, housemates should be tested as well given their likely similar lifestyle and swimming habits.[12]

DIAGNOSIS

Fecal saline sedimentation, a commercially available PCR assay, and histopathologic examinations of biopsy samples can be used to diagnose canine schistosomiasis.

Fecal Testing

In the authors' experience, fecal saline sedimentation can be a very reliable noninvasive diagnostic test for asymptomatic dogs, which may be shedding fewer eggs or have a lower parasite burden. Intact fluke eggs do not float and therefore cannot be detected by flotation methods. While fecal saline sedimentation does not require elaborate technical equipment, *H americana* eggs come in varying colors, shapes, and sizes, making it challenging for clinicians and laboratory personnel to accurately identify them. *H americana* eggs have a thin shell and can appear clear, golden, or brown, with a round or ovoid shape (**FIGURE 2**). The most significant morphologic feature is the larval miracidium that can be observed within the egg.[13]

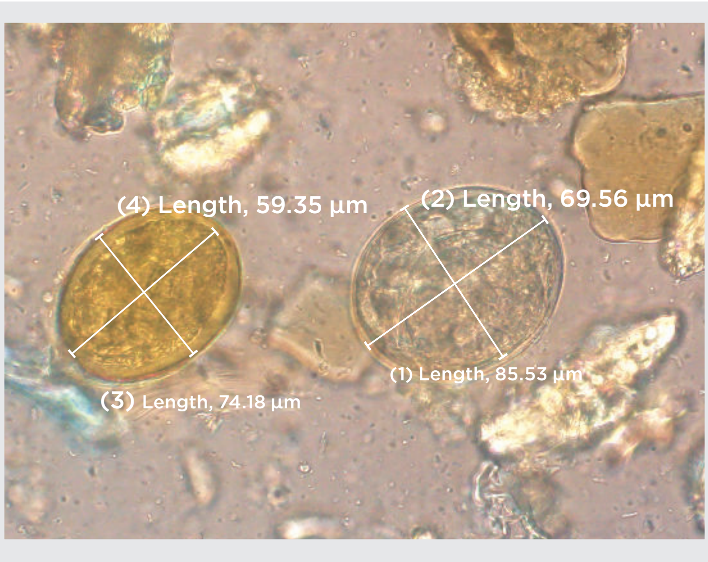

FIGURE 2. Two *Heterobilharzia americana* eggs, each harboring a fully developed miracidium (40× magnification).

A quantitative PCR test for detecting *H americana* in fecal samples is offered by the Gastrointestinal Laboratory at Texas A&M University[14] and performs well in dogs that are symptomatic with *H americana* infection.

The authors are currently evaluating the utility of PCR as a screening test in asymptomatic dogs.

Histopathology

Histopathologic examination of tissue samples is the most reliable, albeit most invasive, method of confirming the presence of *H americana*. Eggs are commonly found infiltrating the small and large intestine, liver, lymph nodes, pancreas, stomach, spleen, and lungs. Eggs are often partially mineralized and surrounded by granulomatous inflammation (**FIGURE 3**).[9,10,15]

FIGURE 3. Two *Heterobilharzia americana* eggs, each harboring a fully developed miracidium (40× magnification).

Diagnostic Imaging

One study demonstrated that certain sonographic findings can serve as highly specific indicators of schistosomiasis and warrant diagnostic testing when observed.[16] These features primarily consist of pinpoint hyperechoic foci in the small intestine, hepatic parenchyma, or mesenteric lymph nodes (**FIGURE 4**). In the small intestine, this aberration is commonly accompanied by an abnormal and heterogenous layering of the intestinal wall. In infected dogs, pinpoint hyperechoic foci may also be found in the large intestine, pancreas, and kidneys. They indicate areas of mineralization that consist of calcified eggs and surrounding granulomatous inflammatory reactions.[15]

