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Proventricular Dilatation Disease
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Round Table Discussion

Proventricular Dilatation Disease

Proventricular dilatation disease (PDD) is one of the most important infectious diseases facing aviculture and the avian practitioner today. This disease is also known as proventricular dilatation syndrome (PDS), neuropathic gastric dilatation, and macaw wasting syndrome. With an apparently long incubation period, the possibility of long-term viral shedding in asymptomatic birds, and the difficulty of diagnostic screening, this disease is particularly enigmatic. By anecdotal accounts, the disease does appear to be more prevalent and spreads more rapidly in indoor aviaries, especially in northern climates. In this forum, I have asked Dr. Michael Taylor, Dr. Christopher Gregory, and Dr. Robert Schmidt to respond to questions concerning their experiences with PDD. Dr. Taylor is an Assistant Professor at the Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada. Dr. Gregory is a member of the psittacine disease research group at the College of Veterinary Medicine, University of Georgia, Athens, GA. His research is focused on resolving PDD. I have also included excerpts from Dr. Gregory's recent paper[1] in the 1997 *Proceedings of the Annual Conference of the Association of Avian Veterinarians*. Dr. Schmidt is a Diplomate of the American College of Veterinary Pathologists and resides in Davis, CA.

Susan L. Clubb, DVM
Associate Editor

Question: What progress have you made in characterization of the etiologenic agent of proventricular dilatation disease, or PDD?

Dr. Gregory:

We have consistently recovered an approximately 80-nm enveloped virus from naturally and experimentally infected birds. We have yet to demonstrate the presence of this virus in any bird that does not have clinical signs or gross lesions suggestive of PDD. We have developed an assay to detect antibodies against the suspected PDD virus, and we are currently collecting blood samples from flocks experiencing outbreaks to determine what epizootiologic information this assay provides. DNA probes show promise in detecting viral nucleic acid shed in the feces of infected birds. We have confirmed that eastern equine encephalomyelitis virus or an autoimmune process do not cause PDD, as suggested by others. We have confirmed an experimental incubation period that can vary from weeks to greater than 3 months. Experimentally, a suspension containing only one detectable virus can cause clinical signs that vary from bird to bird.

Question: What do you recommend as diagnostic screening tests for PDD?

Dr. Taylor:

Some of the procedures commonly used to screen birds for PDD include fluoroscopy, endoscopy, and biopsy. Fluoroscopy shows great promise in demonstrating abnormalities of gastrointestinal motility far earlier than does radiography. However, a positive fluoroscopic study requires that pathologic lesions are present in the nerves of the gastrointestinal tract to cause recognizable motility abnormalities. Fluoroscopy is superb for demonstrating crop, esophageal, ventricular, and duodenal effects on normal motility caused by PDD, and the fluoroscopic examination can be recorded for analysis and review.

Endoscopy is useful in examining the isthmus region of the proventriculus and the ventriculus from the left caudal thoracic and left abdominal air sacs. Endoscopy is especially helpful in confirming inflammation of the serosal surface of the proventriculus and ventriculus, which is evident in early cases by markedly increased vascularity.

Biopsy of the crop, proventriculus, and ventriculus have all been reported, and currently represents the only method for achieving an antemortem, definitive diagnosis of PDD. The crop remains the only really accessible portion of the gastrointestinal tract from which to harvest the myenteric plexus. The advantages of crop biopsy are ease of exposure and surgical technique, low morbidity and mortality, and ability to collect a large tissue sample. A disadvantage is the chance of a false negative result

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because of the segmental nature of the pathologic lesions of this disease. Biopsy of the proventriculus is contraindicated because of its thin wall, relative lack of accessibility, and high risk of morbidity or mortality if there is leakage of gastric fluids. The ventriculus is somewhat more approachable, and a serosal biopsy sample can be safely collected because of the thick muscularis. However, problems arise because of the small size of biopsy samples. Lesions can be missed because of the segmented occurrence of PDD lesions, or the sample may contain no nervous tissue.

Radiography is not useful in the routine screening of birds for PDD unless the disease is advanced.

Although results of a plasma biochemical analysis are generally not helpful in diagnosis, hypoalbuminemia is a consistent finding in birds with advanced disease. A high concentration of plasma lipase is a frequent, but inconsistent, finding in birds with PDD. This may reflect the segmental involvement of the duodenum in some birds with this disease.

Dr. Gregory:

A presumptive diagnosis of PDD often is based on historical information, clinical signs (progressive weight loss, regurgitation, and passing undigested food, with or without neurologic signs), and radiographic evidence of proventricular dilatation or dysfunction. However, definitive diagnosis of PDD requires histologic examination of tissues collected by biopsy or at necropsy, which confirms the presence of lymphoplasmacytic ganglioneuritis.

Survey and contrast radiographs are useful for demonstrating gastric dysfunction in suspect birds. Distention of the proventriculus and slow transit time of barium are common findings in chronically infected birds. The proventriculus of neonates is normally dilated, a condition that should not be misinterpreted as PDD. The proventriculus of a neonate attains its adult tone and size as the bird enters and completes weaning. Ultrasonic examination may reveal a dilated and impacted proventriculus. Endoscopic examination may show an impacted, ulcerated, and dilated proventriculus.

Question: How reliable is crop biopsy in detecting birds with subclinical disease or in confirming disease in birds exhibiting clinical signs?

