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Exotic Animal Practice

Vet Clin Exot Anim 8 (2005) 85–105

Viral diseases of companion birds

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The term "companion bird" in this article includes parrots, pigeons, canaries, and finches. Many viruses are known to cause disease in companion birds and have been well-studied. Other avian viruses have not been well-studied and some are not yet classified; probably others exist that have not been discovered because not all known viral families have been documented in birds. The following viral families are discussed alphabetically: Adenoviridae, Circoviridae, Coronaviridae, Flaviviridae, Herpesviridae, Orthomyxoviridae, Papovaviridae, Poxviridae, Reoviridae, Retroviridae, Rhabdoviridae, Togaviridae, and a virus from an unclassified virus family, proventricular dilatation disease. When appropriate, each virus family includes a description of the virus, the species affected, the transmission route and recommended type of disinfection, clinical signs, diagnosis, treatment, prognosis, and prevention modalities available.

Adenoviridae

Adenoviruses are nonenveloped viruses that are known to infect vertebrates. Avian viruses in the Adenoviridae family are in the genus Aviadenovirinae and include duck hepatitis, quail bronchitis, pigeon adenovirus, turkey viral hepatitis, goose adenovirus, marble spleen disease of pheasants, egg drop syndrome, turkey hemorrhagic enteritis, and the newly described hydropericardium syndrome in poultry [1]. Viruses in the Aviadenovirinae genus are separated into strains based on virus neutralization tests. The following description focuses on pigeon adenovirus, which is a tentative species within the genus Aviadenovirinae, and an adenovirus of psittacine birds that is not assigned to a genus according to the International Committee on Taxonomy of Viruses [2]. Pigeons are susceptible to some serotypes that infect chickens and some serotypes that are pigeon specific.

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doi:10.1016/j.cvex.2004.09.005 vetexotic.theclinics.com

Recently, fowl adenovirus serotype 4, strain KR5 was isolated from African gray parrots and cape parrots that had clinical disease [3].

Transmission of adenoviruses can occur horizontally, through the fecal oral route, and vertically. Vertical transmission has been demonstrated in chickens. Latent infections with periodic shedding are believed to occur.

Generally, adenoviruses cause the most severe lesions in birds that are less than 1 month of age. Most often, aviadenoviruses are considered to be opportunistic pathogens and usually are involved with concomitant infections or immunosuppression.

Pigeons

Pigeons from hatching to 5 years of age are susceptible to pigeon adenovirus; clinical signs are seen most commonly in birds that are 2 to 4 months of age. Clinical signs include depression, anorexia, dyspnea, a crouched stance, polydipsia, polyuria, and a slimy green diarrhea [4]. Young birds can die within 48 hours of displaying clinical signs. Mortality ranges from 0% to 60% and is greatest at 3 to 4 days postinfection [5,6].

Psittacine birds

Clinical signs in psittacine birds include depression, anorexia, and cloacal hemorrhage, followed by hepatitis, enteritis, pancreatitis, encephalitis, splenitis, conjunctivitis, and death [4]. Because birds that had no clinical signs exhibited intranuclear inclusion bodies that were consistent with adenovirus, it is suggested that asymptomatic infections can occur in psittacine birds. Usually budgerigars, lovebirds, cockatiels, Moluccan cockatoos, and red-rumped parakeets exhibit the typical clinical signs that were described above; however, other species can be found dead, including Amazon parrots, Patagonian cunures, Eastern rosellas, hyacinth macaws, lesser sulfur-crested cockatoos, and budgerigars. There is one report of an entire shipment of 59 eclectus that were shipped to a zoologic park dying of adenovirus [7]. This outbreak was associated with severe weight loss and liver disease that consisted of friable livers with whitish surface mottling and subcapsular hemorrhage.

