

Epizootiology of Proventricular Dilatation Disease in Breeding Cockatiels

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Abstract: Proventricular dilatation disease (PDD) continues to cause morbidity and mortality among companion and aviary birds. Field observations have been used to suggest that the etiologic agent of PDD can be spread through horizontally and vertically. To evaluate issues related to the natural transmission of the PDD agent, breeding pairs of cockatiels in which at least one individual of each pair was histologically positive for PDD were placed in enclosures to facilitate breeding and both the adults and their offspring were followed to determine the rate of transmission in the population.

Introduction

Proventricular dilatation disease (PDD) is used to describe an inflammatory response characterized by the accumulation of lymphocytes and plasma cells in the central and peripheral nervous systems, especially the nerves that supply the muscles in the proventriculus and other digestive organs including crop, ventriculus and small intestine. Central nervous system signs associated with PDD, which may occur in addition to or independent of gastrointestinal signs, may include ataxia, abnormal head movements, seizures and proprioceptive or motor deficits.¹ The most common clinical signs of PDD include depression, weight loss (with or without decreased appetite), constant or intermittent regurgitation, and/or passage of undigested food in the feces indicating a malabsorptive or maldigestive disorder.¹

This disease was first discussed in the late 1970's in birds imported into the United States and Germany.^{1,2} Subsequently, a PDD epornitic has been occurring in psittacine birds in North America and Europe, probably as a result of the widespread importation and shipment of birds to satisfy the demands of the pet trade. There is no reference to spontaneous disease in free-ranging psittacine birds; however, they should be considered psittacine or non-psittacine birds susceptible. Given the severe nature of PDD and its potential to affect a wide range of bird species, the importation of psittacine birds or their eggs into any region with indigenous Psittaciformes must be considered extremely risky.

Etiology of PDD

Since its initial description in the late 1970's, multiple etiologies have been proposed for PDD; however, none have been proven to be the etiologic agent.¹⁻⁶ Adenovirus-like particles were demonstrated within intranuclear inclusion bodies in one affected bird. Paramyxovirus-like viral particles were demonstrated within inclusion bodies located in the neural cells of the spinal cord and in visceral nerve ganglia of another bird. Similar inclusion bodies have been described in the nerves of pigeons with paramyxovirus infections.² Birds with PDD have been shown to lack detectable levels of antibodies to paramyxovirus (serotypes 1, 2, 3, 4, 6 and 7), Pacheco's disease virus (an avian

herpesvirus), avian polyomavirus and avian encephalitis virus.¹ An eastern equine encephalomyelitis (EEE) virus was recovered from neonates with abdominal distention from an aviary with a history of PDD. The disease in these neonates was termed avian viral serositis. This finding was used to suggest that PDD may be caused by EEE virus,⁴ even though EEE virus occurs primarily in the eastern portion of the United States, and PDD has been shown to occur throughout the United States, Canada and Europe. Experimental and epizootologic findings suggests that EEE virus is not the cause of PDD.⁵

A paramyxovirus related to Hitchner B1 was recovered from birds with PDD. Antibodies to this virus could be detected using a virus specific ELISA, however, antibodies were not detected using standard hemagglutination-inhibition assays available for paramyxoviruses.^{6,7} Experimentally infected African grey parrots either died soon after inoculation or seroconverted and shed virus with morphologic characteristics suggestive of paramyxovirus in their excrement.⁶

Using electron microscopy, viruses with morphologic characteristics suggestive of paramyxovirus, enterovirus, coronavirus and reovirus have been detected in tissues, secretions or excretions from birds that have been either histologically positive for PDD or with gross distention of the proventriculus. None of these viruses have been consistently demonstrated in all birds with confirmed PDD.

Diagnosing PDD

Clinical laboratory findings in PDD-affected birds are inconsistent.¹ Survey and contrast radiographs are useful for demonstrating gastric dysfunction in suspect birds. Distention of the proventriculus and increased transit time of barium are common findings in chronically affected birds. The proventriculus of neonates is normally dilated, a condition which should not be misinterpreted as PDD. Ultrasonic examination may be used to demonstrate dilatation and impaction of the proventriculus. Endoscopic examination may show impaction, ulceration and dilatation of the proventriculus.^{1,8} Fluoroscopy has been used to demonstrate reduced gastric motility which can be an indication of PDD.⁸

A presumptive diagnosis of PDD often is based on historical information, clinical signs, and radiographic evidence of proventricular dilatation or dysfunction. However, the presence of characteristic histologic lesions in nervous tissues is necessary for a definitive diagnosis.^{1,2} In most cases, a post-mortem diagnosis is rendered when a complete set of tissues (including proventriculus, ventriculus and brain) are examined microscopically. In some suspect patients, it is possible to obtain a diagnosis before death by submitting a biopsy of the crop. In one study, histologic evaluation of a crop biopsy correctly diagnosed PDD in (68%) of positive birds⁹ and in another study the sensitivity of crop biopsy was 76%.¹⁰ Thus, a positive crop biopsy in a bird with suggestive clinical changes is of diagnostic value, but a negative crop biopsy in a bird with suggestive clinical changes does not rule out PDD. To increase the likelihood of histologic detection of PDD-associated lesions, practitioners should obtain full-thickness crop biopsies

containing **at least one large blood vessel** and its associated ganglia. Evaluating step sections of the biopsy sample may also increase the likelihood of detecting segmental lesions.

