

**STOP IT AT THE SOURCE**  
 Because cytauxzoonosis is associated with a high mortality rate and costly treatment for domestic cats, tick prevention is critical.

PARASITOLOGY

# Cytauxzoonosis

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Cytauxzoonosis, caused by the hematoprotzoan pathogen *Cytauxzoon felis*, is a devastating illness of domestic cats. However, although this illness was once thought to be uniformly fatal for domestic cats, we now recognize that domestic cats can survive infection and serve as reservoirs for pathogen spread via the tick vectors, as occurs with the wild felid reservoir hosts. Nonetheless, infection of domestic cats most often leads to an acute illness that is costly to treat and associated with a high mortality rate.

## TRANSMISSION

Much of the *C. felis* life cycle is inferred from what we know of related Piroplasmidae such as *Theileria*.

Experimental studies are made more difficult because, thus far, the pathogen has not been cultured in vitro. *C. felis* infection of the wild felid reservoir hosts (e.g., bobcats) typically causes mild to moderate illness with a low mortality rate.<sup>1</sup> Reservoir hosts maintain chronic erythroparasitemia that permits transmission of the pathogen to tick vectors. Survivors remain infected with *C. felis* but no longer have cytauxzoonosis (TABLE 1).

In the United States, the predominant tick vector is the lone star tick, *Amblyomma americanum*, but other ticks might also transmit infection.<sup>2</sup> While feeding on an infected reservoir host, the naive tick ingests organisms that then undergo sexual reproduction in the tick

**TABLE 1** Cytauxzoonosis (Acute Illness) Versus Chronic *Cytauxzoon felis* Infection

CYTAUXZOOONOSIS	CHRONIC <i>C. FELIS</i> INFECTION
Acute illness	Inapparent carrier
High morbidity/mortality in domestic cats	Uncommon in domestic cats
Low morbidity/mortality in wild felids	Common in wild reservoir felids
Tissue schizonts characteristic	Tissue schizonts absent
Schizonts rarely seen on blood smear	Schizonts never identified
Red blood cell piroplasms often numerous	Red blood cell piroplasms rare
Polymerase chain reaction positive	Polymerase chain reaction positive

