Adenoviral encephalitis in a Princess Parrot (*Polytelis alexandrae*)

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Abstract

Adenoviruses have been isolated from psittacine birds worldwide, but reports in Australian literature are few. Most infected birds are asymptomatic, and it is believed that some form of immunosuppression or concomitant disease is necessary for the infection to become clinically apparent. This paper reports a case of a Princess Parrot (*P. alexandrae*) with concurrent Psittacine Circoviral Disease (PCD) and adenoviral infection.

Case Report

A mature blue mutation female Princess Parrot (*P. alexandrae*) was presented for a sudden onset of ataxia and tremors. On examination, the bird was in good physical condition, but had the white (instead of blue), dystrophic feathers typical of PCD in this species and colour. The bird was alert, but was unable to fly or stand properly, and used its outspread, dropped wings to maintain a standing posture.

Differential diagnoses at this stage included heavy metal toxicity, encephalitis (bacterial, viral or fungal), head trauma, cerebro-vascular accident, or an encephalopathy of unknown origin. Treatment was begun with calcium disodium versanate (Calsenate, Parnell), enrofloxacin, and dexamethasone. Faecal examination revealed ascarid eggs, and the bird was treated with moxidectin (*Cydectin*).

After 36 hours without any response, and given the likelihood of concurrent PCD, the bird was euthanased and necropsied. There were no significant gross changes, and tissues were submitted in formalin for histopathology.

Newly erupted feathers showed inflammation of the pulp cavity with irregular densely basophilic inclusions in some cells, strongly suggestive of PCD and confirming the author's clinical impression.

Brain sections had mild multifocal encephalomalacia with some mineralisation. Occasional vascular endothelial nuclei had large, lightly basophilic inclusions. Similar inclusions were seen in endothelial nuclei in the heart, and in the nuclei of epithelial cells lining the collecting tubules in the kidney. Transmission electron microscopy of formalin fixed kidney revealed that the intranuclear inclusions contained numerous non-enveloped, virus-like particles. The size and shape of these particles was similar to those seen in known adenoviral infections.

Based on these findings, a diagnosis was made of adenoviral infection secondary to PCD.

In-contact birds were physically examined for evidence of PCD, but were normal, and remain so at the time of writing (5 months after diagnosis).

Discussion

Adenoviruses are non-enveloped, 65 – 90 nm double stranded DNA viruses.[1, 2, 3] The Adenoviridae family consists of two genera: Mastadenovirus (mammalian strains) and Aviadenovirus (avian strains).[2] Aviadenoviruses can be divided into 3 groups according to common group antigens. These groups are:

Group 1.	Fowl Adenovirus (FAV)	12 serotypes isolated from chickens, turkeys, pigeons, Budgerigars, Mallard Ducks, guineafowl, pheasants, geese and Muscovy Ducks.

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Group 2.	Turkey haemorrhagic enteritis virus, marble spleen disease virus, chicken splenomegaly virus.
Group 3.	Infectious salpingitis in galliforme birds (egg drop syndrome)[2]

Adenoviruses are frequently isolated from persistently infected, but asymptomatic, birds.[3] The virus is excreted mainly in the faeces, and transmission is known to be through the oral route, although inhalation is also suspected. Egg transmission also occurs in many species, but its occurrence in psittacine birds is unclear. [1, 2] Morbidity and mortality rates appear to vary with the host and the strain of virus. In psittacine birds, age does not appear to be an important factor, with all ages being susceptible. Virulence of some strains appears to be greater in non-adapted hosts than it is in more typical hosts. [3] Adenovirus is generally considered to be an opportunistic pathogen. Often, infections are asymptomatic unless a state of immunosuppression exists. Affected birds have been shown to carry concomitant polyomavirus, chlamydial, fungal, bacterial or parasitic infections. However, some highly virulent strains can produce disease alone, or may trigger secondary infections through producing mild lesions without clinical signs. [2]

There have been many reports of adenoviral infections in psittacine birds, although reports of neurological disease are rare. Typically, syndromes including enteritis, hepatitis, conjunctivitis, renal disease, and pancreatitis are reported, especially in *Agapornis spp*. Neurological signs were reported in an outbreak in a flock of Budgerigars, with affected birds showing acute torticollis, opisthotonus, tremor and convulsions. *Pionus spp*. and *Neophema spp*. have been described with persistent torticollis and other CNS signs. A Moluccan Cockatoo with progressive neurological disease (ataxia, tremors, head pressing and eventually death) was diagnosed with adenoviral infection. [2]

Diagnosis of adenoviral infection is best achieved through viral culture and isolation, or DNA probes. Gross pathology and even histopathology are not sufficient to differentiate inclusion bodies due to adenovirus, polyomavirus or even circovirus. In this case, transmission electron microscopy was used to give a final diagnosis. Antemortem tests include ELISA and agar-gel diffusion tests, and haemagglutination-inhibition tests. It should be noted that, as adenovirus can be isolated from asymptomatic birds, its presence does not confirm its involvement in a disease process. [3]

In all likelihood, the Princess Parrot described in this case report was immunosuppressed by the concurrent circoviral infection, well known for its affects on the immune system. The presence of adenoviral-like inclusions in the cerebral blood vessels, with associated encephalomalacia, makes secondary adenoviral encephalitis a likely diagnosis.

Australian clinicians need to add adenovirus to their list of differential diagnoses for birds showing neurological disorders, particularly if affected birds have concurrent, potentially immunosuppressive, conditions.

References

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