Plight of the Parrots

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Introduction:

Sarcocystis falcatula is a protozoan parasite causing the disease sarcocystosis in Old World parrots. Old World parrots are those that originate on the continents of Africa, Asia, and Australia such African Greys, cockatoos, and eclectus parrots.[6] While Old World parrots are more likely to develop severe clinical disease, New World parrots such as Amazons, conures, and macaws typically demonstrate benign cysts with infrequent clinical signs. The most commonly diagnosed birds are those in zoos, aviaries, personal collections, and breeding programs where the disease can be detrimental to the population.

History and Presentation:

During 2017-2018, a zoo has presented eleven out of their one-hundred fifty birds to the MSU-CVM Diagnostic Laboratory Services for clinical signs including nasal discharge, diarrhea, and being found dead in the bottom of the cage. A limited history of the birds was given with the only details being that the birds were separated by species into smaller aviaries and no recent introductions had been made. Also, in March 2017, two other birds on the premises were treated with enrofloxacin for diarrhea, which resolved, and minimal diagnostics were performed. Below, four individual cases submitted to the MSU-CVM DLS are described.

Case 1, a 17-year-old male African Grey, presented deceased to the referring DVM on March 30, 2017 after losing 91g in 38 days. Shortly before his death, he was treated with oral enrofloxacin at 10mg/kg. He presented to MSU-CVM on April 3, 2017 for a full diagnostic necropsy. On June 15, 2017, Case 2, a male conure, was also transported to MSU-CVM for diagnostic necropsy after being found dead in his exhibit. Case 3, a 22-year-old male parrot, presented to the MSU-CVM on May 31, 2017 after being found deceased in his enclosure.

Lastly, Case 4, a 20-year-old female African Grey, presented to the referring DVM from the zoo with mild clear nasal discharge first noted on May 17, 2017. Radiographs were taken and demonstrated mild congestion of the air sacs. She died on May 18, 2017 after an episode of acute respiratory distress and was necropsied that same day.

Pathophysiology:

Sarcocytosis is a severe and often fatal disease caused by *Sarcocystis* species. Its life cycle is a predator-prey relationship that is non-pathologic for the definitive host (predator) but pathologic for the intermediate hosts.[10] Birds are the intermediate host of *Sarcocystis falcatula* with opossums as the definitive host. The opossum sheds infective sporocyts in its feces for long periods of time, which results in environmental contamination. Birds become infected when opossums, attracted to psittacine diets, contaminate the enclosures or feed. Cockroaches, which serve as a paratenic hosts, are also attracted to psittacine diets and may contaminate the feed with feces or may be eaten by the birds. Sporocyts are ingested and then release sporozoites in the small intestine where they disperse to various tissues via the blood stream. Schizonts asexually reproduce in arteriole, capillary, and venule endothelial cells causing vascular damage and leakage. There are five clinical forms of sarcocystosis which are described based on the organ system affected. The pulmonary form is the most acute and the most common presentation, which will be the focus of this paper. The muscular form causes mild myositis resulting in muscle weakness in the legs and wings. The cardiac muscle form is uncommon and typically only identified on histopathologic analysis of the myocardium. The incidental muscular form, most common in New World avian species, is present when cysts are identified but do not result in death. Lastly, the encephalitis form is rare and causes inflammation of the brain.

Old World psittacines (including African Grey Parrots) are predisposed to the pulmonary form where the primary site is the lungs, but lesions can be seen in the liver, spleen, kidneys, intestines, cardiac and skeletal muscle, and brain. Merozoites are released and form cysts within skeletal muscle.[4]

Diagnostic Approach/Considerations:

Although presumptive diagnosis and treatment can be made if clinical signs are noticed before death or if other individuals in the collection have been diagnosed; however, confirming this disease in a live bird is challenging. Diagnostic testing and treatment are similar to any issues arising in a population (population medicine) where one bird is necropsied, samples are collected, and the results are used to develop a treatment plan for the remaining birds.[11]

Sarcocystosis in Old World psittacines often does not result in clinical signs but animals are found dead. When present, clinical signs include weakness, dyspnea, neurologic changes, and anorexia. Gross lesions include pulmonary edema, congestion, and hemorrhage with splenomegaly and hepatomegaly. Histologic findings include pulmonary edema with schizonts present with capillary endothelial cells.[5] The significant and detrimental host response of Old World psittacines to *Sarcocystis* is likely due to their evolution and interaction with opossums, given that *Sarcosystis falcatula* are indigenous to the Americas.[7]