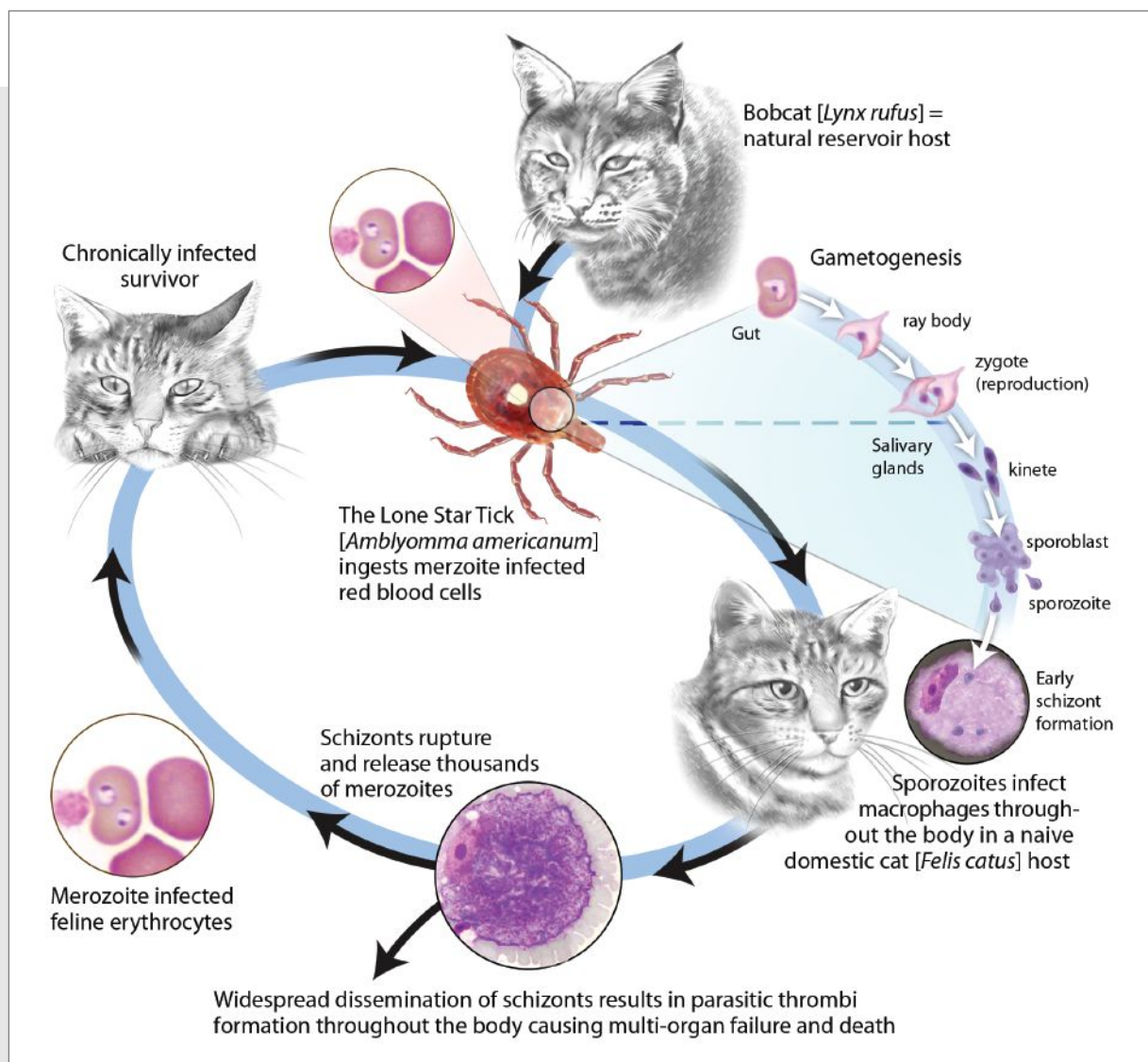


(FIGURE 1). The parasite persists in the tick as it transitions from one life stage to the next (transstadial transmission). During the tick's next blood meal, *C. felis* zygotes move into the tick's salivary glands, where they mature into infective sporozoites. In as few as 36 hours after the tick next feeds on a naive host, sporozoites enter the host's mononuclear cells, where they undergo schizogony, a form of asexual reproduction.<sup>4</sup> Schizont-laden mononuclear cells are characteristic of the acute disease phase of infection (i.e., cytauxzoonosis).

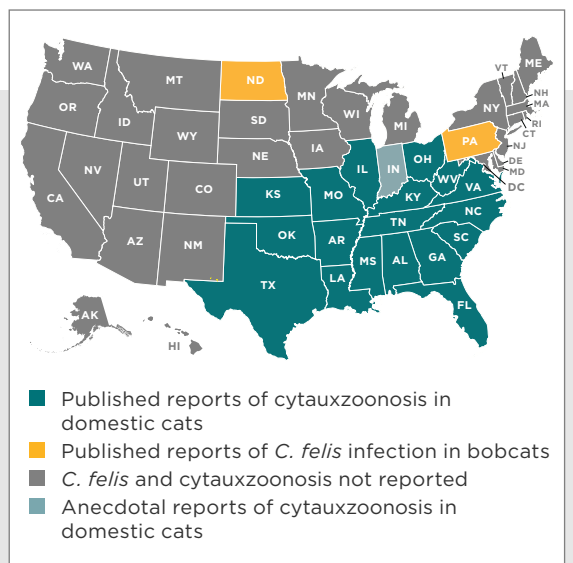
If the infected cat survives long enough, merozoites are released from ruptured macrophages and are taken up

by red blood cells (RBCs), where they can be visualized as piroplasms. At the time of disease diagnosis, most cats have both identifiable tissue schizonts and RBC piroplasms. In surviving cats (wild or, less commonly, domestic), asexual reproduction comes to a halt in the mononuclear cells but continues in the RBCs. For months to years, carrier cats maintain a low-level parasitemia, which facilitates parasite transmission to feeding ticks.

