TRENDS IN AGRICULTURE SCIENCE
ISSN:2583-7850

Popular Article
Domain: Veterinary Science
Vol 4 Issue 12, December 2025, 6000-6008

Hemoparasites in cats: An update on Hepatozoon, Cytauxzoon and Babesia

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DOI:10.5281/trendsinAgri.17848464

Abstract

Feline vector-borne diseases (VBDs) are increasingly recognized as major health threats worldwide, driven by the expanding domestic cat population and the broader distribution of vector arthropods. These infections, caused by haemoparasites such as *Hepatozoon*, *Cytauxzoon*, and *Babesia* species, often present with nonspecific clinical signs or remain subclinical, making early diagnosis challenging. *Hepatozoon felis* is the most commonly reported species in domestic cats, transmitted primarily through ingestion of infected arthropods, and may cause lethargy, fever, icterus, and muscle involvement. *Cytauxzoon felis*, transmitted by ixodid ticks, induces acute and frequently fatal disease characterized by anaemia, hepatosplenomegaly, and multi-organ dysfunction, with chronic carriers posing ongoing infection risks. Feline babesiosis, caused by both small and large *Babesia* species, results in anaemia, icterus, and variable disease severity, influenced by host immunity and concurrent infections. Diagnosis relies on microscopic examination of blood smears, complemented by molecular techniques such as polymerase chain reaction for enhanced sensitivity and accurate species identification. Management strategies include targeted antiprotozoal therapy alongside intensive supportive care, while preventive measures focus on limiting vector exposure. Improved understanding of the epidemiology, clinical manifestations, diagnostics, and treatment of feline VBDs is essential for early intervention, effective therapy, and the development of preventive strategies to safeguard feline health globally.

Introduction:

Feline vector-borne diseases (VBDs) are increasingly recognised as a growing health threat to cats worldwide, with their prevalence rising in parallel to the rapid expansion of the global domestic cat population. Despite their significance, knowledge of VBDs in cats remains limited due to constantly changing epidemiological patterns, wider geographical spread, and increasing global incidence driven by factors such as climate change, environmental alterations, demographic shifts, and human behaviour. Many vector-borne

pathogens are capable of causing severe illness or death in infected cats, yet clinical identification remains difficult because symptoms are often non-specific or completely absent, allowing infections to persist even in apparently healthy animals. Additional complications arise from co-infections involving haemoparasites, retroviruses, or haemotropic mycoplasmas, which can result in immunosuppression and exacerbate disease outcomes. Although light microscopy remains a common technique for parasite identification, advanced molecular diagnostic tools such as polymerase chain reaction (PCR) and DNA sequencing have greatly improved diagnostic accuracy and epidemiological understanding. Comprehensive data on the distribution and occurrence of haemoparasites in cats are essential for early diagnosis, timely treatment, and the development of efficient disease-control strategies. Cats with outdoor access are particularly vulnerable to these infections due to increased exposure to ectoparasite vectors that transmit disease-causing agents.

FELINE HEPATOZOONOSIS

The genus Hepatozoon comprises Apicomplexan parasites belonging to the family Hepatozoidae, with more than 340 species currently identified in a wide range of hosts, including mammals, birds, reptiles and amphibians. Feline hepatozoonosis was first documented in India in 1908, after which the disease has been reported in domestic cats from multiple regions such as South Africa, Asia, southern Europe, and the United States. Despite these findings, the precise taxonomy of the Hepatozoon species infecting cats, along with their pathogenic potential and the vectors responsible for transmission, has not been fully elucidated. To date, three species, Hepatozoon felis, Hepatozoon canis, and Hepatozoon silvestris, have been described in association with domestic cats, among which H. felis is recognised as the most frequently detected species in feline hepatozoonosis cases in India.

Transmission:

Blood-feeding arthropods serve as the definitive hosts for *Hepatozoon* species; however, the specific arthropod vectors responsible for feline infections have not yet been clearly confirmed. Based on current evidence, ticks are believed to act as the vectors for *H. felis* and *H. silvestris*. Within these arthropods, the parasites undergo sexual reproduction and sporogony, and transmission to vertebrate hosts occurs primarily through the ingestion of the arthropod—or parts of it—containing mature oocysts. In addition to this classical route, transplacental transmission and predation have recently been documented. Inside the vertebrate host, the parasite continues its development, with merogony and gametogony occurring in host tissues, and the resulting gamonts typically appearing in leukocytes, especially neutrophils. Moreover, the presence of small tissue cysts harbouring single or multiple parasitic stages (cystozoites) has been reported in several *Hepatozoon* species,

further emphasizing the complexity and diversity of their life cycles.