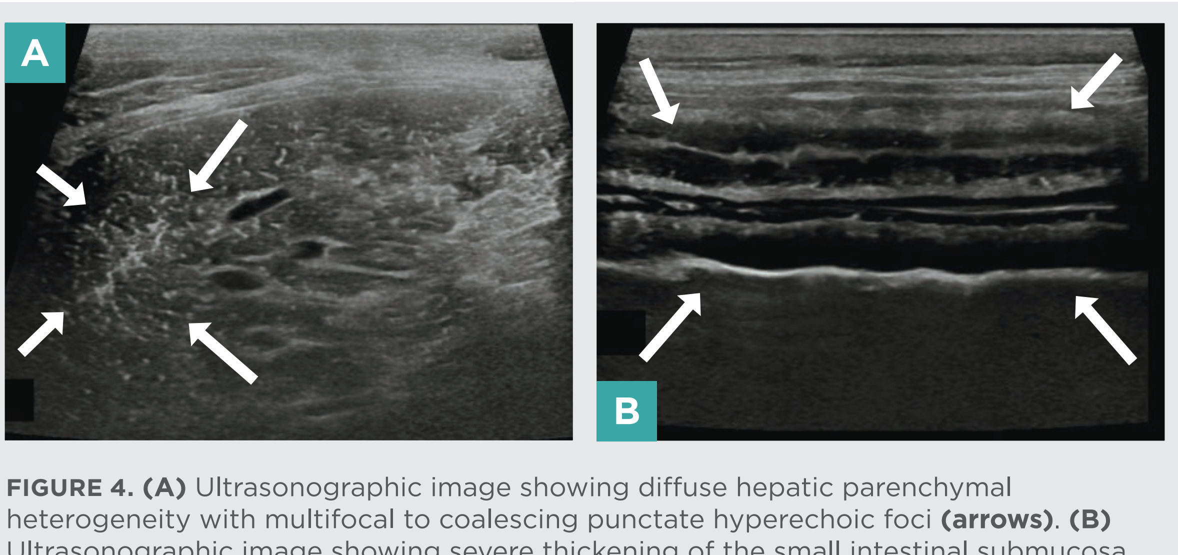

FIGURE 4. (A) Ultrasonographic image showing diffuse hepatic parenchymal heterogeneity with multifocal to coalescing punctate hyperechoic foci **(arrows)**. **(B)** Ultrasonographic image showing severe thickening of the small intestinal submucosa with numerous submucosal and adjacent mucosal punctate hyperechoic foci **(arrows)**.

Abdominal radiographs can further aid in visualizing areas of mineralization. In the small intestine, these can be organized linearly within the intestinal wall (**FIGURE 5**).[17,18] Previous studies have confirmed an enlargement of the liver in schistosomiasis-positive dogs.[18,19] Slight abdominal distention and blurred serosal margins, indicative of peritoneal effusion, have also been observed.[17]

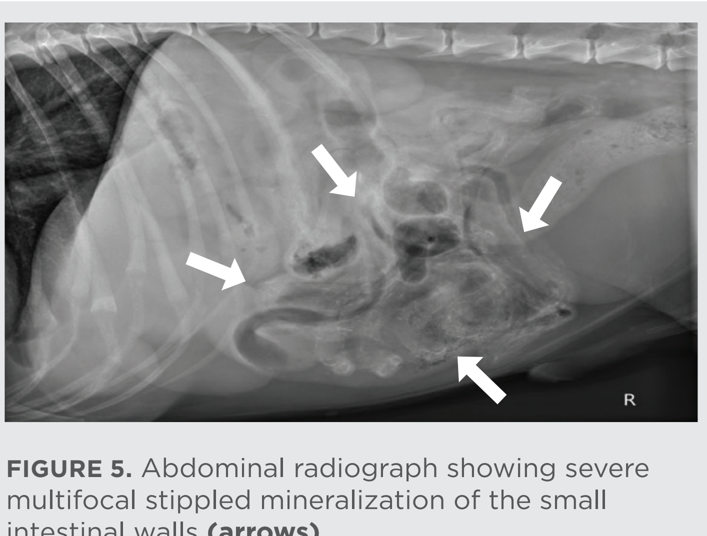

FIGURE 5. Abdominal radiograph showing severe multifocal stippled mineralization of the small intestinal walls **(arrows)**.

Figure 1: courtesy Anil Baniya, PhD, Connor Goldy, Department of Nematology at the University of California, and Jiranun Ardpairin, Department of Microbiology and Parasitology, Faculty of Medical Science, Naresuan University, Phitsanulok, Thailand. Figure 2: courtesy Joe Luksovsky, Diagnostic Parasitology, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University

Diagnostic Testing for Tick-Borne Diseases: Recommendations and Interpretation of Results

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In recent decades, tick-borne diseases in companion animals and humans have emerged as a growing concern throughout North America. After mosquitoes, ticks are considered the most important vectors of pathogens worldwide, including many important bacterial, protozoal, and viral agents.[1] In the United States, 95% of the vector-borne diseases reported annually are vectored by ticks, with dogs acting as sentinels for potential human exposure.[2]

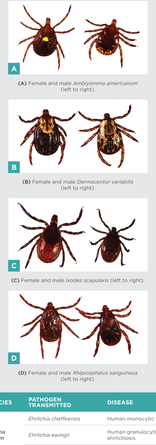
As the range and prevalence of vector tick species continue to expand—aided by climate change and host movement—the need for quick, accurately interpreted diagnostic testing has increased.[3] Many diagnostic tools are available to veterinary clinics in the United States; however, given the complicated nature of tick-borne disease epidemiology and vector species phenology, interpretation of easily accessible diagnostic tests is not without challenges. It is thus important to recognize that no single diagnostic test alone is sufficient to diagnose tick-borne disease. This article aims to provide clinically applicable advice regarding testing procedures and interpretations of common benchtop pathogen tests for tick-borne illness.

