Circoviral Disease in Wild Australian Psittacines

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Abstract

Circovirus is the causative agent of psittacine beak and feather disease in psittacine birds. It causes a chronic, un-treatable and debilitating disease characterised by feather loss, elongation of the upper and lower beaks and immunosuppression eventually resulting in death. This paper will look at beak and feather disease from a wildlife care perspective and provide background information on the virus, its lifecycle and epidemiology and discuss prevention and control.

Keywords: Circovirus, PBFDV, beak and feather disease, psittacine

Introduction

Psittacine circovirus is a 14-16nm non-enveloped icosahedral DNA virus belonging to the family Circoviridae. It is the causative agent for psittacine beak and feather disease virus (PBFDV), a highly infectious and un-treatable disease of psittacine wildlife in Australia. The virus was first reported in sulphur-crested cockatoos (*Cacatua galerita*) in Australia in 1903. Since then, it has been shown to have a worldwide distribution and to affect greater than 60 species of captive and free-living psittacine species (Raidal 2012, Ha et al. 2007).

Epidemiology

Outside of the bird, the virus is exceptionally resistant to desiccation, and remains infective for months or even years in the right environment. Transmission occurs horizontally; via inhalation, ingestion or via the cloaca in the nest box with bursal absorption (Phalen 2006). Infected birds shed the virus in their faeces, crop secretions and feather dander (Ritchie et al. 2003; Phalen 2006). Contaminated fomites (clothes, feeding equipment, carry cages, aviaries etc) can cause indirect horizontal transmission. Nesting hollows provide a suitable environment for heavy viral loads to persist year on year, with faeces, feather dander and crop secretions carrying high viral loads, because of their immature immune capacity, juvenile birds are the most at risk age group (Phalen 2006). There have been episodes of viral DNA being detected in embryonated eggs, suggesting the possibility of vertical transmission (from parent to offspring before birth).

Pathogenesis and Clinical Signs

Once inside the bird, virus replication occurs in the bursa of Fabricus, thymus, gastro-intestinal associated lymphoid tissue, and circulating lymphocytes (Ritchie et al. 1989; Phalen 2006). Ultimately the virus targets the epidermis and feather follicles (Raidal 2012).

Three forms of psittacine beak and feather disease (PBFD) occur:

Peracute disease occurs in neonates resulting in acute death.

Acute

Disease is usually seen in young or fledgling birds during their first feather formation and causes death in one to two weeks. Prior to death, nestlings may be depressed, regurgitate, develop green diarrhoea and may be systemically ill (Greenacre 2005; Raidal 2012). Newly developing feathers will have annular constrictions (pinching of the feather shaft) and they break easily and are easily pulled out (Phalen 2006).

Chronic

Chronic PBFD usually occurs in birds aged six to 12 months undergoing their first adult moult but can also be seen in older individuals. Members of the cockatoo (*Cacatua* spp.) family and the galah (*Eolophus roseicapilla*) are the most diverse in their clinical presentations (Phalen 2006). Clinical signs in these species will begin with the birds producing a smaller amount of powder down, the beak and toes will become dark and shiny. The powder down feathers beneath the wing on the lateral body walls will become short and stumpy and will no longer produce the characteristic powder. Feather abnormalities will follow with primary flights, tail feathers and body contour feathers all affected. Initially often the crest feathers are affected. The feathers will show pinching of the calamus (quill) often with blood in the feather shaft. The feathers may be discoloured (brown) or dull and this is attributed to the lack of powder down. As the disease process becomes chronic, the beak and claws will become elongated, fragile and break easily. Secondary to these beak changes the birds may have difficulty prehending and eating food and often rely heavily on supplementary feeding by members of the public.

Some birds, especially rainbow lorikeets (*Trichoglossus moluccanus*), can be latent carriers shedding virus while appearing clinically normal (Raidal 2008). In PBFDV infected wild rainbow lorikeets, approximately one third will lose their primary flights and never grow them back, one third die before their first molt and the remainder develop normal feathers after their first moult and can become latent carriers (Phalen 2006). Polyomavirus affects rainbow lorikeets with identical clinical signs as described above. For this reason, the distinction between PBFDV infected birds based on clinical signs is exceptionally difficult. A similar difficulty is faced in budgerigars (*Mellopsittacus undulatus*) that are affected by both viruses, with molecular diagnostic testing the only way to differentiate the disease syndromes (Phalen 2006). Affected parrots often succumb to secondary infections because of the immunosuppressive effects of the virus (Ritchie et al. 2003). Death generally occurs six months to two years after the onset of clinical signs due to the immunosuppressive nature of the infection.

Many other species will develop characteristic feather abnormalities or they may only develop feather discolouration with feather shafts that allow the feather to be easily plucked (Raidal 2012). Various other species that are not psittacines (*Corvidae* spp., *Coraciiformes* spp. etc) can also be infected with circovirus with characteristic feather loss and immunosuppression causing disease.

Diagnosis

Veterinarians can often reach a presumptive diagnosis based on clinical signs (Phalen 2006). If a diagnosis is sought anti-mortem, serological methods (haemagglutination assay (HA), haemagglutination inhibition (HI) assay) can be carried out on feather, blood and faecal samples. A PCR assay on heparinised blood will often enable confirmation of viral infection (Phalen 2006). If a clinically affected bird is submitted for post mortem examination, histopathological examination will reveal large basophilic intranuclear and intracytoplasmic

inclusion bodies within macrophages, keratinocytes, hepatocytes and other tissues (Raidal 2012). The cost for PBFDV testing with PCR, HI and HA testing is approximately $350, the cost for histopathology is similar. For this reason, it is very uncommon for PBFDV testing to be carried out in wild birds (these costs must be bourne by the veterinary clinic sending samples away).

Prevention and Control

Psittacine circovirus is highly resistant to desiccation in the environment and to common disinfectants. Products that are often suitable for inactivating environmentally resistant viruses such as Virkon-S are recommended when attempting to disinfect contaminated surfaces. Where disinfection procedures cannot safely or readily be employed (ie. in a caring situation with carpet, dirt floors or surfaces that are not disinfectable), control is most often limited to preventing introduction of a shedding bird into a naive environment. Appropriate quarantine, biosecurity and routine screening of new birds should enable identification of infected birds (Raidal 2012). Birds identified as being infected should be immediately removed as they shed large numbers of virus particles (Phalen 2006). Any birds exposed to an infected bird should also be quarantined. An inactivated vaccine has been produced in Australia, although it is not commercially available (Raidal 2012).

Further Reading

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