Emerging Infectious Diseases of Chelonians

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KEYWORDS

• Testudines • Chelonians • Adenovirus • *Iridovirus* • *Ranavirus* • Coccidiosis
• Cryptosporidiosis

KEY POINTS

- Intranuclear coccidiosis of Testudines is a newly emerging disease found in numerous chelonian species that should be on the differential list for all cases of systemic illness and cases with clinical signs involving multiple organ systems. Early diagnosis and treatment is essential, and PCR performed on conjunctival, oral and choanal mucosa, and cloacal tissue seems to be the most useful antemortem diagnostic tool.
- *Cryptosporidium* spp have been observed in numerous chelonians globally, and are sometimes associated with chronic diarrhea, anorexia, pica, decreased growth rate, weight loss, lethargy, or passing undigested feed. A consensus PCR can be performed on feces to identify the species of *Cryptosporidium*. No treatments have been shown to clear infection, but paromomycin did eliminate clinical signs of disease in a group of *Testudo hermanni*.
- Iridoviral infection is an emerging disease of chelonians in outdoor environments that usually presents with signs of upper respiratory disease, oral ulceration, cutaneous abcessation, subcutaneous edema, anorexia, and lethargy. The disease can be highly fatal in turtles, and can be screened for with PCR of oral and cloacal swabs, and whole blood.
- Adenoviral disease is newly recognized in chelonians and presents most commonly with signs of hepatitis, enteritis, esophagitis, splenitis, and encephalopathy. Death is common in affected chelonians. PCR of cloacal and plasma samples has shown promise for antemortem detection, although treatment has proved unsuccessful to date.

Several infectious diseases continue to be prevalent in captive and wild chelonians. Important diseases that have been well described include mycoplasmosis and herpesvirus in tortoises, and herpesvirus-associated fibropapillomas in sea turtles. Diseases that are currently emerging as important pathogens include intranuclear coccidiosis of Testudines, cryptosporidiosis in tortoises, *Iridovirus*, and adenovirus.

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This article describes each of these emerging pathogens by its biology, epidemiology, clinical signs, diagnosis, treatment, gross pathology, histopathologic changes, and any anticipated future trends.

INTRANUCLEAR COCCIDIOSIS OF TESTUDINES

Biology and Epidemiology

A disease-causing, primarily intranuclear, coccidian parasite of Testudines (TINC) was first identified in radiated tortoises in 1990.[1] Subsequently, the same pathogen (GenBank accession number AY728896), determined by sequencing of a coccidial 18S rRNA consensus polymerase chain reaction (qPCR) product, has caused systemic disease in impressed tortoises (*Manouria impressa*); leopard tortoises (*Stigmochelys* [*Geochelone*] *pardalis*); Forsten tortoises (*Indotestudo forstenii*); bowsprit tortoises (*Chersina angulata*); spider tortoises (*Pyxis arachnoides*); flat-tailed tortoises (*Pyxis planicauda*); Galapagos tortoises (*Chelonoidis nigra becki*); eastern box turtles (*Terrapene carolina carolina*); and Arakan forest turtles (*Heosemys depressa*).[2–5] The authors have also confirmed the disease in several other chelonian species (Paul M. Gibbons, DVM, unpublished data, 2013) postmortem by histopathology usually after severe acute illness. Cases have been reported in Germany and the United States including New York, Louisiana, Florida, Georgia, Texas, California, and Hawaii, although no systematic prevalence study has been undertaken to date.[1–7] Most of these cases occurred in zoologic collections and a few had been recently imported from Indonesia.

The life cycle and method of transmission of the TINC organism are unknown and the organism has not been studied in vitro. Although it has not been assigned to a genus, the phylogenetic position of the organism has been characterized by sequencing an 18S small ribosomal unit. It is most closely related to *Eimeria arnyi*, a species identified by oocysts in the feces of the eastern ringneck snake (*Diadophis punctatus arnyi*).[3,8] Life stages of the TINC organism including trophozoites, meronts, merozoites, macrogametocytes, microgametocytes, and nonsporulated oocysts have been found in the nucleus, cytoplasm, and extracellularly in tissues by histology and electron microscopy. Reports have failed to describe TINC oocysts in feces, and the authors have been unable to find oocysts on fecal flotation from several polymerase chain reaction (PCR) test-positive animals with clinical signs of illness. The TINC organism has not been identified outside of cells or tissues, and samples of various invertebrates and substrate from the enclosures of affected tortoises during an outbreak of TINC at a zoologic facility were negative when tested by quantitative PCR.[4]

Clinical Signs

Although the organism is often reported in highest number in the kidney or pancreas, infection is frequently disseminated to an array of organs so clinical signs vary among cases and are not specific to TINC. Clinical signs range from mild chronic conjunctival or nasal erythema or discharge to severe gasping, subcutaneous edema, and ulceration of the cloacal mucosa. Additional clinical signs can include anorexia, lethargy, lack of normal diurnal behavior patterns, increased respiratory effort, mouth breathing, and rapid weight gain or loss. Rapid weight gain is common, and results from ascites or retention of urine in the urinary bladder (Paul M. Gibbons, DVM, personal communication, 2013). Death can occur within a few days of initial clinical signs, or after months of clinical management including improved husbandry and anticoccidial drug therapy (Paul M. Gibbons, DVM, personal communication, 2013). Stress and thermoregulatory challenges (eg, insufficient heat or evaporative heat loss) seem to