SYSTEMIC VIRAL DISEASES OF PET BIRDS

A Continuing Education Program Prepared by

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INTRODUCTION

The purpose of this slide studyset is to review several of the important systemic viral diseases which are unique to pet birds. There will be no mention of the various viruses confined to the gastrointestinal tract such as rotaviruses, coronaviruses, astroviruses and reoviruses which have been occasionally isolated from pet birds and remain poorly described or are of uncertain pathogenicity. Nor will those viral diseases which occur in a wide variety of avian families be described, as most avian diagnosticians will already be familiar with the manifestations of paramyxovirus, avian influenza, and poxviruses in various avian species. Three important systemic viruses which are unique to pet birds will be described: herpesvirus (Pacheco's disease), circovirus (psittacine beak and feather disease), and polyomavirus (budgerigar fledgling disease).

Pet birds are among the most common pets in North America today, with an impressive diversity of species represented predominantly by psittacines (parrot-like birds) and passerines (perching birds). As expanding development by man diminishes the natural habitat of birds throughout the world, many species are becoming threatened, endangered, or even extinct. In response, concerned aviculturists are encouraging captive breeding of various pet bird species instead of continued wild-trapping and importation. Therefore, it has become more important than ever for avian diagnosticians to be familiar with the potentially fatal infectious diseases affecting these species, in order to maintain

healthy captive breeding populations and reduce the need for importing birds.

This script and accompanying slide studyset will provide an overview of the clinical features, gross and microscopic lesions associated with each disease, and provide additional information concerning disease transmission, pathogenesis, diagnosis and control measures when that information is known. Pet bird medicine is still a relatively new subspecialty of veterinary medicine, and much of the information about pet bird viral diseases has only recently been discovered, and much additional investigation is still needed. It is hoped that this material will assist avian diagnosticians with the recognition and differential diagnosis of pet bird systemic viral diseases.

SLIDE STUDYSET

Pacheco's Disease

Pacheco's disease was first described in 1930 and has also been called Pacheco's parrot disease and inclusion body hepatitis. The disease is limited to psittacines, however, a large number of psittacine species are susceptible. This is generally a peracute to acute disease with rapid clinical course and high mortality. Signs include lethargy, anorexia, ruffled plumage, polydypsia, polyuria and diarrhea. Conjunctivitis, sinusitis, hemorrhagic enteritis, and tremors are less frequently seen. This disease commonly occurs following periods of stress, particularly movement of birds to a new location. Asymptomatic carriers are associated with many outbreaks, with both Nanday and Patagonian conures being commonly implicated as carriers. Epidemics can be quite severe due to the highly infectious nature of the causative herpesvirus, which is horizontally transmitted through the feces.

Slide 1. Birds dying of Pacheco's disease are generally in good body condition due to the acute course of the disease.

Slides 2 and 3. Gross lesions of Pacheco's disease include enlarged, mottled and hemorrhagic livers (slide 2) and spleens (slide 3). Secondary bacterial infection of the lungs resulting in pneumonia may also occur. The intestinal mucosa may also appear congested or hemorrhagic in some cases.

Slide 4. Microscopically, multifocal to confluent areas of coagulative necrosis are commonly seen in the hepatic parenchyma, as well as multifocal congestion or hemorrhage. There may also be

mild to moderate mixed inflammatory infiltrates.

Slide 5. The microscopic hallmark of Pacheco's disease are large intranuclear eosinophilic inclusions within hepatocytes, associated with zones of hepatic necrosis. In some cases the inclusions may not be found, and there have been reports of basophilic intranuclear inclusions as well. The spleen frequently also develops necrotic foci and intranuclear inclusions within reticuloendothelial cells. Rarely, intranuclear inclusions may be found in the kidney within either glomerular tufts or tubular epithelial cells. Ultrastructurally, the virions are 190 to 210 nm in diameter and enveloped. Testing procedures to confirm the virus presence include inoculation of embryonated eggs, production of cytopathic effects on chicken embryo fibroblasts, virus neutralization, fluorescent antibody tests, ELISA tests, and the use of agar gel immunodiffusion assays.