Of note, no evidence of naturally occurring cat-to-cat transmission in the absence of a tick vector has been found. Although perinatal transmission of closely related *Theileria* and *Babesia* species is well



**FIGURE 1.** Life cycle of *Cytauxzoon felis*. The acute schizogony phase is typically fatal in the domestic cat. Hosts that survive develop a chronic erythrocytosis with merozoite-infected red blood cells, which are the basis of cytologic screening.



**FIGURE 2.** U.S. regions with recognized cytauxzoonosis and/or *Cytauxzoon felis* infections.

documented, perinatal transmission of *C. felis* has not been demonstrated.<sup>5</sup> Ingestion of infected ticks does not result in cytauxzoonosis.<sup>4</sup> Transfusion of blood from recovered cats permits erythroparasitemia but does not result in clinical illness.<sup>6</sup> Experimentally, disease can be caused by parenteral administration of schizont-laden mononuclear cells from tissues (i.e., spleen) or blood collected from cats during the period of clinical illness.<sup>7</sup>

## GEOGRAPHIC INCIDENCE AND PREVALENCE

First recognized in Missouri in 1976, cytauxzoonosis was believed to be limited to the south-central United States for many years thereafter.<sup>8</sup> More recently, it has dramatically expanded geographically within the United States, and the same or similar organisms have been recognized in South America, Europe, and Asia. Expansion in the United States probably corresponds to

an expanding territory for the lone star tick vector. The disease has now been recognized in most states in the south-central, southeastern, and mid-Atlantic United States (FIGURE 2).<sup>9,10</sup> In most of the United States, survival of untreated domestic cats is rare; however, there are geographic pockets (e.g., northwestern Arkansas) where disease in domestic cats can be mild or inapparent.<sup>11,12</sup> The cause for virulence differences in various geographic regions remains unexplained.

The incidence and prevalence of *C. felis* depend on geographic location. Because cytauxzoonosis is not reportable, and many cats probably die without having received veterinary care, estimating disease incidence is extremely difficult. The authors have personal knowledge of rural and suburban private veterinary practices in cytauxzoonosis-endemic regions that diagnose cytauxzoonosis in 20 to 40 cats each summer, while practices only 100 miles away rarely make that diagnosis. Cytauxzoonosis was the reason for 1.5% of all cat admissions to the Boren Veterinary Medical Teaching Hospital at Oklahoma State University from 1998 through 2006.<sup>13</sup> Prevalence would be low in areas where incidence of infection is low or if most infected cats quickly succumb. Prevalence among apparently healthy domestic cats from *C. felis*-endemic regions has been reported to range from zero to 15.5%; prevalence was higher in the region of Arkansas reported to have less virulent disease.<sup>12,14,15</sup> Among wild cats, the reported prevalence in endemic regions is up to 79%.<sup>10</sup>

## HISTORY AND PHYSICAL EXAMINATION

Clinical signs of cytauxzoonosis usually become apparent 8 to 12 days after infection.<sup>2</sup> Macrophages become distended with schizonts, leading to variable degrees of vessel occlusion in the liver, spleen, pulmonary parenchyma, and central nervous system. As schizonts rupture, the liberated merozoites enter RBCs, leading to immune-mediated destruction of the