Adenoviruses replicate in the nucleus and form basophilic intranuclear inclusion bodies that most often are recognized in hepatocytes and enterocytes [3]. Many different tests are available for poultry-associated aviadenoviruses, including ELISA, agar gel immunodiffusion (AGID), fluorescent antibody (FA), virus neutralization (VN), and hemagglutination inhibition (HI); however, the best test that is commercially available for psittacine birds is the DNA in situ hybridization (this is offered at the University of Georgia's Infectious Disease Laboratory, Athens, Georgia) [8,9]. A polymerase chain reaction (PCR) test has been developed to detect pigeon adenovirus [10]. Treatment consists of supportive care.

Because adenoviruses are nonenveloped viruses, they remain infectious for a long time in the environment and are difficult to destroy with disinfectants. Exposure to formalin, aldehydes, or iodophores for longer than 1 hour is recommended to destroy adenoviruses. Free-ranging pigeons and waterfowl may acts as reservoirs to birds that are housed outdoors. There is no vaccine available for psittacine birds.

Circoviridae

In 1989, this new family of pathogenic animal viruses was described, including circoviruses that were isolated from cockatoos—called psittacine beak and feather disease (PBFD)—chicken anemia virus, and porcine circovirus [11]. These nonenveloped viruses are one of the smallest yet described (14–16 nm). Later, pigeon circovirus and goose circovirus were described; both are antigenically different from PBFD [12].

Psittacine beak and feather disease

The PBFD virus most commonly causes clinical signs in captive and free-ranging Old World (Australian and African) psittacine birds, such as cockatoos, lovebirds, African gray parrots, and cockatiels. Less commonly, it causes clinical signs in New World (Americas) psittacine birds, such as Amazon species, macaws, and conures. A few cases of clinical PBFD have been documented in New World species, including scarlet macaws, red-lored and blue-fronted Amazon parrots, and Janday conures [4,13]. The PBFD virus is endemic in many free-ranging flocks of psittacine birds in Australia.

PBFD virus is shed in feces, feather dander, and various excretions and secretions. Asymptomatic birds can shed the virus for years before exhibiting any clinical signs. Vertical transmission probably occurs as evidenced by the development of PBFD in artificially-incubated chicks from infected hens. Chicken anemia virus (CAV) was proven to be transmitted vertically to the eggs from hens that were infected during the laying period [4]. Because the virus is nonenveloped, it is stable, can survive years in the environment, and is resistant to destruction by common disinfectants. A cell culture–derived CAV was killed by 1% iodine, sodium hypochlorite, 0.4% B-propriolactone, 1% gluteraldehyde, or 80°C for 1 hour.

Peracute, acute, and chronic forms of PBFD occur in parrots. Generally, the progression of the disease is dictated by the age of the bird when clinical signs first appear. Younger birds have a faster progression of the disease.

Peracute PBFD is common in cockatoo and African gray parrot neonates and is associated with sepsis, pneumonia, enteritis, weight loss, and death. Some present with severe leukopenia and liver necrosis [14]. Histologic

changes that are observed with peracute disease include feather follicle cell edema and bursal necrosis.

Acute PBFD affects chicks that are as young as 28 to 32 days of age. Clinical signs include depression and rapidly progressing feather dystrophy (necrosis, hemorrhage, and premature shedding of newly-developing feathers). In 1 week, 80% to 100% of feathers can be affected [4]. A nonregenerative anemia can be present, with a packed cell volume (PCV) of 14% to 25%.

Chronic PBFD is characterized by symmetric, slowly-progressive dystrophy of developing feathers that worsens with each successive molt. These are birds that survive the acute phase and then go on to have a prolonged incubation period that may involve years before clinical signs appear. The feather dystrophy includes retained feather sheaths, hemorrhage within the pulp, curled feathers, and circumferential constrictions of the feather shaft. Usually, the down and contour feathers are affected first, whereas the primary feathers are affected later. Birds can go on to develop complete alopecia, and sometimes, beak abnormalities that consist of progressive elongation of the beak and necrosis of the palate rostrally. These birds often are immunosuppressed and die of secondary bacterial or fungal infections.

