Proventricular Dilatation Disease – the Situation in Australia

Bob Doneley

Introduction

In the late 1970's and 1980's reports emerged of a wasting syndrome in macaws and other parrot species in North America and Europe. Known variously as Macaw Wasting Disease, Macaw Fading Syndrome, Myenteric Ganglioneuritis, Infiltrative Splanchnic Neuropathy, Neuropathic Gastric Dilatation, and Proventricular Dilatation Syndrome, it spread at an alarming rate. It is now well established as a disease of captive psittacine birds in the United States, Canada, the United Kingdom and Europe. The only previous report in Australia was in a legally imported Green Wing Macaw (*Ara chloroptera*) in 1993.[1]

Now known as Proventricular Dilatation Disease (PDD), it is characterised by a non-suppurative lymphocytic, plasmocytic ganglioneuritis of central and peripheral nerve tissue.[2] It appears to be a segmental neuropathy, with clinical signs dependent on the organs affected. As well as the classical syndrome of weight loss associated with regurgitation and the passage of undigested food in the faeces, other clinical signs also include ataxia, abnormal head movements, progressive paresis, proprioceptive deficits, anorexia, lethargy and, occasionally, sudden death.[3]

A variety of aetiological agents have been proposed, including paramyxovirus, togavirus, adenovirus, and Eastern Equine Encephalitis virus. Other suggested aetiologies included immune-mediated reactions to an unknown viral agent. However, no consistent viral isolation or serological findings have confirmed the involvement of any one virus. Ritchie[3] (USA), Taylor (Canada, personal communication) and Gough[4] (UK) have all reported the presence of 80-140 nm pleomorphic enveloped viral-like particles in fresh faeces from affected birds. Additionally, similar viral-like particles have been detected in the cytoplasms of cells in the brain of an affected bird.[5] Ritchie was also able to transmit the disease by exposing susceptible birds to a tissue homogenate containing these viral-like particles. To date, this viral-like agent has not been identified and classified; thus a test has not been developed for its detection.

At the time of writing, diagnosis of PDD is made on the basis of the history, clinical signs, radiological and fluoroscopic imaging, and detection of the characteristic ganglioneuritis in biopsy samples. Radiology and fluoroscopy will often reveal a flaccid, enlarged and poorly functioning proventriculus.[3] Ante-mortem biopsy of the crop, proventriculus and adrenal gland have been reported as being highly specific for PDD, but with a low sensitivity.[2]

Over the last 18 months the author has diagnosed five confirmed cases of PDD in south-east Queensland, and another suspect case.

Species	Age	Weight loss	Neurological signs	Gastrointestinal signs	Laboratory Confirmation
Eclectus	6 yrs	✓	✓	-	✓
Eclectus	6 yrs	✓	-	-	✓
Moluccan cockatoo	7 mnths	✓	-	✓	✓
Sun Conure	6 yrs	✓	✓	✓	✓
African Grey	14 wks	✓	-	✓	✓
Moluccan Cockatoo	7 mnths	✓	✓	-	-

Clinical signs exhibited by these patients were:

Eclectus hen	weight loss, polyuria/polydipsia, loss of proprioception
Eclectus cock	weight loss
Moluccan cockatoo	weight loss, anorexia
Sun Conure	seed in droppings, tremor, unable to perch
African Grey	presented dead after 1 week history of weight loss and anorexia
Moluccan cockatoo	unable to walk, foot clenching, weight loss.

All birds had evidence of proventricular dilatation, either radiographically or on gross autopsy.

Histologically, all the birds had a lymphocytic-plasmocytic ganglioneuritis, although the degree of severity varied. Sections from several birds were sent to Bob Schmidt (USA) for a second opinion: he confirmed probable PDD in some and definite PDD in others. Serositis was also present in two cases.

Discussion

The only report of PDD in Australia was in an introduced green winged macaw in 1993.[1] These cases, and several others seen by other veterinarians (Gallagher; Black: personal communications), represent convincing evidence of a disease not previously seen in parrots bred in Australia. As such, PDD can no longer be regarded as an 'exotic' disease.

