

## Avian Bornavirus in Clinical Practice



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Avian Bornavirus (ABV) is an enveloped single-strand RNA virus with worldwide distribution that is responsible for the condition we now know as Proventricular Dilatation Disease (PDD). This condition has been identified in psittacines since the 1970s, and it was originally described as “Macaw wasting disease.” The causative agent, ABV, was only recently identified in 2008, although many other questions regarding this condition still remain. While this disease has been identified in numerous types of birds, this article will be focusing on psittacines, or parrot species, as they are the most commonly seen birds in clinical practice.

### Proventricular Dilatation Disease

The description given to the disease process that is caused by ABV is itself somewhat controversial and contested. The pathology of the disease involves nonpurulent inflammation of peripheral nerves. While the autonomic nerves of the gastrointestinal (GI) tract are commonly affected, this inflammatory process can also affect the central nervous system, as well as other organs entirely, leading to symptoms that may not be related to the GI tract at all. The classic presentation of PDD is a bird that is weak and lethargic, often with a normal appetite. If handled, the owner may notice a decrease in weight, but often this is not noted until there is a significant change. The plumage may appear dull or tattered, due to chronic malnutrition. Feces may contain undigested seeds or other food material, and vomiting or regurgitation is often noted. Most of these symptoms are identified in the more advanced stages of the disease. Symptoms not related to the GI tract may include ataxia, lameness, blindness, or even seizures. These may be seen concurrent with, or



independent of GI changes. Sudden death has been reported, in the absence of clinical symptoms. This may be due to changes or irregularities in cardiac conductivity, although this has not been proven.

Signalement for PDD is quite varied and can include any psittacine, although African grey parrots, Amazons, cockatoos, macaws, and cockatiels appear overrepresented. No age predilection has been identified, although a study in the early 1990s found the average age of affected birds to be 3.8 years old. Latent periods are suspected to be possible, with lengths being quite variable. Single-housed birds have become symptomatic after years of no known contact with, or exposure to, infected individuals.

Differential diagnoses for confirmed proventricular dilatation are numerous and should be ruled out prior to making a diagnosis of PDD. A partial list should include bacterial or mycotic infections, particularly *Macrorhabdus ornithogaster*, neoplasia, parasitism, heavy metal toxicity, or a gastrointestinal outflow obstruction. While proventricular dilatation may be seen with these conditions, they do not typically cause thinning of the proventricular wall as PDD does. If changes to the wall are noted, they usually involve a thickening secondary to chronic inflammation.

## Transmission

Viral RNA has been identified in several bodily fluids and materials, including feces, urine, tears, and oral secretions. Due to these findings, transmission has been assumed to be fecal-oral route. Several studies have found varying degrees of success with this hypothesis. Infected birds introduced into “clean” flocks resulted in spread of the disease to some, but not all, birds. Other studies have shown that horizontal transmission through direct contact was not a sufficient route in immunocompetent birds. Often these patients will test positive for the virus on feathers, or skin biopsies, but will never seroconvert. Due to these findings, Piepenbring et al concluded that exposure most likely did not achieve persistent infection. This hypothesis has been strengthened by the fact that multiple reports have identified infected and non-infected birds living together for years, with no transmission.



At the time of this publication, transmission is not fully understood. Both horizontal and vertical transmission appear possible, but several variables are thought to affect each of these, including individual health of the patient, genotype of the virus, and any possible co-infections.

## Diagnosis

Diagnosis of a clinical ABV infection is, in a word, challenging. Initial detection of the virus in the living patient is typically done through reverse-transcription polymerase chain reaction (RT-PCR) testing. Samples submitted include cloacal/choanal swabs, tissue, and whole blood. Submitting a swab from only one site, or blood without accompanying swabs, dramatically reduced the likelihood of a positive sample. It is recommended to combine swabs and blood into one tube, although individual labs may have different submission requirements. Less commonly run tests to identify ABV include immunohistologic staining, and viral isolation. ABV-specific antibody detection (ELISA, Western blot) may provide valuable information for diagnosis, although at the present time it is unclear when antibodies are produced, or if they can potentially be correlated to clinical signs.

Another factor that adds even more confusion is the fact that some birds become infected with ABV, shed the virus, but never seroconvert. Several theories for why this may happen have been introduced, although none have been proven. Based on this information, the following protocols have been proposed to help identify positive and negative birds. A bird that is found to be positive for ABV using both PCR and serology should be considered a positive infection. A bird that is positive on PCR, but negative on serology, should be retested in 4-6 weeks to check

for a false positive PCR and also to monitor for seroconversion. Repeated positive PCR tests, and/or seroconversion should be considered a positive bird. Negative PCR paired with a positive serology are thought to be carriers, although this has not been proven. Persistent elevated titers are indicative of infection in mammals infected with bornavirus, and should be considered the same for birds. Some birds appear to be able to clear the infection, which would explain the positive serology and negative PCR, although this too has yet to be proven.

## Treatment

Several therapies have been proposed for treatment of both the symptoms of ABV, as well as for the virus itself. To date, there are no successful antiviral therapies reported. Previous reports of nonsteroidal anti-inflammatory drugs have failed to show significant, repeatable success. One study using meloxicam in infected cockatiels actually showed more severe lesions, and increased mortality, when compared to the control group. Immunosuppressive treatments, including the use of glucocorticosteroids, have been proposed at recent conferences. No published data has been made available as of yet, and clinicians should be cautious of these currently unproven methods. Symptomatic treatment, including diet modification, analgesia, and control of secondary infections should be the basis of treatment for these cases.

## Conclusion

Despite the recent advances in identifying and diagnosing this condition, there are still many questions left unanswered. While diagnostics have improved considerably, interpretation of these tests can still be challenging. A positive ABV diagnosis should not result in euthanasia, especially since many patients who test positive will never show symptoms. That being said, once patients become symptomatic, the chances of long term survival are very low, and quality of life should be assessed. Along with the discovery of additional symptoms, not related to the GI tract, some have proposed a name change from proventricular dilatation disease to avian or neuropathic ganglioneuritis. While this more accurately describes the condition, a name change has not been uniformly agreed upon at this time.

## References and Additional Resources:

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