Clinical signs:

Feline hepatozoonosis is related to infection of muscle tissues. Meronts of Hepatozoon are recognized in the myocardium and skeletal muscles of domestic cats with hepatozoonosis. In felines, clinical hepatozoonosis is rare and poorly understood. The affected cats may present with lethargy, icterus, fever, anorexia, and ruffled hair. In affected felines, laboratory findings may include leukopenia, thrombocytopenia, and Elevated muscle enzyme creatinine kinase (CK). and elevated serum concentrations of bilirubin and symmetric dimethylarginine (SDMA), indicating possible hepatic and renal involvement. Although animals with hepatozoonosis more frequently exhibit elevated total protein levels as well as polyclonal hyperglobulinemia and hypoalbuminemia, both conditions can result in elevated liver enzyme levels.

Diagnosis:

Microscopic examination of blood smears has limited sensitivity for detecting *Hepatozoon* gamonts because parasitaemia is typically very low, with less than one percent of neutrophils and monocytes containing visible parasites. In feline infections, *Hepatozoon felis* gamonts are most often observed within the cytoplasm of neutrophils and monocytes, sometimes causing compression of the host cell's lobulated nucleus. Compared with *H. canis* gamonts, which measure approximately 11 µm in length, *H. felis* gamonts are slightly shorter, averaging around 10.5 µm. The meronts of *H. felis* are located primarily in muscle tissues, especially the myocardium and skeletal muscles, and, unlike the characteristic wheel-spoke appearance of *H. canis* meronts, they do not form that structure. Instead, *H. felis* meronts contain 10–15 micromerozoites that are larger, rectangular to triangular in shape, and positioned perpendicularly to the meront wall. In contrast, *H. silvestris* meronts display a wheel-spoke configuration with a thin capsule and include 20–30 small, round-to-oval micromerozoites. Because of the low sensitivity of blood smears, polymerase chain reaction (PCR) is currently considered a superior diagnostic method for *Hepatozoon* infection in cats, with the most commonly used assay employing Piroplasmid-F and Piroplasmid-R primers to amplify part of the 18S rRNA gene of *Hepatozoon* spp. Comprehensive characterisation of feline *Hepatozoon* infections requires the integration of both morphological and molecular findings, supported by large-scale epidemiological studies.

Treatment:

At present, there is no universally established treatment protocol for feline hepatozoonosis; however, individual case reports have demonstrated successful management using doxycycline or oxytetracycline in combination with primaquine. A more effective

therapeutic approach reported in domestic cats involves the administration of imidocarb dipropionate at a dosage of 6 mg/kg body weight subcutaneously for two doses given 14 days apart, together with doxycycline monohydrate at 5 mg/kg body weight orally twice daily for four weeks. Despite the lack of definitive evidence regarding transmission routes and specific vectors, preventive control measures targeting blood-feeding ectoparasites such as ticks and fleas are strongly recommended to reduce the risk of infection.

FELINE CYTAUXZOONOSIS

Cytauxzoonosis is an emerging tick-borne disease affecting both domestic and exotic felids, caused by haemoparasites of the genus *Cytauxzoon*, which belong to the family Theileriidae within the phylum Apicomplexa. Multiple species of *Cytauxzoon* have been identified, among which *Cytauxzoon felis* is considered the most clinically significant. The parasite was first described in Missouri in 1973, and infection in domestic cats is associated with an extremely high fatality rate. In contrast, bobcats (*Lynx rufus*) act as the primary reservoir hosts for *C. felis*, allowing the parasite to persist in nature while exhibiting no overt clinical disease.

Transmission:

Cytauxzoon felis exhibits a complex lifecycle consisting of an asexual developmental phase in a felid host and a sexual reproductive phase within a competent ixodid tick vector. Transmission occurs when ticks feed on parasitaemic hosts and subsequently transmit the parasite to new felid hosts during blood meals. The lone star tick, *Amblyomma americanum*, is recognized as the primary vector, while *Dermacentor variabilis* (the American dog tick) is also regarded as a vector capable of transmitting the infection. In bobcats, infections are typically mild or subclinical, enabling them to act as persistently infected carriers and serve as the major reservoir population that sustains the parasite in nature. Domestic cats that survive the acute phase of the disease may likewise remain chronically parasitaemic, harbouring piroplasms in their red blood cells for life. These subclinical chronic carriers represent an ongoing source of infection and contribute to the continued risk of *C. felis* exposure among susceptible domestic cats.