The presence of lymphoplasmacytic ganglioneuritis and variable clinical signs (GI only, CNS only or GI and CNS) has led several researchers to propose that PDD might be caused by more than one etiologic agent. Experimental transmission studies have demonstrated that the same virus containing inoculum can cause varying clinical changes even within birds of the same species. Additionally, a morphologically similar virus has been recovered from the tissues or excrement of birds that were diagnosed by crop biopsy with PDD, even though some of these naturally affected birds had predominately CNS signs, some had gastrointestinal signs and some had both.

It should be cautioned that PDD should not be diagnosed based on clinical changes or gross lesions, particularly in birds with predominately neurological signs. Paramyxovirus-3 has been isolated from several birds with neurologic signs suspected to be associated with PDD, and serology^a was effective in documenting these infections (Dr Judy St Leger, personal communication). We suggest that tissues (including the pancreas when paramyxovirus is suspected) from birds that die following a progressive neurologic disease be submitted for virus isolation (available through many state diagnostic laboratories or the Infectious Diseases Laboratory at the UGA College of Veterinary Medicine).

Epizootiology of PDD in Cockatiels

To evaluate the epizootiologic characteristics associated with naturally and experimentally induced disease, breeding pairs of cockatiels in which at least one individual of each pair was histologically positive for PDD were placed in a closed indoor room and provided nesting boxes. Most offspring were allowed to mature without interference while some were experimentally inoculated with tissue homogenates known to induce PDD. Adults and their offspring were monitored for clinical changes suggestive of PDD and birds that developed morbidity were euthanized and tissues were histologically evaluated. Results of this study confirm that some birds can be in direct contact with PDD positive birds for prolonged periods without developing disease, diseased adults can produce clinically normal offspring, chicks produced by positive parents are susceptible to disease, any immunity passed from a hen to her chicks is only transient and the period from exposure to the suspect PDD agent to development of overt clinical signs can be more than a year.

Prevention

Proventricular dilatation disease can occur in any aviary despite excellent hygiene, valid quarantine procedures and the absence of new additions to the flock. In some aviaries, numerous cases of PDD will occur simultaneously. In others, several affected birds may die, and the problem seemingly resolves, only to reappear 1 to 2 years later.^{1,4} In other cases, a single bird in a breeding pair may die, with no subsequent losses in the aviary even 4 to 5 years later. It is common for many birds exposed directly or indirectly to an affected bird to remain subclinical.

Mates, offspring or siblings of birds that are diagnosed microscopically with PDD should be considered at extra risk of developing the disease; however, they should not be euthanized. Many of the birds that are directly exposed to those with PDD never develop the disease, an

epizootiologic observation that was confirmed in the cockatiel study. Until appropriate preventative measures can be developed, it would be prudent to place exposed birds in isolation.

Provided with an easily digested high energy diet, a stress-free environment and treatment for secondary bacterial or fungal infections, affected companion birds can survive for months or years. Any bird with the disease that is being treated should be placed in strict isolation with no direct or indirect contact with other birds. Some birds with clinical changes suggestive of PDD have been reported to recover when provided supportive care. However, a positive diagnosis of this disease requires the demonstration of microscopic lesions in the nerves and none of the reported recoveries have been in birds confirmed to have PDD.

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Services or Products Mentioned in the Text

- a. Serologic screening for paramyxovirus, California Veterinary Diagnostic Laboratory, San Bernardino, CA, 909-383-4287.

References

1. Gregory CR (1995). Proventricular dilatation disease. In: Ritchie BW, ed. *Avian Viruses: Function and Control*. Lake Worth, FL, Wingers Publishing; 1995:439-448.
- 2.. Gerlach S. Macaw wasting disease- a 4 year study on clinical case history, epizootiology, analysis of species, diagnosis, microbiological and virological results. *Proc Europ Chap Assoc Avian Vet*. 1991;273-281.
3. Mannl A, Gerlach H, Leipold R. Neuropathic gastric dilatation in Psittaciformes. *Avian Dis*. 1987;31:214-221.
4. Gaskin JM, Homer B, Eskelund K. Preliminary findings in avian viral serositis. A newly recognized syndrome of psittacine birds. *J Assoc Avian Vet*. 1991;5:27-34.
5. Gregory CR, Latimer KS, Niagro FD, et al. Investigations of eastern equine encephalomyelitis virus as the causative agent of psittacine proventricular dilatation syndrome. *J Avian Med Sur*. 1997;11:187-193.
6. Grund C, Grimm F, Kusters J, et al. Serological studies on persistent PMV-1 infection associated with PDD. *Proc Assoc Avian Vet*. 1999;19-23.
7. Grund CH, Werner O, Gelderblom HR, et al. Avian paramyxovirus serotype 1 isolates from the spinal cord of parrots display a very low virulence. *J Vet Med B Infect Dis Vet Public Health*. 2002;49:445-51.
8. Taylor M, Dobson H, Hunter BD, et al. New research in psittacine gastrointestinal motility in normal and disease states. *Proc Assoc Avian Vet*. 1997;131-132.
9. Doolen M. A low risk diagnosis for neuropathic gastric dilatation. *Proc Assoc Avian Vet*. 1994;193-196.
10. Gregory CR, Latimer KS, Campagnoli RP, et al. Histologic evaluation of the crop for diagnosis of proventricular dilatation syndrome in psittacine birds. *J Vet Diag Invest*. 1996;8:76-80.