**TABLE 2** Abnormal Physical Examination Findings at Presentation for Cats with Cytauxzoonosis

MOST COMMON	COMMON	LESS COMMON
<ul style="list-style-type: none"> <li>■ Fever (90% of cats)</li> <li>■ Lethargy/depression</li> <li>■ Icterus</li> <li>■ Elevated nictitating membranes</li> <li>■ Tachypnea</li> <li>■ Dehydration</li> </ul>	<ul style="list-style-type: none"> <li>■ Tachycardia</li> <li>■ Attached ticks</li> <li>■ Pallor</li> <li>■ Vocalization</li> <li>■ Discomfort on abdominal palpation</li> <li>■ Hepato/splenomegaly</li> <li>■ Lymphadenomegaly</li> </ul>	<ul style="list-style-type: none"> <li>■ Murmur and/or gallop heart rhythm</li> <li>■ Ataxia</li> <li>■ Hemorrhagic panuveitis</li> <li>■ Seizures</li> <li>■ Obtundation/coma</li> <li>■ Petechia</li> <li>■ Hypothermia (poor prognostic indicator)</li> <li>■ Respiratory distress</li> </ul>



infected cells and subsequent anemia. Although no history or examination finding is pathognomonic for cytauxzoonosis, there are typical characteristic features.

Cats of any age, breed, or sex can be infected, but most commonly affected are active adult cats that spend time outdoors in endemic rural or semirural areas.<sup>13</sup> In most areas, disease incidence peaks in early spring, continues throughout the summer, and peaks again (smaller peak) in early autumn.<sup>13</sup> Although infection is more likely in cats not receiving ectoparasite prophylaxis, inappropriate choice of product or mistakes in application might allow for infection despite the client's reported use of ectoparasite control. Often, clients will note that their previously healthy cat suddenly demonstrates reduced appetite, followed rapidly by anorexia, lethargy, and sometimes vocalization, interpreted as pain or discomfort.

The most consistent abnormality detected by physical examination of an acutely infected cat is pronounced fever.<sup>7,16,17</sup> Although a large majority of infected cats are febrile at presentation, hypothermia can occur late in the course of disease and is a very poor prognostic sign. Other findings may include icterus, pallor, and organomegaly (TABLE 2). The entire disease course progresses rapidly, and death within 36 hours of presentation for veterinary care is common.

## DIAGNOSTIC TESTING

Confirmation of cytauxzoonosis depends on identification of the parasite microscopically or via molecular methods in combination with typical clinical findings. A number of other diagnostic tests might be

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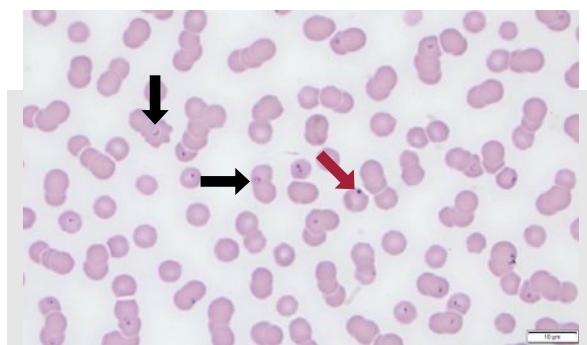
indicated to help identify comorbid conditions or disease complications that may require intervention. For example, tachypnea and respiratory distress are not uncommon complications, resulting from either pulmonary vascular obstruction with schizont-distended cells or from pleural fluid accumulation. When respiratory signs are present, thoracic radiographs or thoracic ultrasonography may be helpful. Abdominal imaging often confirms organomegaly but seldom alters therapy.

Although cytauxzoonosis results in a number of abnormalities commonly recognized on routine blood and urine tests (TABLE 3), the only specific finding is the pathogen.<sup>7,16,17</sup> Serum biochemical findings often include hyperbilirubinemia, hyperglycemia, and hypoproteinemia; occasionally, electrolyte derangements such as hypokalemia require intervention. Coagulation tests often reflect disseminated intravascular coagulation (DIC).<sup>18</sup> Anemia may not be present initially, but as merozoites invade RBCs, hemolysis results and might warrant transfusion. Pancytopenia is common, and lymphocytes sometimes appear large and granular.