Acyclovir has been successfully used as treatment in some outbreaks. It may be administered at 80 mg/kg TID by gavage, or added at higher dose rates (up to 240 mg/kg) to the food or water of birds which have not yet developed clinical signs. Preventative measures include quarantine of newly purchased birds for a minimum of 30 days, minimizing stresses which could cause virus shedding in asymptomatic carriers, and the use of killed virus vaccines. Killed Pacheco's disease vaccines are available from Maine Biological Laboratories, Inc., and BioMune, Inc. Vaccination appears to lower mortality due to virus challenge when administered before exposure; there does not appear to be any benefit from

vaccination on virus shedding from asymptomatic carriers.

Psittacine Beak and Feather Disease

Disease syndromes referred to as "psittacine beak and feather disease" (PBFD) or "French moult" have been recognized since the mid-1970's. Most commonly affecting Old World and South Pacific psittacine species, especially cockatoos, PBFD has now been recognized in over 40 psittacine species. Birds are most susceptible to the disease as juveniles, but adults can also develop clinical diseases. Only recently has the causative agent been recognized as a small (14-17 nm diameter), nonenveloped, single-stranded circular DNA containing virus, now classified as a circovirus. At present no suitable *in vitro* culture system for propagation of the circovirus has been identifed.

PBFD may manifest as either an acute or chronic disease. The acute form is generally seen in juveniles, often when the first contour feathers begin to replace the down. Affected nestlings become lethargic, anorexic, develop crop stasis, diarrhea, and may be pancytopenic. Feather lesions may not develop as affected birds die rapidly in many cases. In acute cases the bursa may appear small and the thymus necrotic.

Slides 6 and 7. The classic manifestation of PBFD is the chronic form associated with progressive feather dystrophy which continues for months or years before killing the affected individual. Affected feathers do not mature normally and retain their feather sheaths resulting in club-shaped and constricted feathers (slide 7), many may exhibit a fractured rachis. With each succeeding molt

a greater number of the feathers are affected, generally in a symmetrical pattern. Contour feathers are usually affected first, and as the condition worsens the primaries, secondaries, crest and tail feathers become involved.

Slide 8. Beaks may develop a dry crusted or powdery surface, become elonged or deformed, and are also easily fractured. Secondary bacterial infections are relatively common, and the lesions may progress to involve the oral cavity as well. Less commonly, the feet may become similarly involved.

Slide 9. Microscopic lesions include hyperkeratosis of the feather shaft, which inhibits the normal exsheathing process of the feather leading to the gross feather abnormalities.

Slides 10 and 11. Additional microscopic findings include necrosis and ballooning degeneration of epithelial cells within the epidermal collar and developing rachis; follicular epithelial cells may also be affected less frequently. Within the feather pulp (slide 11) a mixed infiltration of heterophils, plasma cells, and macrophages occurs.

Slides 12 and 13. The viral inclusion bodies of PBFD are basophilic, and are generally single large intranuclear inclusions within epithelial cells (slide 12), and multiple smaller cytoplasmic inclusions within macrophages (slide 13). The beak also develops hyperplastic and hyperkeratotic epithelium, areas of necrosis, and the outer layers of the epithelium may separate from the underlying layers of the oral cavity and tongue.

The inclusions bodies of PBFD are remarkably widespread beyond

the integument, illustrating the systemic nature of the disease. The inclusions have been found in the palate, tongue, crop, esophagus, intestinal epithelium, parathyroid, thyroid, adrenal gland, spleen, bursa, thymus, Kupffer cells of the liver, and the bone marrow. Identification of the inclusions is aided by immunohistochemical staining for the viral antigen. Another diagnostic aid is a commercially available DNA probe (Avian Research Associates Inc., Milford, Ohio) which can detect the viral nucleic acid in whole anticoagulated blood, epithelial cells from abnormal feathers, and fresh or fixed necropsy tissues. Sera from live birds can be tested using a hemagglutination inhibition test, and an agar gel immunodiffusion test is also available.

At present no effective treatment is available for PBFD, nor are any vaccines commercially available. Asymptomatic carriers are believed to exist, so birds which test positively for the PBFD virus but are non-clinical should be kept in isolation from other birds, and maintained in a stress-free environment. The virus may be transmitted either horizontally or vertically. The virus has been found in feather dust, crop secretions and feces, so transmission may occur through any of these routes.