Clinical signs:

Clinical illness associated with *Cytauxzoon felis* typically develops 1 to 3 weeks after infection, and the initial signs are nonspecific, including anorexia, dehydration, lethargy, dyspnoea, pallor and pale or icteric mucous membranes. A physical examination generally reveals marked pyrexia, although hypothermia may be present in moribund animals. Tachypnoea and tachycardia are common, with or without evident respiratory distress, and abdominal palpation often indicates splenomegaly and/or hepatomegaly. As the disease

progresses, neurological manifestations such as altered mentation, vocalisation, seizures and, ultimately, coma may occur. The infection advances rapidly, and most clinically affected domestic cats die within approximately one week of symptom onset. Anaemia is a characteristic feature of *C. felis* infection and results from the schizogonous phase, during which schizont-laden mononuclear phagocytes obstruct blood vessels in organs such as the liver, lungs, lymph nodes and spleen. Once maturation is complete, the parasite transitions to the erythrocytic phase, invading red blood cells as piroplasms and causing haemolysis. Reported haematobiochemical abnormalities include reduced haematocrit and haemoglobin concentrations, thrombocytopaenia, and elevated levels of glucose, hepatic enzymes and serum albumin.

Diagnosis:

Diagnosis of cytauxzoonosis is most commonly performed through microscopic examination of peripheral blood smears, where the parasite may display variable morphology. The typical appearance of *Cytauxzoon felis* is a small ring-shaped structure measuring 1–3 µm in diameter with a prominent, rounded nuclear region positioned along the ring—often referred to as a "signet ring" form. Additional morphological variants may be observed, including elongated "safety-pin" forms characterized by two nuclear bodies positioned at opposite ends, as well as comma-shaped or linear forms that lack a distinct nuclear thickening. Schizonts of *C. felis* can also be detected in fine-needle aspirates from infected organs such as the spleen and lymph nodes, or through histopathology and impression smears. Molecular confirmation is typically achieved using polymerase chain reaction (PCR), which offers markedly greater sensitivity and specificity than light microscopy. Common genetic targets for PCR-based identification include 18S rRNA, internal transcribed spacer 1 (ITS1), cytochrome b (cytb) and cytochrome c oxidase subunit III (cox3). However, these advanced diagnostic methods require more time and are relatively costly compared with microscopic approaches.

Treatment:

The current recommended treatment for feline cytauxzoonosis involves a combination of atovaquone (15 mg/kg orally every 8 hours) and azithromycin (10 mg/kg orally every 24 hours), alongside aggressive supportive and nursing care, which has been associated with high survival rates. Traditional drugs such as diminazene aceturate have generally proven ineffective and are frequently associated with adverse effects; however, Meier and Moore reported some efficacy with diminazene (2–3.5 mg/kg intramuscularly) or imidocarb (2 mg/kg intramuscularly), administered twice within one week, in combination with adjunctive heparin and intravenous fluid therapy. Other agents, including parvaquone or buparvaquone,

may offer potential benefits, but evidence supporting their effectiveness is limited. Supportive and critical care are crucial for improving prognosis, as cats that survive infection may remain chronic carriers. Because cytauxzoonosis commonly causes hemolysis, interventions to enhance the oxygen-carrying capacity of the blood are often necessary, including transfusions of whole blood, packed erythrocytes, or hemoglobin-based oxygen carriers such as Oxyglobin (Biopure Corporation). Whole blood transfusions provide plasma constituents that may help manage disseminated intravascular coagulation (DIC), whereas Oxyglobin offers strong oncotic support. Given that DIC is a frequent complication, prophylactic heparin therapy (100–150 U/kg subcutaneously every 8 hours) is also recommended to improve clinical outcomes.

FELINE BABESIOSIS

Babesiosis is a tick-borne haemoparasitic disease caused by protozoa of the genus *Babesia*, belonging to the phylum Apicomplexa. The first clinical cases of feline babesiosis were reported in Cape Town, South Africa, in 1937. Historically, feline *Babesia* species were classified based on their morphology as either "small" or "large" organisms. Small species, measuring 1.0–2.5 µm, include *B. felis* and *B. cati*, whereas large species, ranging from 2.5–5.0 µm, comprise *B. herpailuri* and *B. pantherae*. Among these, *B. felis* is considered the most virulent species in cats, while *B. cati* is generally regarded as the least pathogenic.

Transmission:

Although experimental transmission studies in cats are lacking, all *Babesia* species are generally believed to be transmitted through tick vectors. Ticks belonging to the genera *Ixodes*, *Dermacentor*, *Rhipicephalus*, *Amblyomma*, and *Haemophysalis* are known to infest cats and are considered likely vectors. Mechanical transmission through other biting arthropods may also occur. While transmission via blood transfusion or cat bites and fights has not been documented, these routes remain theoretically possible. Infection is established when ticks ingest erythrocytes containing merozoites from parasitized hosts, which subsequently undergo multiple fission within the arthropod's salivary glands to produce infective sporozoites. These sporozoites are then inoculated into the host during the tick's blood meal. The prepatent period for *B. felis* ranges from 3 to 28 days. Both transstadial and transovarial transmission in adult female ticks play a crucial role in maintaining the parasite within vector populations and sustaining infection cycles in endemic areas.