**TABLE 3** Abnormal Laboratory Findings for Cats with Cytauxzoonosis

PARAMETER	COMMON	LESS COMMON
<b>Complete blood count</b>	<ul style="list-style-type: none"> <li>■ Pancytopenia</li> <li>■ Leukopenia</li> <li>■ Thrombocytopenia (moderate to severe)</li> <li>■ Preregenerative or regenerative anemia</li> <li>■ Red blood cell piroplasms</li> </ul>	<ul style="list-style-type: none"> <li>■ Neutrophilia +/- left shift and toxic changes</li> <li>■ Mononuclear schizonts (rare but pathognomonic)</li> </ul>
<b>Chemistry</b>	<ul style="list-style-type: none"> <li>■ Hyperbilirubinemia</li> <li>■ Hyperglycemia</li> </ul>	<ul style="list-style-type: none"> <li>■ Hypoalbuminemia/hypoproteinemia</li> <li>■ Electrolyte derangements               <ul style="list-style-type: none"> <li>■ Hypokalemia</li> <li>■ Hyponatremia</li> <li>■ Hypocalcemia</li> </ul> </li> <li>■ Hyperlactatemia</li> <li>■ Elevated liver enzymes (mild)</li> </ul>
<b>Urinalysis</b>	<ul style="list-style-type: none"> <li>■ Bilirubinuria</li> </ul>	<ul style="list-style-type: none"> <li>■ Proteinuria</li> </ul>
<b>Coagulation</b>	<ul style="list-style-type: none"> <li>■ Prolonged coagulation times (activated partial thromboplastin time and prothrombin time)</li> </ul>	<ul style="list-style-type: none"> <li>■ Increased fibrin degradation products</li> </ul>

The most useful diagnostic method is identification of organisms on Wright- or Wright-Giemsa-stained blood smears, but this method is not perfectly sensitive or specific. Intraerythrocytic merozoites (i.e., piroplasms) most commonly appear as signet ring-shaped inclusions 1 to 2 μm in diameter but may appear as tetrads, bipolar oval structures, or round anaplasmod bodies ranging from 0.2 to 2.5 μm (FIGURE 3). The organisms are indistinguishable from *Babesia felis*, a pathogen not yet identified in the United States. Although piroplasms are often numerous during clinical disease (up to 50% of RBCs may contain parasites), they may be absent at the onset of clinical signs. In these very early infections, piroplasms can become apparent on re-examination of a new blood smear even just 12 hours later. Note that occasional piroplasms remain identifiable in recovered carrier cats;<sup>11,19</sup> thus, identification of piroplasms can demonstrate infection but does not confirm acute disease (i.e., cytauxzoonosis) in the absence of compatible clinical signs. Rarely, schizont-laden mononuclear cells (FIGURE 4) are seen on a peripheral blood smear. Unlike piroplasms, visualization of these cells is confirmatory for the disease because schizonts are not present in chronically infected carriers.<sup>20</sup> Although identifying schizonts on a blood smear is rare, identification of schizonts in tissues is both common and diagnostic for cytauxzoonosis. Fine-needle aspirates from spleen, lymph nodes, liver, or lungs might demonstrate schizonts before piroplasms are found on a blood smear, but obtaining aspirates from hypocoagulable patients may lead to hemorrhage.



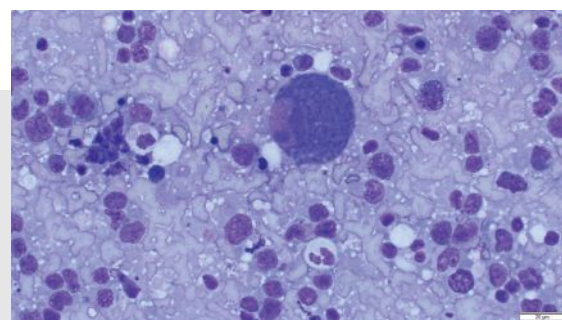
**FIGURE 3.** Blood film from a cat with cytauxzoonosis (magnification ×100, modified Wright-Giemsa stain). Many intraerythrocytic *C. felis* merozoites, approximately 2 to 3 μm in diameter, can be seen (black arrows). The merozoites are often observed in the classic signet ring shape with a small (<1 μm), peripherally placed, deep purple nucleus and pale blue cytoplasm extending in crescent formation away from the nucleus. The red arrow denotes a Howell-Jolly body on the surface of a red blood cell for size comparison.