The source of infection in these cases is difficult, if not impossible, to establish. The four cases described above came from four separate geographical areas, the owners of the birds were unknown to each other except by name, and there was no obvious connection between the birds themselves (breeders, hand rearing facilities, etc). How the disease was introduced into Australia is also a matter of conjecture. Anecdotal reports of bird smuggling into Australia indicate that eggs, rather than live birds, are smuggled in. It is still unclear if PDD is egg-transmitted. It would be premature at this time to assign responsibility for this disease's introduction into Australia solely to bird smugglers.

PDD is usually regarded as a psittacine disease and in the America aviculture industry, African grey parrots, macaws, Amazon parrots and cockatoos are the most commonly affected species.[2] However, there have been reports of its occurrence in other species. Suggestive lesions have been described in toucans, honey-creepers, canaries, weaver finches, Canada geese, and roseate

spoonbills.[6] A recent report[7] also described its occurrence in a Peregrine falcon. These reports of the disease's occurrence in other species raise questions about both the introduction by and spread of this disease by/to wild birds, and the potential for its impact on Australia's avi-fauna. This disease appears to be particularly infectious in aviary situations with poor hygiene, inadequate ventilation and uncontrolled traffic flow; it is less common in better managed aviaries. This, along with the fact that enveloped viruses are rarely hardy outside their host, suggests that a pandemic situation is unlikely to occur (Taylor, personal communication).

Veterinarians presented with birds showing neurological and/or gastrointestinal signs need to add PDD to their list of differential diagnoses. Once other possibilities, such as lead toxicosis, have been excluded, a crop biopsy (that includes a major blood vessel) may provide diagnostic information. The suspicion of PDD must be communicated to the pathologist as the segmental nature of this disease means that stepped tissue sections may be needed to detect the lesions. Crop biopsy has been reported to be an effective method of antemortem diagnosis although some experienced diagnostic pathology practices claim only 30-35% diagnostic success.[2] If the bird dies, or is presented dead, a wide range of tissues should be submitted for histopathology including crop, proventriculus, ventriculus, duodenum, intestinal tract, adrenal gland, spleen, brain and perhaps spinal cord.

Although all cases presented here had lesions typical of PDD, 2 of the cases (Case 1 and Case 4) had evidence of concurrent polyserositis. Polyserositis is not typical of classical PDD and raised the possibility that Australian strains of the disease have slightly different histological presentation from American strains (Schmidt, personal communication).

In recent years the treatment of individual birds with non-steroidal anti-inflammatory drugs such as celecoxib and meloxicam has been advocated and, in many cases, proven to be apparently successful (Dahlhausen, personal communication). Combined with the reduction of environmental stress, fluids, gastrointestinal motility enhancers and antimicrobial therapy as indicted, many affected birds have returned to normal. However, it is still unclear as to whether these birds have cleared the pathogen from their body or if they have been converted to asymptomatic carriers. Until this is ascertained, such birds should be regarded as potentially infectious and isolated from other birds.

Until the aetiological agent is positively identified it is not possible to comment authoratively on routes of transmission and incubation periods. It is likely that faeco-oral transmission is the most probable route of transmission, but aerosol or egg transmission cannot be excluded. Experimental and anecdotal evidence suggests that the incubation period could be as short as several weeks or as long as several years.

Australian veterinarians may well, in the very near future, be faced with the prospect of advising an aviculturist on the management of their collection after one of their birds is diagnosed with PDD. Until the causative agent and its transmission are clearly determined, and an effective diagnostic screening method developed, it is difficult to give specific advice. Ritchie[4] states that in-contact birds are often not affected. It therefore follows that the best advice that may be given at this time is to place in-contact birds in strict isolation and to institute sound management techniques including good hygiene, traffic control measures and the immediate and thorough investigation of any sick or dead birds. Speer (personal communication) recommends an isolation period of 2-3 years without any fresh incidence of PDD before declaring an aviary truly free of the disease. Veterinarians in this situation must be mindful that their client's reputation, and possibly their income, is dependent on how they handle the confidentiality issues associated with a diagnosis of PDD. This must be

balanced against the 'greater good' of dealing with a disease that has the potential to have a devastating impact on aviculture and Australia's native avi-fauna. PDD is not, as yet, a notifiable disease in Australia and veterinarians and their clients should not expect any government assistance or funding in the detection and eradication of this disease.

References

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