VECTORS AND ASSOCIATED PATHOGENS

Many ticks of veterinary and medical importance are frequently found parasitizing domestic dogs and cats in the United States, including species of the genera *Amblyomma*, *Dermacentor*, *Ixodes*, and *Rhipicephalus*.[4] Pathogens of concern have some degree of specificity to vector species, which are in turn associated with preferential host species and environmental conditions, and pathogen–vector–host dynamics are responsible for maintenance of diseases in wildlife reservoirs. Seasonality and geography of vector species are important factors to consider when assessing a patient's risk of tick-borne disease, and species identification of ticks can be an important guide for diagnostic differentials and plans.

Tick-borne pathogens include numerous species of bacteria, protozoa, and viral agents. **BOX 1** features a selection of commonly tested pathogens of veterinary importance and their vectors in North America.[5]

BOX 1 Common Tick Species in the United States and Select Vectored Pathogens



(A) Female and male *Amblyomma americanum* (left to right).

(B) Female and male *Dermacentor variabilis* (left to right).

(C) Female and male *Ixodes scapularis* (left to right).

(D) Female and male *Rhipicephalus sanguineus* (left to right).

TICK SPECIES	PATHOGEN TRANSMITTED	DISEASE
Amblyomma americanum	*Ehrlichia chaffeensis*	Human monocytic ehrlichiosis
	Ehrlichia ewingii	Human granulocytic ehrlichiosis
	Cytauxzoon felis	Cytauxzoonosis
Dermacentor variabilis	*Rickettsia rickettsii*	Rocky Mountain spotted fever
	Francisella tularensis	Tularemia
Ixodes scapularis	*Anaplasma phagocytophilum*	Canine granulocytic anaplasmosis
	Borrelia burgdorferi	Lyme disease
Rhipicephalus sanguineus	*Anaplasma platys*	Canine cyclic thrombocytopenia
	Babesia vogeli	Canine babesiosis
	Rickettsia rickettsii	Rocky Mountain spotted fever
	Ehrlichia canis	Canine ehrlichiosis

Pathogen Transmission Times

Experimental evidence regarding the transmission time for various pathogens is convoluted and often difficult to accurately determine; as an example, transmission times for *Borrelia burgdorferi* have been reported to be between 17 and 72 hours.[6] Differences in transmission time vary according to pathogen, tick species and life stage, environment, and type of host. For example, viruses often require no incubation period within the tick and are transmitted almost instantly with the feeding process, whereas many bacterial species rely on chemical mechanisms associated with tick saliva and host blood during feeding to facilitate transmission into the host.[7] It has been documented that interrupted feeding by ticks can hasten the transmission rate for some pathogens.[8]

When to Test

Surveillance and testing for vector-borne diseases should be year-round.[2] In addition to routine testing of patients that are at risk based on their geographic location, travel history, or lifestyle, testing should be pursued in patients that are symptomatic. Positive serologic results may have different implications for sick patients than for asymptomatic patients, and additional diagnostic modalities may be recommended.

DIAGNOSTIC TESTING MODALITIES

Point-of-Care Testing

The accessibility of point-of-care tests (POCTs) continues to expand, with multiple tests being commercially available.[2,9,10] These tests often require a small amount of blood or serum and provide quick results. **TABLE 1** describes commercially available in-clinic tests, all of which are labeled for use in dogs.

TABLE 1 Point-of-Care Serologic Tests Commercially Available in the United States for Detection of Vector-Borne Diseases

POINT-OF-CARE TEST	PATHOGENS DETECTED	ANALYTES
SNAP 4Dx Plus Test (IDEXX, idexx.com)	*Anaplasma* species	Antibodies against *Anaplasma phagocytophilum* and *Anaplasma platys*
	Borrelia burgdorferi	Antibodies against C_6 peptide
	Dirofilaria immitis	Adult female heartworm antigen
	Ehrlichia species	Antibodies against *Ehrlichia canis* and *Ehrlichia ewingii*
Vetscan Flexé Rapid Test (Zoetis, zoetisus.com)	*Anaplasma* species	Antibodies against *Anaplasma phagocytophilum* and *Anaplasma platys*
	Borrelia burgdorferi	Antibodies against OspC, flagellin, and VlsE peptides
	Ehrlichia species	Antibodies against *Ehrlichia canis*, *Ehrlichia chaffeensis*, and *Ehrlichia ewingii*
	Dirofilaria immitis	Adult female heartworm antigen