Clinical signs:

Babesiosis in domestic cats is generally associated with nonspecific clinical signs such as anorexia, lethargy, anaemia, and icterus. Acute infections caused by *B. felis* often result in severe illness, with the severity influenced by the host's immune status, the chronicity of

infection, and the presence of concurrent diseases. Haematological and biochemical abnormalities commonly observed include macrocytic hypochromic regenerative anaemia, hyperbilirubinemia, and elevated alanine aminotransferase (ALT) activity. Co-infection with immunosuppressive viruses such as feline leukemia virus (FeLV) and feline immunodeficiency virus (FIV) can exacerbate clinical signs and contribute to higher levels of parasitaemia, further complicating disease management.

Diagnosis:

Examination of stained blood smears has traditionally been the standard diagnostic method for feline babesiosis and is reliable when parasitaemia is moderate to high. Capillary blood samples, obtained from sites such as the ear tip or toenail, can improve detection, and freshly prepared smears are recommended for accurate identification. Automated flow cytometric techniques have also been employed to detect *Babesia* in both reticulocytes and mature erythrocytes. The piroplasms of *B. felis* are small and typically appear as single or paired annular bodies (signet ring), pear-shaped forms, and, rarely, tetrads (Maltese cross). In contrast, *B. cati* is observed as single or paired annular bodies within erythrocytes and generally causes milder disease. Among the larger feline *Babesia* species, *B. herpailuri* appears as single or paired annular bodies, while *B. pantherae* piroplasms are smaller than those of *B. herpailuri*. Molecular characterization is essential for differentiating species and describing novel taxa, with 18S rRNA gene-based PCR offering high sensitivity and specificity for detection and identification of *Babesia* species. Although indirect fluorescent antibody (IFA) testing can detect antibabesial antibodies in infected or exposed animals, it is not recommended for routine diagnostic use due to limitations in specificity and practical application.

Treatment:

Treatment of feline babesiosis, particularly infections caused by small *Babesia* species, is challenging due to a poor initial therapeutic response and a high likelihood of relapse. Current management primarily focuses on resolving clinical signs and correcting anaemia rather than achieving complete parasitic clearance. Monitoring through routine haematological assessments and blood smear examination is recommended. Primaquine phosphate is considered the drug of choice for small feline *Babesia* infections, administered at 0.5–1.0 mg/kg orally, intravenously, or intramuscularly either as a single dose or daily for three consecutive days. Clinical improvement and reduction of parasitaemia are typically observed within 24–72 hours; however, doses exceeding 1 mg/kg can be fatal, and oral administration frequently induces vomiting. Long-term therapy decisions should be guided by haematologic parameters and clinical status rather than parasitaemia alone. For large

Babesia species, diminazene aceturate is a potential option, with a single intramuscular dose of 3.5 mg/kg showing variable efficacy in treating *B. felis*. Imidocarb dipropionate is ineffective against *B. felis*, but a single dose of 2.5 mg/kg IM, alone or combined with doxycycline, has proven effective for resolving clinical signs in infections with *B. canis* subsp. *presentii* and *B. herpailuri*. Adverse effects of treatment, often linked to the anticholinergic properties of the drugs, may include salivation, lacrimation, vomiting, diarrhoea, muscle tremors, restlessness, tachycardia, and dyspnoea. Supportive care is crucial and includes fluid therapy to maintain blood volume, ensure adequate tissue perfusion, correct acid-base and electrolyte imbalances, promote diuresis, and prevent red blood cell sludging. Blood transfusions, preferably packed red blood cells, may be required in cases of severe anaemia resulting from haemolysis. Additional supportive measures include administration of vitamins and minerals, corticosteroids, anabolic steroids, nonsteroidal anti-inflammatory drugs, antioxidant therapy, nutritional support, and appetite stimulants to aid recovery.

Conclusion

Feline vector-borne diseases, including hepatozoonosis, cytauxzoonosis, and babesiosis, represent significant and growing threats to domestic cat health worldwide. These infections are often challenging to diagnose due to nonspecific or subclinical clinical signs and the low parasitaemia associated with many haemoparasites. Molecular diagnostic tools, particularly PCR, have greatly enhanced detection and species differentiation, while blood smear examination remains useful in certain cases. Effective management requires species-specific antiprotozoal therapy combined with supportive care to address complications such as anaemia, hemolysis, and organ dysfunction. Preventive measures targeting blood-feeding vectors, such as ticks and fleas, are critical to reducing disease incidence. Greater epidemiological surveillance, improved understanding of transmission dynamics, and early intervention strategies are essential to safeguard feline health and limit the spread of these vector-borne pathogens.

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