Schizonts are readily identifiable on tissue biopsy samples (rarely performed) or necropsy, making cytauxzoonosis a very straightforward postmortem diagnosis (FIGURE 5).<sup>21</sup>

Molecular testing for *C. felis* infection is commercially available and extremely sensitive; in experimentally infected cats, positive results precede clinical disease and recognition of organisms on blood smear by up to several days.<sup>22,23</sup> Because polymerase chain reaction (PCR) is so sensitive, results can remain positive in recovered carrier cats.<sup>24</sup> Thus, as for piroplasm identification, a positive PCR does not confirm a disease diagnosis in the absence of clinical findings. Serologic testing is not commercially available.

## TREATMENT AND PROGNOSIS

There is no simple or inexpensive cure for cytauxzoonosis. Over the years, numerous antiprotozoal drugs have been investigated and were largely found to have little efficacy. The current treatment of choice is oral administration of the antimalarial drug atovaquone (Mepron; GlaxoSmithKline, [gsk.com](http://gsk.com)) and the antimicrobial drug azithromycin (Zithromax; Pfizer, [pfizer.com](http://pfizer.com)) combined with supportive care (BOX 1).<sup>16</sup> Although without treatment the mortality rate is nearly 100%, with appropriate treatment the rate falls to 40%. Atovaquone works by targeting cytochrome b; treatment failures might be explained by mutations in cytochrome b in some parasites because different parasite cytochrome b genotypes resulted in different



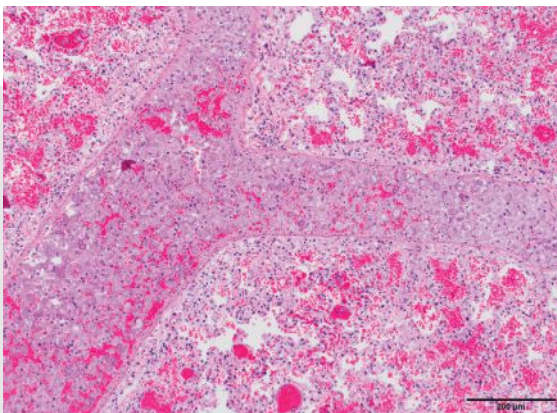
**FIGURE 4.** Fine-needle aspirate from the spleen of a cat with cytauxzoonosis (magnification ×100, modified Wright-Giemsa stain). A single schizont-laden macrophage can be seen in the center of the image. This macrophage is approximately 60 μm in diameter with the displacement of a swollen, reniform nucleus peripherally. Within the cytoplasm are 3 distinct packets containing large numbers of small (<1 μm), deep purple, *Cytauxzoon* merozoites. These schizont-laden macrophages can range from 15 to 80 μm, depending on the phase of parasite development.

Courtesy Dr. Erin Burton, DVM, MS, DACVP, University of Minnesota (2).  
Opposite: Courtesy Dr. Gayle Johnson, DVM, MS, MPVM, PhD, DACVP, University of Missouri.



