Psittacine Proventricular Dilation – a disease all keepers should be aware of

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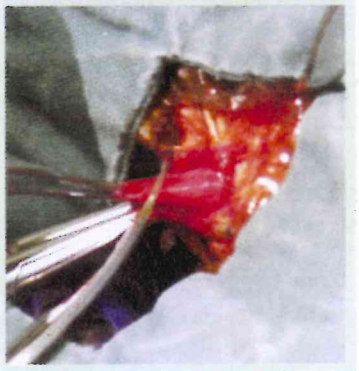
Proventricular Dilitation Disease, also known as Macaw Wasting Syndrome, Psittacine Wasting Syndrome, Psittacine Proventricular Dilation Disease, Myenteric Ganglioneuritis, Proventricular Hypertrophy, Infiltrative Splancnic Neuropathy, Lymphoplasmacytic and Encephalomyelitis, was first diagnosed in 1977 and is a very complex disease.

PDD is predominantly a disease of captive psittacine birds and is a common cause of illness and death of UK psittacines. Other species have been affected though and avian wildlife populations may be a reservoir for infection.

Birds may present just as sick, off-colour parrots, some may show gradual weight-loss and on occasions the passage of undigested seed in the faeces will be found. A small percentage will present with acute onset central or peripheral nervous signs, including blindness, fits, seizures and falling from the perch.

Undigested seed in the faeces – whilst pathognomonic, it remains a rare presentation.

Individual birds may be affected but equally infection 'storms' (epidemics) may affect collections with a percentage of birds being acutely ill and dying within 11 days of infection. Others show gradual weight loss and gastro intestinal signs, usually developing at least three months after exposure to the infective agent.



Collection of crop biopsy for histological examination of nerve tissue for evidence of characteristic nerve changes.

Other birds will show no clinical signs, but if radiographed or an endoscopy is performed, it will show mild to moderate proventricular dilation. These birds may survive but they never regain full gut normality. Other birds in the collection will be unaffected.

Incubation period 11 days to 7 years +

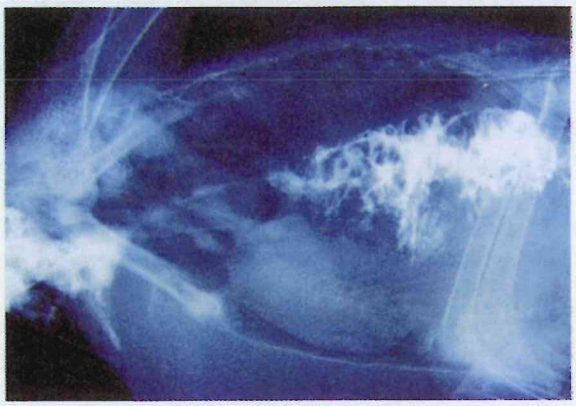
Cockatoos, African Greys and macaws are most commonly affected by this disease, but any psittacine species should be considered to be at risk - the disease having been proven in over 50 different psittacine species and birds of all ages are vulnerable.

Consistent gross post mortem and histologic lesions have also been reported in toucans, honey creepers, canaries, weaver finches, roseate spoonbills and free-living Canada geese.

Incubation periods are variable from 11 days to 7 years or more. The disease is not considered to be highly infectious, and the pathogen does not survive outside the host for more than 72 hours. Signs of infection may include an acute sick bird, or more chronic signs such as crop impaction, vomiting, slow crop emptying, abdominal distension, progressive weight loss (often despite a good appetite in food consumption) and central nervous signs.

The passing of undigested seeds in the faeces is observed in a minority of cases, in appetence, or just a bird which is not in very good condition.

A veterinary diagnosis is made based on history within a collection, vomiting, undigested seed in faeces, progressive weight-loss, neurologic signs, radiography (proventricular dilation or delayed gut transit times). A barium contrast radiograph or fluoroscopy may assist in diagnosis. Diagnostic confirmation may be achieved with a crop biopsy, although even this test is only 55-76 per cent sensitive, i.e. in 100 affected birds a positive test result will only be found in 55-76 cases.



Barium contrast proventricular radiography of PDD positive case.

More recently, radiography (if necessary with barium contrast), has been shown to be a relatively sensitive diagnostic test for proventriclar dilation (if the proventricular depth is more than 48 per cent of the greatest depth of the carina of the sternum).