Budgerigar Fledgling Disease

Budgerigar fledgling disease (BFD) was first reported in 1981 as a disease of hatchling budgerigars. Since that time the etiology has been determined to be a polyomavirus in the papovavirus family and many additional psittacine and even a few passerine species (canaries and finches) have been found to be

affected.

Slide 14. Most commonly affected and highest mortality rates (up to 100%) are seen in budgerigar hatchlings which are affected by the acute, generalized form of the disease. There are areas of the body where feathers do not emerge, while others emerge malformed similar to the feather abnormalities seen with PBFD. The abdomens may be distended and the skin discolored, chicks are often depressed with crop stasis, polyuria, head and neck tremors, and subcutaneous hemorrhages.

Slides 15 through 17. Internal lesions in affected budgerigars include ascites (slide 15) and hydropericardium (slide 16), enlarged hearts, and enlarged livers with multifocal necrosis and hemorrhages (slide 17).

Slides 18 and 19. Microscopic lesions are variable but may involve nearly every system. The spleen is the best site to look for the typical inclusions, as there are usually present within histiocytes and frequently in large numbers (slide 18). The typical intranuclear polyomaviral inclusions are very large - resulting in karomegaly - and palely basophilic to amphophilic. Recently developed immunohistochemical staining techniques may allow for detection of polyomaviral antigens in numerous tissues (slide 19 - immunohistochemistry of spleen), even in tissues where the inclusions are uncommon and difficult to find with routine hematoxylin and eosin (H&E) staining. Immunohistochemistry also allows for positive differentiation of polyomaviral inclusions from other intranuclear inclusions, such as those of PBFD.

Slide 20. Livers often have a centrilobular to submassive parenchymal necrosis. BFD inclusions are not always associated with the hepatic necrosis, but are easiest to find in the intact histiocytes in the non-necrotic periportal zones.

Slide 21 and 22. Zones of myocardial degeneration or necrosis may be found in the heart (slide 21 - H&E). While BFD inclusions may be difficult to identify in H&E stained sections, immunohistochemical staining often reveals large numbers of positive-staining nuclei within histiocytes and cardiomyocytes (slide 22 - immunohistochemistry).

Slides 23 through 28. Other tissues in which BFD inclusions may be identified include the skin and feather follicle - where they are associated with ballooning degeneration of the epidermis (slide 23 - H&E; slide 24 - immunohistochemistry), renal glomerular tufts (slide 25 - H&E; slide 26 - immunohistochemistry) or tubular epithelium, lung - where they are associated with an interstitial pneumonia (slide 27 - H&E), and the brain (slide 28 - H&E). Other tissues not illustrated in which BFD inclusions have been reported include the pancreas, adrenals, gonads, crop, proventriculus, intestinal lamina propria, skeletal muscle and bone marrow. BFD inclusions are frequently found in histiocytes, but are also seen in epithelial cells, endothelial cells, and various mesenchymal cells (neurons, cardiomyocytes).

Polyomaviral DNA probes can be used to detect small amounts of virus in cloacal swabs or feces of live birds, or fresh tissues from necropsied birds, and is commercially available (Avian

Research Associates Inc. Laboratory, Milford, Ohio). An alternative to immunohistochemistry for fixed tissues is a recently described *in situ* hybridization technique. Serologic test for BFD include agar gel immunodiffusion and virus neutralization assays.

The polyomavirus is believed to be transmitted both vertically and horizontally. Asymptomatic carriers are recognized, and adult infections have also been more frequently recognized in recent years. Routes of transmission likely include through the feces, the urates, and possible through air-borne feather dander similar to Marek's disease transmission in chickens.

Control measures presently involve testing of breeding birds, maintaining closed breeding facilities, and quarantine of new birds. In the face of a severe outbreak in an aviary, depopulation has been used. An alternate recommendation is to discontinue breeding birds for several months or even years, which seems to have helped in some aviaries. At present, no practical therapy has shown to be effective. Vaccines are not presently available, however, researchers are working on developing an efficacious killed vaccine against BFD.

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