Proventricular Dilatation Disease (PDD

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Properly known as proventricular dilatation disease (PDD), this suspected viral disease has been given many other names over the years, including macaw wasting disease, as it was thought to be most prevalent in these species. However, the disease has been identified in approximately 50 different species of psittacine birds, including African grey parrots and cockatoos. An epornitic (bird epidemic) of this disease is currently occurring in imported and captive-bred psittacine birds in North America and Europe.

PDD was experimentally reproduced and transmitted by exposing susceptible adult psittacine birds to a viral suspension derived from affected birds. The incubation period is variable, ranging from several weeks to more than 3 months.

PDD causes damage to the neurons of the autonomic (involuntary) nervous system of the GI tract, leading to segmental dysfunction of the crop, esophagus, proventriculus, and small intestine. It has also been found to affect the central nervous system and peripheral nerves. Common clinical signs of PDD include depression, gastrointestinal dysfunction (chronic weight loss, constant or intermittent regurgitation, and sometimes the passage of whole seeds or portions of undigested food in the droppings), and central nervous system signs (ataxia, abnormal head movements, and proprioceptive (sense of body position) and motor deficits). Neurologic signs may be present terminally. Some birds will show only gastrointestinal dysfunction, some will show only CNS signs, and some will show a combination of both. Other infected birds will be asymptomatic but shed the virus continually, serving as the cause of outbreaks in susceptible birds in an aviary or pet retail facility. In some outbreaks, numerous cases of PDD will occur simultaneously. Sometimes, several birds will die in an outbreak and the problem will appear to resolve, only to reappear years later. It is more common, however, for many of the birds exposed to an affected bird to remain asymptomatic.

Definitive diagnosis can be achieved through the histologic demonstration of lymphoplasmacytic (particular type of white blood cells) infiltrates within nerves, ganglia, and neuropil and through the absence of normal myenteric plexi (nerve centers in the muscle), especially within the nerve tissue of the gastrointestinal tract. Most frequently, the diagnosis is made through postmortem submission of a complete set of tissues, including proventriculus, ventriculus, brain, and spinal cord tissue. At necropsy, emaciation, pectoral muscle atrophy, and dilation of the GI tract, including the proventriculus, are observed. Some gross necropsies of birds with PDD may appear normal, and histologic changes can be easily missed, as well.

Ante mortem (before death) diagnosis of PDD is difficult, since no specific diagnostic test is available commercially at the time of this writing. Rather, diagnosis is based upon observing the characteristic clinical signs and ruling out other conditions that may produce those signs. Plain and contrast (barium) radiographs are useful for demonstrating gastric abnormalities in affected birds. Common radiographic findings are proventricular dilatation or distension and increased barium transit time. However, veterinarians should not automatically assume that a bird with a dilated proventriculus has PDD, since other conditions may also produce this radiographic change. Further, the proventriculus of neonates (young birds) is normally dilated and should not be misinterpreted as indicating PDD. Conditions that demonstrate clinical signs similar to PDD include GI foreign bodies and impactions, gastroenteritis, and heavy metal toxicosis (poisonings).

Ante mortem diagnosis is further hampered by the fact that proventricular and ventricular biopsies are not generally recommended due to the risk they pose and the tremendous distension produced by the condition. Full-thickness crop biopsies have shown some promise in the detection of PDD and are commonly used as a definitive test. However, they are helpful only if the results are positive; since false negative results can occur. Because it is important to include nerve tissue samples in this type of biopsy, the crop should be sampled in an area that has at least one large blood vessel and associated ganglia (nerve center). Further, the biopsy size should be at least 1 cm x 1 cm, and it should be taken from the left midlateral aspect of the crop. In several studies, changes consistent with PDD were reliably detected in 68–78% of the crop biopsies collected from positive birds. No birds were observed to have lymphoplasmacytic infiltrates in the crop without concomitant lesions in the proventriculus and/or ventriculus.

Fluoroscopic studies demonstrating abnormal esophageal and ventricular motility have been suggested as being useful in early diagnosis. Blood chemistry may also be useful, since serum amylase and lipase levels are elevated in some patients, and hypoalbuminemia may be present. Other possible indications of PDD are elevated CPK and feather changes.

Treatment for PDD is usually unrewarding, but careful nursing may prolong the life of an affected bird. Celebrex has been indicated as a cure for PDD, however, although it may relieve the infiltration of cells around the nerves, it does not correct the disease condition. It only provides symptomatic relief but in many cases that can prove useful in easing the discomfort of the bird.

Strict isolation of affected or suspect birds is recommended. The mates, offspring, and siblings of birds that have been definitively diagnosed with PDD through histologic evaluation should be considered at extra risk of developing the disease and should be carefully monitored, but they should not be euthanized. Until a vaccine or an accurate diagnostic test is developed that can detect the presence of the virus or an antibody response to it, birds that have been exposed to PDD should be isolated or kept in single-bird households where there is no risk of exposure to other birds.

Adapted from *Essentials of Avian Medicine: A Guide for Practitioners, Second Edition* by Peter S. Sakas, DVM, MS. Published by the American Animal Hospital Association Press. (2002)