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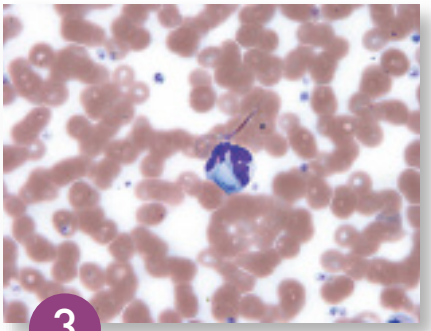
Peer Reviewed

Canine Hepatozoonosis

Amblyomma maculatum (H americanum)

1

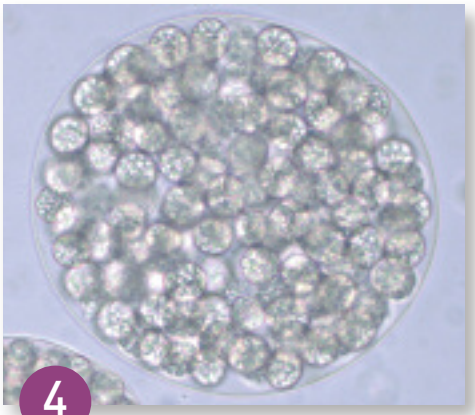


3

Gamonts (magnification, 100×)

Rhipicephalus sanguineus (H canis)

2



4

Polysporocystic oocysts from tick body *(H americanum)* (magnification, 40×)

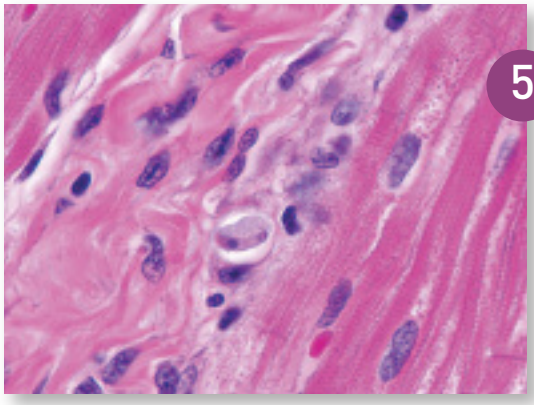
Canine hepatozoonosis, caused by *Hepatozoon canis*, has been recognized as a mild disease of dogs in India since the early 1900s. It was subsequently seen in southern Europe, southeastern Asia, and Africa and was more recently reported in the Americas.

American canine hepatozoonosis (ACH) was first reported in the Gulf Coast of Texas in 1978. Because of differences in parasite structure, tissue tropism, clinical signs, laboratory abnormalities, pathologic manifestations, and tick vectors, the North American organism was designated and later confirmed as a new species, *H americanum*, which is prevalent throughout the southeastern United States.

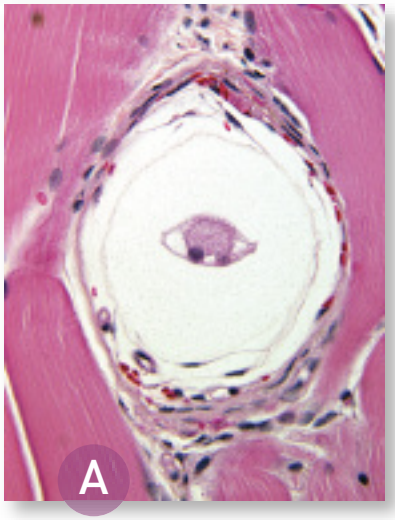
ACH = American canine hepatozoonosis

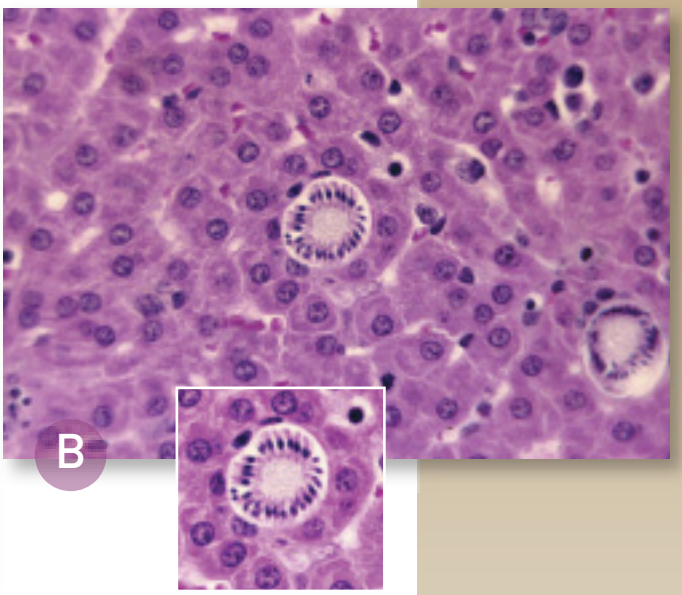
LIFE CYCLE

These *Hepatozoon* have a 2-host lifecycle. The definitive hosts, *Amblyomma maculatum* (**Figure 1**; *H americanum*) and *Rhipicephalus sanguineus* (**Figure 2**; *H canis*), acquire gamonts (**Figure 3**) during feeding. Canine intermediate hosts are exposed by ingestion of polysporocystic oocysts (**Figure 4**; *H americanum*) in the tick body cavity or cystozoites (**Figure 5**; *H americanum*) in tissues of a paratenic host; transmission can also occur transplacentally (*H canis*).