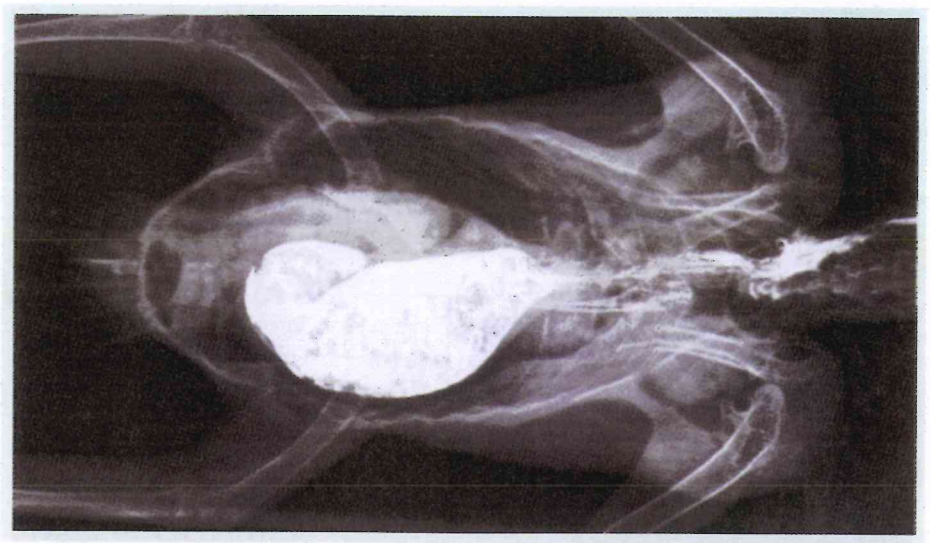
Gene-based fingerprints and serological tests are now also available for Borna virus, although results still appear inconsistent.

Other differential diagnosis for PDD include, foreign body ingestion, heavy metal (Pb, Zn, Cu, Fe) poisoning, GIT parasitism, bacterial or fungal (e.g. candida or macrorhabdus ornithogaster) infection.

Disease progression

Many confirmed clinical cases respond well to Cox-2 specific NSAID medication (due to reduction of the immune response to Borna virus). Celecoxib was the drug first recommended, although many clinicians are using meloxicam with apparent success. Intermittent gut active antibiosis may be required to control infections arising due to increased gut transit times.

Initial advice was to treat affected birds for 6-8 weeks, then cease NSAID therapy, however many such cases immediately reoccur. It is important not to use NSAID for longer or at higher doses than necessary, in view of the potential



DV Radiographic view showing barium contrast, proventricular dilation, with asymmetry of the coeliomic outline with lateral distension on the patient's left side.

gastric and renal side effects.

Birds on long term therapy will benefit from periodic renal parameter evaluation. My experience is that owner feedback in respect of response to therapy is frequently misleading and frequently leads to erroneous decisions in respect of onward medical treatment. Barium contrast radiography, or better still conscious fluoroscopy, give a much better indication of current proventricular size and more importantly peristaltic activity.

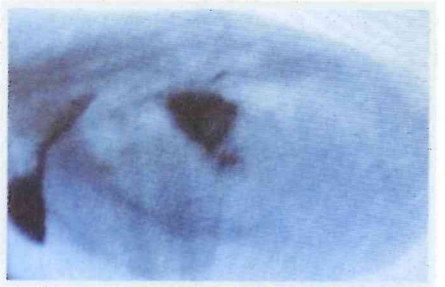
Regular small meals of soft high energy, readily digestible high fibre gruel or pelleted foods, a stress-free environment and control of secondary infections (which occur intermittently due to delayed gut transit times) are all recommended. In such cases some birds appear to make a clinical recovery, others survive for many years, but never regain full normal gut peristaltic activity.

What remains unknown is how long an infected bird is viraemic and hence potentially infectious for. Any proven and clinically affected bird must be considered to be a potential infectious risk to other birds and kept in absolute isolation.

Secondary infections should be controlled with antibiotics and anti-fungal agents and gut function is maximised with prokinetics and NSAID as indicated.

In contact birds, should be kept isolated for at least one year following disease of their mate, even so this may be insufficient to allow detection of all latent cases (incubation is stated to extend to 7+ years). In contact birds should not however be euthanised as many never develop clinical disease.

In an aviary situation, after the loss of a number of confirmed cases over a short period, even after an interval of 1-2 years with no signs of disease, the disease can suddenly reoccur.



Fluoroscopic view showing barium in the crop and the dilated proventriculus.

Prevention

PDD can occur in any aviary despite excellent hygiene, effective quarantine precautions and the absence of new additions to the flock. There have been suggestions that infection may be spread on occasions from feral birds visiting the aviary, indeed avian wildlife populations may act as reservoirs of infection. There is however no doubt, that the more new birds that are introduced into a collection the more inevitable it is that you will introduce this infectious disease.

Post Mortem examination showing marked proventricular dilation.

Screening of new birds is challenging due to the potentially protracted incubation period. Lateral radiographs looking for evidence of proventricular enlargement may be useful, but will not detect infected, but currently sub-clinical cases. Borna virus serology or viral finger print tests may sound attractive, but results are currently inconsistent. ■

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