Microsporidiosis in a Flock of Tricolor Parrot Finches (Erythrura tricolor)

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The Microspora is a diverse phylum of spore-forming, obligate intracellular protozoan parasites of a wide range of animal hosts [1]. It contains well-known species of veterinary importance, such as Encephalitozoon cuniculi, which is a common and well-documented infection in rabbits. However, a wide range of microsporidians infect invertebrate and vertebrate hosts and should be considered as potential pathogens in avian and exotic veterinary patients. Furthermore, several Microspora genera of animal origin have zoonotic potential for immunocompromised humans. In this report the authors describe a case of microsporidiosis that occurred in a flock of tricolor parrot finches.

Case report

Mortalities (n = 7) that occurred over the period of 1 week in a small aviary flock of tricolor parrot finches were investigated by necropsy and histopathologic examination. The flock of approximately 60 birds was housed in a well-constructed and -managed indoor aviary. Other species housed in the facility included red-headed parrot finches (E psittacea) and several species of waxbills. The birds were kept in pairs and housed as one or two pairs in each flight. The flooring substrate consisted of concrete covered by a plastic liner, on top of which dry meadow grass (hay) was placed. Water was provided by an automatic watering system attached to the fronts of the flights. These were constructed so as to prevent spillage onto the floor.
Food was provided in trays and consisted of a dry seed mix, live food, including fly larvae and mealworms, soft food, and a variety of soluble and insoluble grits. Two tricolor finches were presented for necropsy examination; similar lesions were present in both. There was a pale thickening of the serosal surfaces of the gastrointestinal tract, pancreas, and airsacs. Smears of the coelomic cavity were stained with Diff Quik (Lab Aids Pty, Narrabeen, Australia) and Gram’s stain and demonstrated macrophages and occasional variably stained rods.

**Histopathologic findings**

Formalin-fixed tissue was processed in paraffin for routine histologic sectioning and staining with hematoxylin and eosin. Histologic examination demonstrated a widespread nodular to diffuse granulomatous inflammation of the serosal surfaces of the gastrointestinal tract (Fig. 1), peritoneum, perirenal airsacs and connective tissue, bone marrow, dura, and conjunctiva. This inflammation was composed predominantly of foamy macrophages containing numerous intracytoplasmic organisms measuring 1 to 2 μm in diameter that were refractile under polarized light. The organisms had a thick capsule and stained positive using a modified Trichrome stain and variably positive with Gram’s, Giemsa, periodic acid-Schiff, Ziehl-Neelsen, and Silver staining techniques (Fig. 2). The brain, spinal cord, and retina appeared normal. A diagnosis of severe disseminated granulomatous peritonitis, meningitis, osteomyelitis, and conjunctivitis was made based on the histologic findings.

**Electron-microscopic examination**

Formalin-fixed tissues were post-fixed in glutaraldehyde and osmium for resin embedding and electron-microscopic examination. The organisms seen in hematoxylin and eosin–stained histologic sections were demonstrated as short, blunt-ended rods 1.0 to 1.5 μm long and 1 μm in

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Fig. 1. Histologic section through the duodenum showing a diffuse thickening of the serosal surfaces and infiltration of the spleen with macrophages (arrowheads) (hematoxylin-eosin).
diameter packed in parasitophorous vacuoles (Fig. 3). They had a capsule with a thin outer electron-dense membrane and a thick inner electron-lucent zone. The cell matrix was electron-dense and contained occasional membrane-bound structures. No evidence of apical complexes characteristic of apicomplexan parasites was seen. However, numerous sections demonstrated coiled filaments present at the pole of the organism, consistent with the polar filament of microsporidia.

Discussion

The cause of the mortalities in this case report was diagnosed as microsporidiosis based on morphologic and electron-microscopic features. These included the presence of large numbers of intracytoplasmic spores associated with widespread granulomatous inflammation and ultrastructural features consistent with microsporidia, such as the presence of a coiled polar filament and an electron-dense exospore and thick, electron-lucent endospore. Definitive identification of microsporidian parasite species requires molecular, immunologic, or biochemical analysis. Although suitable samples for DNA analysis of the tissue were not available in this case, it presents morphologic and ultrastructural features consistent with infection of *E hellem* or some other, similar microsporidian parasite. *E hellem* appears to be the only microsporidian identified to the species level in birds: it has been identified using molecular methods in various psittacine birds [2–9], pigeons [10], an ostrich [11], hummingbirds [12], and finches [13]. However, molecular techniques should not be the only tool used for making a diagnosis of microsporidiosis, because of environmental contamination and the potential for asymptomatic infection and shedding of spores [10].

Differential diagnoses that were considered according to the history and lesions were infection with intracellular organisms, including coccidian and other apicomplexan parasites such as *Isospora*, *Eimeria*, and blood
parasites; *Chlamydophilosis*; disseminated mycobacteriosis; and other bacterial and fungal species. Emerging obligate intracellular parasites in birds include a yeast-like organism that has been recognized as a cause of death in muscovy ducks throughout the world. However, the disease in ducks mainly involves the lungs and was first reported as a blood parasite [14,15], then as an intracellular bacterium [16,17], before it was shown to be yeast-like by electron-microscopy [16].

In humans, the most effective drug for treating microsporidiosis is albendazole [18,19], which has been used successfully to treat a cockatoo with conjunctivitis [9]. However, the drug is not effective against all microsporidians. Topically administered fumagillin, an antibiotic produced by the fungus *Aspergillus fumigatus*, is highly effective for treating keratoconjunctivitis due to *Encephalitozoon* species [18] but may be toxic when given systemically. Topical fumagillin for an ocular infection has been used in combination with systemic albendazole to treat microsporidian keratoconjunctivitis in a double yellow-headed Amazon parrot (*Amazona ochrocephala oratrix*) [7]. Chitin synthesis inhibitors such as lufenuron may also be effective systemic treatment options [19].

Although systemic, conjunctival, and pulmonary lesions are recognized in microsporidian infections, the most likely site for primary infection is the intestines. This results in fecal transmission of spores, which are relatively resistant to environmental conditions but are readily disinfected by a range of chemicals, including chlorine [20–22]. However, eradication from an infected closed population may be difficult or impossible, because vertical transmission of microsporidians is well known in invertebrate and vertebrate hosts [23–26]. Some microsporidians rely on vertical transmission as their major transmission method; some vertically transmitted
Microsporidia may selectively kill or feminize their male hosts, which can significantly alter host population sex ratio and stability [1,24]. In some hosts, horizontal transmission is associated with high parasite burdens and pathologic conditions, whereas vertical transmission is characterized by low virulence. Evidence for vertical transmission of *E. cuniculi* has been documented in rabbits [25], as well as in chickens [26].

**References**