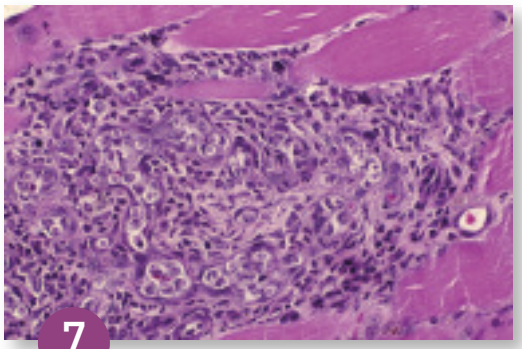


5 *H americanum* cystozoite in tissue of rabbit transport host (magnification, 40×)





6 Typical appearance of 2 *Hepatozoon* species meronts. (**A**) Early *H americanum* with onion skin appearance (magnification, 40×) and (**B**) early *H canis* with wheel-spoke appearance (magnification, 40×). **Inset:** Mature *H canis* (magnification, 40×).



7 Pyogranulomatous inflammation and vasculitis

8 Hypertrophic osteopathy

Merogony occurs in tissues of the canine host (**Figure 6A**, *H americanum*; **Figure 6B**, *H canis*). Released merozoites provoke pyogranulomatous inflammation and vasculitis (**Figure 7**) that may lead to hypertrophic osteopathy (**Figure 8**) with ACH. Merozoites enter neutrophils to become gamonts.

CLINICAL SIGNS

Disease caused by *H americanum* is often severe, whereas clinical illness due to *H canis* is much less serious. Malaise, pyrexia, anemia, myalgia, mucopurulent ocular discharge, and weakness that are nonresponsive to treatment, along with marked neutrophilic leukocytosis (leukocyte count, 20,000–200,000 cells/mm^3), periosteal bone proliferation, and muscle atrophy, strongly suggest ACH.

DIAGNOSIS & TREATMENT

Muscle biopsy, blood smear examination, or polymerase chain reaction testing of blood are used to diagnose *H americanum* infection while blood smear examination is used to diagnose *H canis* infection. Current therapies result in clinical remission but are not curative.

SEE page 44 for **Canine Hepatozoonosis: *Hepatozoon americanum* vs *Hepatozoon canis***—a handout detailing the differences between these 2 infections.

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Canine Hepatozoonosis:
Hepatozoon americanum vs *Hepatozoon canis*

Variable	*H americanum*	*H canis*
Geographic distribution	Southeastern United States	Southern Europe, Middle East, Asia, South America, North America
Modes of transmission	Ingestion of tick host or paratenic hosts	Ingestion of tick host, vertical
Tick host & vector	*Amblyomma maculatum*	*Rhipicephalus sanguineus*, *Haemaphysalis* species, *Amblyomma ovale*
Meront tissue trophism	Skeletal muscle, myocardium	Spleen, lymph nodes, bone marrow
Meront appearance	"Onion skin" cysts	"Wheel spoke"
Clinical signs	• Signs wax and wane • Fever • Lumbar pain • Reluctance to move • Mucopurulent ocular discharge • Stilted gait • Muscle atrophy • Hyperesthesia	• Usually no signs but can be severe with high parasitemia (> 5%) • Fever • Lethargy
Laboratory abnormalities	• Marked leukocytosis with mature neutrophilia • Mild anemia • Elevated alkaline phosphatase • Hypoglycemia • Hypoalbuminemia	• Anemia • Mild to moderate leukocytosis with neutrophilia • Hyperglobulinemia
Histopathologic features	Pyogranulomatous myositis, hypertrophic osteopathy	Necrotizing hepatitis, splenitis, nephritis, pneumonia
Radiographic abnormalities	Periosteal proliferation of bones	Nonspecific
Severity of disease	Severe; guarded prognosis	Usually mild; good prognosis
Treatment	Trimethoprim/sulfadiazine, clindamycin, and pyrimethamine (14 days) and NSAID followed by long-term daily decoquinate	Imidocarb dipropionate at 14-day intervals and doxycycline (21 days)

NSAID = nonsteroidal antiinflammatory drug