treatment outcomes.<sup>25</sup> Despite improved survival rates with this combination therapy, the treatment is expensive and difficult to administer. Atovaquone is a viscous liquid; the authors advise that after filling the syringe, its contents be allowed to settle to ensure that the full volume is present. Cats dislike the flavor of the medication, which is given every 8 hours. For this reason, we advise early placement of a nasoesophageal feeding tube to allow for less stressful administration of medication as well as nutritional support. Although no studies have documented the useful aspects of supportive care, in the authors' opinion, minimizing stress seems to be key to survival. Judicious use of crystalloid fluids and administration of analgesics are probably beneficial. The authors avoid nonsteroidal fever-reducing agents because we have the impression that they lead to a worsened outcome in treated cats; instead, focus is placed on analgesia by using agents such as buprenorphine. Some clinicians advocate the use of anti-inflammatory prednisolone, but there is no evidence that this practice is either beneficial or detrimental. Although the authors have administered heparin for DIC prophylaxis/treatment, no benefit has been documented. Other forms of support may be indicated on a case-by-case basis and include transfusion for anemia, oxygen therapy for pneumonitis, thoracocentesis for pleural effusion, or anticonvulsants for seizures.

Chronic carriers of *C. felis* are not ill and do not require specific therapy. Several treatments have been investigated to determine if the carrier state, and thus



**FIGURE 5.** Pulmonary parenchyma from a cat that died of cytauxzoonosis (hematoxylin and eosin stain). A bifurcating pulmonary vessel is seen in the center of the image. The vessel is occluded by large numbers of merozoite-laden macrophages that give a granular appearance to the cytoplasm.

### BOX 1 Treatment for Cytauxzoonosis

#### Antiprotozoal therapy

- Atovaquone 15 mg/kg PO q8h for 10 days
- Azithromycin 10 mg/kg PO q24h for 10 days

#### Supportive care for most patients

- Minimize stress and handling
  - Nasoesophageal/nasogastric feeding tube (for ease of medication and nutritional support administration)
- Analgesia (e.g., buprenorphine; authors avoid nonsteroidal anti-inflammatory drugs)
- Judicious use of crystalloid fluids
- Correction of electrolyte abnormalities
- +/- Heparin (300 IU/kg SQ q8h)

#### Supportive care for some patients (should be tailored to individual patient's needs)

- Packed RBC or whole blood transfusion
- Appetite stimulant (e.g., mirtazapine 2 mg/cat transdermally q24h)
- Antiemetics (e.g., maropitant citrate 1 mg/kg PO or SQ q24h)
- Oxygen supplementation
- Therapeutic thoracocentesis
- Anticonvulsants
- Fresh or frozen plasma

the risk of acting as a reservoir host, can be eliminated. Atovaquone and azithromycin dramatically reduced pathogen burden but did not completely eliminate infection.<sup>26</sup> Neither diminazene diaceturate nor imidocarb dipropionate has been able to eliminate the pathogen.<sup>26,27</sup>

## PREVENTION

Currently, cytauxzoonosis is best prevented by keeping ticks from feeding on cats. Keeping cats indoors is helpful, but because ticks can come indoors and because indoor cats occasionally escape to the outdoors, tick prevention in cytauxzoonosis-endemic areas is strongly recommended. High efficacy for preventing *C. felis* infection has been shown by the use of collars containing imidacloprid 10%/flumethrin 4.5% (Seresto; Bayer, [animalhealth.bayer.com](http://animalhealth.bayer.com)) or topical application of selamectin plus sarolaner (Revolution Plus; Zoetis, [zoetis.com](http://zoetis.com)).<sup>28,29</sup> Recovered cats should be kept indoors and tick-free to prevent them from serving as a reservoir for infection. Although reinfection has previously been deemed unlikely, a second clinical disease episode was recently documented in a cat 7 years after the first illness.<sup>20</sup> Although candidate targets

for vaccination have been identified, vaccination has thus far been ineffective.<sup>3,30</sup> **TVP**

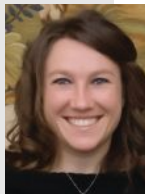
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Dr. Pacholec obtained her DVM degree at Kansas State University and then completed a rotating internship at Auburn University College of Veterinary Medicine, followed by an Internal Medicine residency at the University of Missouri. In alignment with her emerging clinical interest, she started a combined Clinical Pathology residency and PhD program at Virginia Tech in the summer of 2020. Her clinical and research interests include immune-mediated diseases, pharmacology, infectious diseases, and clinical pathology.



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