On gross examination, Case 1 presented with fair body condition with a well-muscled keel and coelomic adipose deposition. The lungs were red to pink with bubbles on the pleural surface, with a mild amount of clear fluid present on cut section. Air sacs were within normal limits. The spleen was enlarged and purple. The liver was light brown with numerous 2-4mm yellow-tan distributed in multiple lobes. The small intestine was distended with a dark liquid

content. On histopathology the lungs were flooded with eosinophilic fluid (pulmonary edema) and the capillaries contained linear basophilic organisms, consistent with protozoal merozoites. Histologically, the liver had areas of hepatocellular necrosis with colonies of bacteria. As an incidental finding, the carotid artery contained epidermal proliferation and lipid vacuoles (atherosclerosis). Additional testing included chlamydia PCR, aerobic culture and sensitivity (liver, small intestine, and cloaca), and influenza testing. Chlamydia PCR was negative. All cultures on the various tissues had a moderate growth of Salmonella, which were serotyped as *Salmonella typhimurium*. This bird had interstitial pneumonia with pulmonary edema secondary to *Sarcocystosis falcatula*. The hepatocellular necrosis and enteritis were due to *Salmonella typhimurium*, and atherosclerosis from an inappropriate diet as well as African Grey's genetic predisposition for the disease process.[7]

Case 2 appeared in poor body condition with a prominent keel. The air sacs were clear and without edema. The spleen was enlarged. The intestinal contents were tan to green. The most notable finding was dense, heavy, dark red to purple lungs that exuded a pink-tan frothy fluid (pulmonary edema). Histologic examination of the lungs demonstrated clusters of elongated basophilic organisms within the capillary endothelial cells. Additional findings included multifocal areas of hepatocellular necrosis. Aerobic culture of the lung grew a moderate growth of *Salmonella sp*. and *Escherichia coli*. Liver aerobic culture also grew a heavy growth of *Salmonella sp*. This bird died as a result of the pulmonary edema secondary to a protozoal merozoites, consistent with *Sarcocystis falcatula*. The acute multifocal hepatocellular necrosis was secondary to *Salmonella sp*.[1,9]

Case 3 presented with good body condition and normal keel musculature. The abdominal and thoracic air sacs were clear and without edema. The lungs were pink, soft, and spongy.

Histologic examination of the lungs demonstrated eosinophilic proteinaceous fluid and protozoal schizonts. Examination of the liver demonstrated hepatocellular necrosis. The spleen contained large numbers of mononuclear cells. Ureteral obstruction by urates was also noted in on lobe of the kidney. Intestinal salmonella serotyping was performed and was differentiated as *Salmonella typhimurium* identical to Case 1. This bird died as a result of pulmonary edema secondary to a protozoal merozoites consistent with *Sarcocystis falcatula*. The salmonellosis was a comorbidity causing severe enteritis, hepatitis, and splenitis.

Case 4 presented with a prominent keel and poor body condition. On gross examination, abnormalities found included an enlarged liver with a 1x1cm yellow lesion on the left liver lobe. The lungs were heavy, pink, and exuded fluid, consistent with pulmonary edema. The kidneys had widely disseminated white pinpoint lesions. The air sacs, gastrointestinal tract, and other organs appeared normal. On histopathologic examination, eosinophilic proteinaceous fluid was noted in the air capillaries of the lungs. There was no evidence of protozoal merozoites. The kidneys had chronic interstitial nephritis with gout tophi. Aerobic culture of the lungs noted light growth of gram positive environmental organisms and faint growth of *Pseudomonas pseudoalcaligenes*.

Treatment and Management:

Typically, infection with *Sarcocystis spp* is an acute presentation; therefore, treatment is not possible. If disease is suspected in a flock, antiprotozoal medications and supportive care are necessary. The preferred combination therapy includes pyrimethamine at 0.5-1mg/kg orally every 12 hours for 2-4 days then 0.25mg/kg orally every 12 hours for 30 days and trimethoprim-sulfadiazine at 5mg/kg IM every 12 hours or 30-100 mg/kg orally every 12 hours for seven days. The treatment, however, may be difficult or unrewarding.[3]

Prevention is key for combating this disease in aviaries. Strategies include preventing opossums from entering aviary mews or feed areas using mess netting, purchasing sealed air-tight containers to store feed, filling in any holes that opossums can use to enter the aviary and feed storage areas, trapping and relocating opossums that reside on the zoo grounds, improving the aviary cleanliness, and cockroach population control.[2,6] Changes in diet to a low protein, pelleted, non-seed diet will decrease the risk of atherosclerosis and renal gout as well as making the storage and cleaning of the aviary easier.[8]

Outcome:

The pulmonary form of sarcocystosis was concluded to be the cause of death in cases 1, 2, and 3. Case 4 did not demonstrate a sarcocystis infection. All four cases demonstrated a bacteremia of either *Salmonella sp.* or *Pseudomonas sp.* origin which in consistent with the flock's history of antibiotic susceptible diarrhea. These co-morbidities can both be influenced by the same husbandry and biosecurity protocols. Salmonellosis is also an important zoonotic disease to be aware of for employee and zoo patron health.

With the wide-spread sarcocysis and salmonellosis present in this aviary, husbandry issues and zoonotic risk were addressed with the zoo. These recommendations included diet storage, cockroach and opossum management, environmental decontamination, and husbandry.

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