Proventricular Dilatation Disease

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Introduction
Proventricular Dilatation Disease (PDD) is predominantly a disease of pet psittacines and is a common cause of morbidity and mortality in parrots in the UK and elsewhere. Species other than psittacines have also been affected and the role of wildlife acting as a reservoir is still being investigated. PDD is a fatal neurologic disease that uniquely affects the enteric and on occasions peripheral and central nervous system.

Undigested seed in the faeces is a common clinical sign in PDD cases
Clinical Features

Avian bornavirus (ABV) has been identified as the cause of proventricular dilatation disease (PDD) in psittacines. The clinical signs relate to an immune mediated reaction to the virus. Gangliocides are produced which cause changes to the nerves which leads to disease. Many healthy birds are infected with ABV, but the development of PDD in such cases is unpredictable and not fully understood. It is currently thought that approximately 30% of bornovirus positive birds will go on to develop clinical PDD. It can take between as little as two weeks and potentially up to seven years or more for an infected bird to start showing clinical disease. Detecting ABV in a sick bird is not proof that it is suffering from PDD and detecting ABV in a healthy bird does not indicate that it will become sick. ABV is not restricted to psittacines and a PDD-like disease has been diagnosed in canaries. Bornavirus has been shown to have a high prevalence in North American waterfowl however no evidence has been produced showing that these waterfowl genotypes can cause disease in psittacines.

The faecal/urate oral route of disease transmission is considered the most significant. The respiratory tract has also been suggested as a route of transmission. ABV has been isolated from the lung of infected birds and high volume air sampling has detected ABV in the air of infected aviary environments. ABV has also been detected in feather calami and feather dander. Bornavirus however does not survive for more than 48 hours outside the birds body. Initial clinical signs are generally non-specific. Birds present as lethargic, and being off-colour. Insidious weight loss if often noticed and undigested food can occasionally be found in the faeces. Crop impaction, delayed crop emptying, vomiting, regurgitation and coelomic distension are the most common GI signs. Birds often have a good appetite but occasionally anorexia is observed. Some bird will be seen to pluck feathers over the coelom. In a small percentage of cases acute peripheral and central nervous system signs are seen including; blindness, fits, seizures and falling from the perch.

Within a collection of birds isolated cases may be diagnosed but equally epidemics of infection affecting a large percentage of the collection can occur with a fast progression of disease from birds being acutely ill to death within 11 days. Other birds will show a more insidious disease progression with gradual weight loss and gastrointestinal signs. Some birds in the collection will be asymptomatic but if endoscopy or radiography is performed will have mild/moderate signs of proventricular dilation. Other birds within the collection will be unaffected.

Cockatoos, grey parrots and macaws appear most susceptible to the disease. However disease has been seen in over 50 species of bird and all psittacines must be considered susceptible. All age groups can be affected. Incubation period is from as little as 11 days to >7+ years. The disease is not considered to be highly infectious and the pathogen is labile – not surviving outside the host longer then 48hours.
Proventricular dilatation disease is difficult to definitively diagnose. It is straightforward to diagnose a dilated proventriculus using common imaging techniques (radiography, endoscopy, fluoroscopy) however proving the dilation is due to a bornovirus infection is in our experience difficult. Clinical signs are non-specific.

If a clinician suspects PDD then radiographs (ideally with contrast instilled in the proventriculus in the anaesthetised bird) or fluoroscopy can help detect proventricular dilation. Dilation of the proventriculus is present if the depth of the proventriculus is greater than 48% of the greatest depth of the carina of the sternum when viewed on a lateral projection. Traditionally a crop biopsy was performed (examined for signs of myenteric ganglioneuritis) as part of the diagnostic protocol however this test is only 55-76% sensitive.

Dilated proventriculus seen on a lateral radiograph – the proventriculus depth is 85% that of the sternal carina – significantly larger than the normal 48%.
Radiograph showing a left sided coelomic distension indicative of a proventricular dilation.
**Barium Contrast Instilled under anaesthetic allows more accurate measurement of the proventriculus**

**Ventral-dorsal radiograph with contrast showing a marked proventricular dilation.**

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**Diagnosis**
To make an informed diagnosis of PDD a clinician should rule out potential differential diagnosis and obtain evidence of ABV infection. It is however important to remember that many clinically healthy birds shed ABV adding to the challenge of making an accurate ante-mortem diagnosis.

Other common causes of proventricular dilation include:
- Gastritis/enteritis
- Parasitic disease
- Heavy metal toxicity (zinc/lead etc)
- Neoplasia
- Foreign body/obstruction

Studies at Texas A&M have suggested reverse transcriptase–PCR as an efficient method of determining the presence of ABV RNA (virus detection). Four serotypes of ABV are currently recognised and many laboratories do not currently test for all serotypes. The selection of appropriate samples for PCR testing is vital to obtaining a meaningful result. Urates/faeces as well as conjunctival, choanal and cloacal swabs are the most appropriate samples to be collected. ABV is shed in greatest volumes in urates and faeces, however shedding is intermittent and false negatives are therefore a problem. Multiple PCR tests may be required in the diagnoses of the disease, which is potentially costly for the owner. Serology (testing for antibodies) has the potential of being a useful diagnostic test as there appears to be correlation between antibody titres and disease development. Also sudden sero conversion has been seen to occur just prior to the onset of clinical disease.

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Fluoroscopy is a useful tool to assess gastrointestinal motility and to monitor response to treatment. Unlike most radiographs no anaesthetic is required.

**Treatment**

Treatment has historically been based around the use of anti-inflammatory medication. This was hypothesised based on the observed histopathologic lesions being inflammatory in nature. The authors’ treatment of choice is Celocoxib (Dalhausen et al 2002). Birds were treated for 6 to 12 weeks with Celocoxib at 10mg/kg BID and showed marked clinical improvement. Fluoroscopy can be used to monitor treatment success and the dose of Celocoxib titrated down to the minimum affective dose. Many birds that first present with PDD have secondary bacterial/fungal crop infections and/or enteritis which when treated lead to significant clinical improvement.

Many authors have progressed to using meloxicam however a recent study using this drug demonstrates it may be contraindicated. Early trials with alternative drugs including Ribavirin (antiviral) and Cyclosporine have not been encouraging.

**Preventing Disease**

Preventing disease in collections is based around good hygiene and biosecurity. Any new birds, sick birds or ABV-positive birds should be isolated and quarantined. Healthy ABV-negative birds should be visited first and traffic from infected/ill birds to the rest of the facility should be avoided. In an effort to
maintain a disease free flock all newly presenting birds should all be tested using multiple PCR tests together with serology.

If it is the owners aim to eradicate ABV from there collection all birds should be tested using repeated PCR tests and serology. Birds should be grouped and if required isolated based on the results of this testing. It has been reported that within a positive group of birds a small number of infected individuals are persistent high-level shedders of the virus. These birds should be targeted and removed as a priority. Due to the intermittent shedding and inconclusive serology testing, testing and separating birds may require years to obtain ABV negative aviaries.
References: