Viral Diseases in Pet Birds

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Viral diseases of pet birds are poorly understood but are obviously important in avian disease etiology. Each year more viruses are identified and associated with pet bird disease states. In the future, vaccines will be available against pet avian viral diseases as they are against the viral diseases of poultry.

Viral diseases known in pet birds include the Avian Pox group (includes Canarypox, Lovebirdpox, Amazonapox, Budgerigar-pox, and many others), Herpes viruses (Pacheco's virus, Amazon tracheitis virus, Budgerigar herpes virus, cutaneous herpes of Cockatoos and Macaws, and others), Papovavirus (Papilloma-like virus in Finches, Papilloma-like virus in the skin of African Greys, probable viral etiology of cloacal and oral papillomas in Amazons, Macaws, and Budgerigars, Budgerigar Fledgling Disease (BFD), and Papovavirus disease seen in large psittacines (same as BFD?), Adenoviruses (Inclusion Body Pancreatitis, Budgerigar Encephalitis, and others), Reovirus infection (Asian Reovirus- Cockatoos, African Reovirus-African Greys), Parvovirus (Psittacine Beak and Feather Disease according to Lowenstine), etc. Probable diseases of viral etiology include Psittacine Wasting Disease and Conure Bleeding Syndrome. Many others will soon emerge in this infant field!

Infectious Diseases

Viral:
- VVND (Exotic Newcastle Disease)
- Pacheco's Parrot Disease
- Avian Pox Viruses
- Avian Influenza Viruses
- Papovavirus (Budgie Fledgling Disease)

VVND (VISCEROTROPIC VELOGENIC NEWCASTLE DISEASE)

Etiologic Agent: a paramyxovirus
General Information:

-A reportable disease (contact local USDA)
-Domestic strains of the virus exist which are significantly less pathogenic.
-A significant and continued threat to the domestic poultry industry.
-U.S. quarantine: all live birds are tested by cloacal swab and virus isolation. All birds which die are tested by virus isolation from tissues taken at necropsy.

Species Susceptibility:

Highly: Cockatiels, cockatoos (high morbidity, high mortality, acute death).

Moderately: Amazon parrots, conures (high morbidity, lower mortality). More likely to see neurologic signs because disease is usually more prolonged.

Relatively resistant: Macaws, Lorys, African Grey Parrot, Finches, Canaries

Transmission: Respiratory aerosols, fecal contamination of food/water, direct contact with infected bird(s), fomites.

Clinical Signs:

Acute death, asymptomatic birds. Depression, anorexia, weight loss, sneezing, nasal discharge, dyspnea, conjunctivitis, bright yellow-green diarrhoea, ataxia, head bobbing, opisthotonos. Neurological signs are seen in birds which survive the acute stage of the disease (uni or bilateral wing and leg paralysis, ataxia, chorea, head bobbing, torticollis, dilated pupils, opisthotonos).

Susan Clubb (Pet Farm, Inc., Miami, FL 33166) reported the following information in an outbreak she witnessed:.

> The incidence of respiratory signs in the total population was low. The incidence of neurological signs in the total population was low. "Respiratory signs (including conjunctivitis) were highest among: Cockatoos, Yellow Crowned Amazons, Cockatiels, Conures, Miniature Macaws. Neurological signs were highest among Coffin Cockatoos, Yellow Crowned Amazons, and some Conures. When these birds were excited, all neurological signs became more severe.

Diagnosis: Virus isolation by qualified USDA laboratory.

Treatment: Symptomatic treatment. Not advisable.

Necropsy:

Gross lesions: Hepatomegaly, splenomegaly, petechial or ecchymotic haemorrhages on serosal surfaces of all viscera and air sacs, air sacculitis, excess straw-colored peritoneal fluid.

Histopathology:

Liver: Erythrophagocytic histiocytes within sinusoids.
Spleen: Necrotising splenitis with erythrophagocytic histiocytes.
CNS: Multifocal non-suppurative meningoencephalitis, focal to diffuse areas of gliosis with mononuclear perivascular cuffing. Lesions most prominent within cerebellum.
Lung: Non-suppurative interstitial pneumonitis with erythrophagocytic histiocytes.
Vaccination: Prohibited in birds entering US. Vaccination does not eliminate the carrier state and hampers the detection of virus during quarantine.

PACHECO'S PARROT DISEASE

Etiologic Agent: a herpes virus

General Information:

* Disease first recognized in Brazilian psittacines in early 1930's by Pacheco and Bier.

* Disease first recognized in UB in 1975.

* Highly contagious, acute disease of psittacines from Central and South America (psittacines from around the world are susceptible).

* Disease is associated with stress (i.e. cold weather, quarantine, etc.) which can cause healthy carriers to shed virus and initiate infection in susceptible birds.

Species Susceptibility:

Highly: Macaws, Amazon parrots, Cockatoos, Cockatiels, Parakeets, some Conures (Half-Moon).

Relatively resistant: Nanday and Patagonian Conures.

Asymptomatic Carriers: Nanday, Patagonian, White-eyed Conures may be natural hosts for virus in wild, and certain individuals among them may be asymptomatic shedders of the virus when stressed. Many others are suspected.

The herpes virus of pigeons is serologically distinct from that of the owl, falcon, and parrot groups. It may be shed in feces of pigeons and is capable of causing death in parrots. The disease is indistinguishable from Pacheco's Parrot Disease.

Clinical Signs: Acute death, vomiting, bright yellow diarrhea, icterus (in Macaws), neurologic signs terminally.

Transmission: Fecal contamination of food/water, direct contact with infected birds.

Necropsy:

Gross lesions: Hepatomegaly, fatty liver with red streaks and petechiae, splenomegaly, friable spleen.

Histopathology:

Liver: Necrosis (multifocal or diffuse) with or without inflammation, eosinophilic intranuclear inclusion bodies within hepatocytes.

Spleen: Focal necrosis, eosinophilic intranuclear inclusion bodies (not as numerous as within liver).

CNS: No histopathologic evidence of inflammation or viral infection usually found.

Vaccination: Not commercially available.

AVIAN POX VIRUSES

Etiologic Agents: Large DNA viruses.

General Information:

* 5 strains: canary, parrot, pigeon, fowl, turkey. (All are usually considered variants of the same species, Poxvirus avium).

* Clinical signs in an individual depend on host susceptibility and virulence of the strain.

* 3 clinical forms:

a) **Cutaneous**: discrete papules, pustules, or crusty scabs (depending on stage of infection) develop on unfeathered parts of body (eyelids, beak, legs, feet).

Mortality is low. Usually self limiting.

b) **Diphtheritic**: extensive fibrinonecrotic respiratory tract, and esophagus (occasionally conjunctivae). Mortality is high.

c) **Combination of a) and b).**

Transmission: Direct contact with infected birds or fomites. Inspects may act as mechanical vectors.

CANARY POX

2 Forms: a) acute septicemia with respiratory signs resulting in death within 1 to 3 days.

b) chronic infection with proliferative dermal lesions around mouth, eyes, nostrils, and on feet. Oral lesions are common. 90-100% mortality is common.

Histopathology: Intracytoplasmic inclusion bodies (eosinophilic) = Bollinger's bodies.

PARROT POX

General Information: Common among some species of psittacines imported from Central and Bo. America as well as lovebirds.

Species Susceptibility:

Commonly Affected:	Amazon parrots (especially Blue-Fronted), Pionus spp., lovebirds, Australian parakeets, and Rosellas.
Relatively Resistant:	Cockatoos, Cockatiels, African Grey Parrots, Grand Eclectus Parrot, Lorys.

Can cause disease in chickens but is less pathogenic than in parrots.

Clinical Signs: Most involve peri-ocular tissues. Early: usually unilateral blepharitis and conjunctivitis -- palpebral edema and closing of affected eye -- ulcers and scabs at medial and/or lateral canthus. Ocular discharge begins as serious -- mucoid. Ocular lesions may develop (keratitis -- ulcerative keratitis, anterior uveitis, possible endophthalmitis). Opacification of cornea is common sequel to infection. Dermal lesions include: scaly papules at commissures of mouth, margins of cere, and around or within external nares. Oral lesions include: superficial raised plaques in the choanal area, base of tongue, posterior pharynx and within esophagus. Anorexia, sneezing, dyspnea, and occlusion of nostrils usually result. Death usually results from bacterial septicemia, pneumonia, asphyxiation (caseous plugs within airway), or starvation. Secondary fungal infections are not uncommon.

Necropsy:

Histopathology: Epidermal hyperplasia with ballooning degeneration, intraepithelial vesicles, and eosinophilic intracytoplasmic inclusion bodies.

Diagnosis: Virus isolation, E.M., histopathology.

Treatment: Parenteral Vitamin A, adenine arabinoside ointment (Vira-A, Parke-Davis), antibiotics (parenteral, topical), immune stimulant drugs, supportive care as needed.

Vaccination: Commercial pigeon and fowl pox vaccines are not effective in psittacines. Conventional fowl pox vaccines do not afford chickens protection against this strain.

AVIAN INFLUENZA VIRUSES

Etiologic Agent: Orthomyxoviruses

General Information: USDA testing procedures for imported birds are designed to isolate and identify influenza viruses. Hemagglutinating viruses often found in passerine birds (especially African Finches)

and relatively uncommon in psittacines.

BUDGIE FLEDGLING DISEASE

Etiologic Agent: A papovavirus.

General Information: A recently recognized disease of very young budgerigars, although other species of psittacines may become infected, producing high mortality in birds at 1-3 weeks of age. Less than 15-day-old birds are most susceptible. Asymptomatic breeding adults may be carriers. Infection may be egg-transmitted.

Species Susceptibility: The budgerigar, although other species of psittacines may become infected.

Clinical Signs: Diarrhea, dehydration, swollen abdomen, erythematosus skin, and death within 1 - 2 days. The following integumentary abnormalities have also been noted: lack of down feathers on back and abdomen, lack of filoplumes on head and neck. Growth of tail and contour feathers is delayed in birds that survive more than 15 days.

Comments: Clinical signs in young adults are similar to those attributed to birds with French Moult (etiology?). French Moult may be milder form because budgies that survive the infection manifest retarded growth of flight and tail feathers.

Necropsy:

Liver: Hepatomegaly, surface may appear pale, congested, and mottled with multiple pinpoint white spots or large yellow foci with petechial haemorrhages.

Kidneys: Swollen, congested; surface may appear mottled with multiple pinpoint white spots and petechial haemorrhages.

Heart: Cardiomegaly, hydropericardium, surface may possess multiple pinpoint white foci.

Histopathology: Intranuclear inclusion bodies within liver, kidneys, heart, spleen, bone marrow, uropygial gland, skin, feather follicles, and others.

Liver: As above, plus periportal mixed inflammatory infiltrates, multiple foci of coagulative necrosis.

Kidneys: As above, plus focal nephritis and nephrosis.

Heart: As above, plus myocardial degeneration.

Vaccination: An experimental vaccine is showing great promise in the control of the disease.

INFECTIOUS DISEASES

FUNGAL: * Candidiasis
* Aspergillosis
* Cryptococcosis

CANDIDIASIS-MONILIASIS

Etiologic Agent: Candida albicans

General Information: Yeast infection usually involving the crop mucosa. Young birds are most susceptible, although adults of some species (especially Lovebirds) are also frequently affected. Candidiasis with secondary bacterial complications is a common cause of crop impactions and death in baby psittacines. Prolonged antibiotic therapy, vitamin A deficiency, and feeding of spoiling or spoiled feed are predisposing factors. This yeast is considered by some to be part of the normal flora if isolated in small numbers. Disease results in overgrowth.

Clinical Signs/Symptoms: Unthriftiness, subnormal weight, increased appetite with concurrent weight loss in adults, thickened crop, regurgitation, slow passage of food in baby birds, raised white lesions on oral mucosae. Agent may invade intestinal mucosa -- malabsorption syndrome. The eyes and lungs may also be affected.

Diagnosis: Culture of lesion, feces; gram stain of wet mount from lesion.

Treatment: Nystatin, Ketoconazole, swabbing lesions with iodine, supplemental vitamin A.

ASPERGILLOSIS

Etiologic Agent: Aspergillus fumigatus

General Information:

* a systemic fungal disease.
* agent is ubiquitous in the environment
* very common disease of raptors and penguins. Not common among psittacines.
* in psittacines, disease is associated with poor husbandry (malnutrition, unsanitary conditions, etc.), excessive stress, concurrent or previous illness, and immunodeficiency.
* very dusty or damp environments or exposure to moldy food or nesting material may increase the incidence of disease.

Transmission: Inhalation of spores.

Clinical Signs: Among psittacines there appear to be two forms:

Acute: (Seen proportionately more often in African Grey Parrot) associated with acute onset of dyspnea in previously healthy birds (usually well husbanded).

Chronic: Chronic, debilitating disease complicated by one or more pathogenic bacteria.

Necropsy:

Gross: (**Acute form**): sometimes, only small caseous plugs within the distal trachea and/or small plaques on one or more of the air sacs that may also appear thickened.

Histopathology: (Acute form): usually limited to the respiratory tissue: (mycotic tracheitis, air sacculitis, and pneumonia).

Gross: (**Chronic form**): usually visible fungal growth on the air sacs and often overgrowing onto the serosal surfaces of the neighboring organs.

Histopathology: (Chronic form): besides mycotic pneumonia and air sacculitis, mycotic hepatitis, nephritis, and adrenalitis are common (mycotic septicemia).

Diagnosis:

* Serologic tests are available but are unreliable, and not recommended.
* Tracheal culture.
* Transtracheal wash with culture and cytology.
* Laparoscopy
* Radiography may aid in a presumptive diagnosis.

Comment: Consider Aspergillosis when a case of respiratory disease has been refractory to antibiotic therapy (usually numerous drugs have been tried in succession) based on culture and sensitivity.

Treatment: Treatment of acute cases may be rewarding. Treatment of chronic cases is very often unsuccessful.

* Amphotericin-B I.V. or I.P. in combination with flucytosine P.O.
* Nebulization or intratracheal injections may be employed.
* Levamisole has been used as an immunostimulant.

CRYPTOCOCCOSIS

Etiologic Agent: *Cryptococcus neoformans*

General Information:

* Organism probably ubiquitous within the environment.
* A rare disease of caged birds.
* We have diagnosed this disease in 3 psittacines in last 1-1/2 years (Green-Winged Macaw, Thick-Billed Parrot, African Grey Parrot). All diagnoses were made post-mortem.

Transmission: Presumably inhalation of the organism.

Clinical Signs:

* Presenting symptoms will depend upon organ(s) involved.
* Organism can cause encephalitis, respiratory disease, as well as other types of systemic disease.

Necropsy:

Gross: Depends upon organ(s) or organ system(s) involved. May note a tapioca puddling-like gelatinous material in area of lesions.

Histopathology: PAS stains may reveal organism within the tissues.

Diagnosis:

* Usually made post-mortem.
* India ink preparations of infected tissues or gelatinous material may yield encapsulated yeast forms.

Treatment: Same as Aspergillosis.