OspC= outer surface protein C; VlsE= variable major protein–like sequence, expressed

Except for *Dirofilaria immitis* antigen tests, most serologic POCTs for vector-borne pathogens detect antibodies, which often indicates previous exposure rather than clinical infection (**FIGURE 1 AND BOX 2**). Correct interpretation according to history and clinical signs is crucial and can help discern various stages of infection.[11]

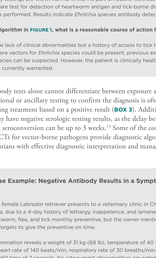


FIGURE 1. Interpretation of possible outcomes of point-of-care test results for *Anaplasma* species and *Ehrlichia* species in dogs. *PCR=polymerase chain reaction*

BOX 2 Case Example: Positive *Ehrlichia* Result in a Healthy Dog

A 5-year-old male castrated German shorthaired pointer presents to a veterinary clinic in central Oklahoma for an annual wellness visit. The dog has a history of hunting with the owner and spends a lot of time roaming wooded areas during hunting season. The owner reports no concerns about the patient's health. Physical examination and routine blood testing (complete blood count and serum biochemistry testing) are within normal limits. As part of routine wellness care, a point-of-care test for detection of heartworm antigen and tick-borne disease antibodies is performed. Results indicate *Ehrlichia* species antibody detection.

Using the algorithm in FIGURE 1, what is a reasonable course of action for this patient?

Based on the lack of clinical abnormalities but a history of access to tick habitats in a region where vectors for *Ehrlichia* species could be present, previous exposure to *Ehrlichia* species can be suspected. However, the patient is clinically healthy and no treatment is currently warranted.

Because antibody tests alone cannot differentiate between exposure and active disease, additional or ancillary testing to confirm the diagnosis is often required before initiating treatment based on a positive result (**BOX 3**). Additionally, acute infections may have negative serologic testing results, as the delay between infection and seroconversion can be up to 3 weeks.[12] Some of the commercially available POCTs for vector-borne pathogens provide diagnostic algorithms to assist veterinarians with effective diagnostic interpretation and management plans.

BOX 3 Case Example: Negative Antibody Results in a Symptomatic Dog

A 1-year-old female Labrador retriever presents to a veterinary clinic in Chester, Pennsylvania, due to a 4-day history of lethargy, inappetence, and lameness. The dog is on a heartworm, flea, and tick monthly preventive, but the owner mentions that sometimes forgets to give the preventive on time.

Physical examination reveals a weight of 31 kg (68 lb), temperature of 40 °C (103.4 °F), heart rate of 140 beats/min, respiratory rate of 30 breaths/min, and a capillary refill time of 2 seconds. No integument abnormalities are noted, but the patient is painful on palpation of hips and elbows. Laboratory testing reveals complete blood count numbers within reference ranges except for a platelet count of 67,000 cells/µL. Serum biochemistry analysis reveals mild hypokalemia and hypoalbuminemia. A point-of-care test to detect tick-borne pathogens is performed, but no antibodies are detected.

Using the algorithm in FIGURE 1, what is a reasonable course of action for this patient?

Although no antibodies were detected for tick-borne pathogens, this patient's history and clinical findings indicate the possibility of an early tick-borne infection. Treatment with doxycycline can be initiated and a whole blood sample submitted for a tick-borne pathogens polymerase chain reaction panel.

Reference Laboratory Testing

Diagnostic reference laboratories can perform additional serologic or molecular testing for tick-borne diseases. Sample submission to referral laboratories is recommended when POCT results need to be confirmed or when conflicting results have been obtained in-clinic.

Compared with POCTs, the advantage of reference laboratory testing is the use of diagnostic methods with increased specificity and sensitivity, such as:

- **Molecular testing**, including polymerase chain reaction (PCR) testing, quantitative PCR testing, and next-generation sequencing (can be useful to confirm an active infection detected by an immunochromatographic lateral flow test previously performed in-clinic)
- **Microimmunofluorescence assay**, which can detect immunoglobulin M and immunoglobulin G antibodies and evaluate seroconversion using acute and convalescent serum samples
- **Enzyme-linked immunosorbent assay** (ELISA)
- **Immunofluorescent assay**, which can detect antigens or antibodies

Waiting time and additional cost may be disadvantages.

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