Dr. Gregory:

We have no information to document the specificity or sensitivity of crop biopsy in the diagnosis of PDD in birds with subclinical disease. In our experience, the sensitivity of crop biopsy is 76% and the specificity of crop biopsy is 100% in evaluating birds that are known PDD positive. The sensitivity of this test appears to be best when a large sample (at least 0.5 × 0.5 cm) of the crop is excised and when the biopsy sample includes a visible blood vessel.[2]

Dr. Schmidt:

In a review of cases submitted by practitioners for histopathologic examination, I found typical lesions of PDD in the crop in only 30–35% of cases, whereas lesions were present in the ventriculus in approximately 99% of cases and in the proventriculus in approximately 98% of cases. I agree that a large sample of crop tissue increases the chances of obtaining a diagnosis, and I would suggest obtaining a section at least 0.5–1 cm long. The greater curvature of the ventriculus is an ideal site for biopsy; the serosa and approximately 3–5 mm of the ventriculus can be obtained without entering the lumen.

Question: Can rapid serial radiographs serve as a substitute for fluoroscopy?

Dr. Taylor:

It is unlikely that rapid serial radiographs could substitute for fluoroscopy to detect abnormalities of gastrointestinal motility. Motility is fluid and relatively rapid. The radiographs would have to be recorded on an extremely fast film system. Also, anesthesia is absolutely contraindicated in motility studies for PDD, as it will impair or completely stop ventricular contractility. Even benzodiazapine tranquilizers, such as midazolam, were found to completely eliminate ventricular contractions in our study.

Question: What is your differential diagnosis when presented with a bird exhibiting clinical and radiographic signs of PDD?

Dr. Taylor:

My differential diagnostic list consists of disorders that can affect intestinal motility, cause intestinal blockage, or cause similar clinical signs or gross necropsy findings. These include chronic lead or zinc toxicosis, fungal ventriculitis, megabacterial ventriculitis, other bacterial ventriculopathies, a ventricular foreign body causing ventriculoduodenal outflow problems, proventricular or ventricular neoplasia, and papillomatous disease involving the proventriculus or ventriculus, in which lesions are partially obstructive. I have yet to see a bird recover that had clinical, histologically confirmed PDD.

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Some birds may clinically improve, but these birds usually are found to be chronically infected. We have confirmed the presence of long-term, chronically infected birds that have survived for three years and yet have no obvious clinical signs.

Dr. Gregory:

Any process that causes partial blockage of the intestinal tract or maldigestion that prevents food passage can cause clinical signs or radiographic changes suggestive of the gastric form of PDD. These include fungal proventriculitis, megabacteriosis, parasitic enteritis, gastrointestinal foreign bodies, neoplasms, bacterial enteritis, papillomatosis of the proventriculus or esophagus, and intraluminal or extraluminal masses. We have yet to confirm that any bird with histologic lesions consistent with PDD has recovered.[3]

Dr. Schmidt:

In a bird with classic signs of PDD, you must consider any obstructive disorder in your differential diagnosis. We have seen numerous cases of carcinoma of the proventricular–ventricular junction, especially in grey-cheeked parakeets (*Brotogeris pyrrhopterus*). Also, consider avian tuberculosis or foreign body obstruction.

Question: What is your recommendation for dealing with birds exposed to PDD, such as mates of birds that have died of PDD?

Dr. Taylor:

I stress that not all exposed birds will become infected. However, many birds that lose a mate are themselves chronically infected birds. Finding a single bird with confirmed PDD should immediately raise the questions: what birds are in the nearby environment of this bird, and which bird is shedding the virus while appearing clinically normal? We screen suspicious birds after reviewing the epidemiologic history of the aviary. Crop biopsy and fluoroscopy, with results analyzed together, have been the diagnostic tools with the highest yield.

As there is currently no successful treatment for PDD, we advise the aggressive prevention of new disease by the elimination of chronically infected birds from the aviary. We have suggested that these birds are the insidious facilitators of new cases of PDD.

Dr. Gregory:

Birds that are directly exposed (mates, offspring, or siblings) to those that have died from PPD (as confirmed by histologic examination) should be considered at risk and placed in isolation for at least 1 year. They should not be euthanatized. In our experience, each 6 months that a bird remains asymptomatic after direct exposure to a PDD-positive bird is a favorable indication that the bird may remain unaffected.[1,3] Many birds that are directly exposed to those with PDD never develop the disease. Until we can confirm the significance of antibody titers or develop a vaccine to prevent infection, it would be prudent to place exposed birds in single-bird households where they will have no direct or indirect contact with other birds. When provided with an easily digested, high-energy diet, a stress-free environment, and treatment for secondary bacterial and fungal infections, affected companion birds can survive for months or years.

Currently, no specific therapy for PDD exists; however, some clinicians report that birds with suspicious clinical changes respond favorably to interferon. The long-term prognosis in birds that do not respond to this therapy remains poor, with death occurring in affected birds from emaciation, secondary infections, autointoxication, or central nervous system disturbances. Until we can develop an effective way to control infection (probably by vaccination), preventive measures such as quarantine of new birds, avoiding direct or indirect contact between isolated groups of psittacine birds, and appropriate hygiene seem prudent.

References

1. Gregory CR, Ritchie BW, Latimer KS, et al. Proventricular dilatation disease. A viral epornitic. In press. Proc Annu Conf Assoc Avian Vet.
2. Gregory CR, Latimer KS, Campagnoli RP, Ritchie BW. Histologic evaluation of the crop for diagnosis of proventricular dilatation syndrome in psittacine birds. J Vet Diagn Invest 1996;8:76–80.
3. Ritchie BW. Avian viruses: function and control. Lake Worth, FL: Wingers, 1995.

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