Intracytoplasmic inclusion bodies are observed in the bone marrow, thymus and bursa. The DNA probe tests that are available at the University of Georgia Infectious Disease Laboratory can distinguish between PBFD and Psittacine Circovirus 2 (PsCV-2) of lories [15]. The PBFD DNA probe tests are performed on whole blood and detect viral DNA; a positive means that there was PBFD viral DNA in the blood. In a bird that has no clinical signs, it is recommended to retest the bird in 90 days to determine if the viral DNA is still present. If it is still present then the bird is infected, but if not, then the bird may have been infected transiently and overcame the infection. Any bird that displays feather abnormalities should have a feather follicle biopsy and DNA in situ hybridization performed in addition to the DNA probe blood test because some clinical birds are so viremic that they will have a negative blood test. This also can occur if a bird is extremely leukopenic [4]. A DNA probe test can be used to detect viral DNA on a swab of the environment to assist in determining the effectiveness of disinfection efforts. Treatment consists of supportive care and antimicrobials for secondary infections.

After clinical signs develop the disease is always fatal. Some exposed birds will mount an immune response and recover and never show clinical signs. These birds do not shed and are considered to be naturally vaccinated. In the United States, prevention through testing and isolation is the best way to control this disease. A vaccine is being developed for use in the United States. In Australia, an inactivated vaccine is available for use in birds, but it is not recommended for use in captive psittacine birds in the United States because of potential loss. In the United States it would be better to wait for a safe subunit or cell culture–derived vaccine and continue to test and isolate [4].

Psittacine beak and feather disease–2 and other strains

A PBFD variant (PsCV-2) has been described in lories; it is not as pathogenic as the originally-described PBFD [15]. Recently, a novel circovirus was described in mulard ducks that is associated with feather dystrophy [16]. Also, recent studies of PBFD viruses that were isolated from free-ranging birds in southern Africa demonstrated several unique genotypes that diverged substantially from PBFD viruses that were isolated from Australia and America [17].

Lories that had PsCV-2 had clinical feather lesions that were similar to PBFD but they had less severe clinical signs and, most importantly, they recovered. This is similar to what occurs with porcine circovirus 1 and 2 in pigs. One is highly pathogenic and the other is of little or no pathogenicity; therefore, it is imperative to distinguish between the two. Bird species other than lories may be infected with PsCV-2.

Pigeon circovirus

Pigeon circovirus is common in Europe. Clinically affected pigeons exhibit lethargy, anorexia, diarrhea, and poor weight gain. The highest mortality is seen in 7- to 8-week-old squabs. An outbreak occurred in pigeons that were 6 weeks to 12 months of age and many had concomitant bacterial, fungal, or parasitic infections. Intracytoplasmic inclusion bodies were observed in spleen, gut-associated lymphatic tissue, and cloacal bursa [12,18]. A DNA in situ hybridization and PCR tests have been developed to detect pigeon circovirus in tissue samples [19,20].

Coronaviridae

Parrots

In 1978, there was a report of a new coronavirus-like agent that was isolated from parrots. In 1982, these same investigators published a retraction of that claim because the samples were found later to be contaminated with chlamydial organisms [21,22]. Whenever the chlamydial organisms were filtered out of subsequent attempts to infect birds, the birds did not show any clinical signs of disease. Therefore, the investigators were unable to prove whether the clinical signs that were observed were due to the coronavirus-like agent that originally was observed on electron microscopy (EM) or to chlamydophila [22]. No further reports have been published.

Pigeons

There is one report of infectious bronchitis virus in a flock of racing pigeons in Australia that was exposed to chickens [23]. Of the 150 affected birds, 22 died from 1 day to 2 weeks after the arrival of clinical signs that

included ruffled feathers, dyspnea, and mucus accumulation at the ricti. These birds had concomitant trichomoniasis. Eight-week-old pigeons that were infected experimentally with the agent that was isolated from the chicken flock did not develop clinical signs, whereas 4-week-old experimentally infected specific pathogen free (SPF) chickens did [23]. No further reports have been published.

Passerine birds

Antibodies to infectious bronchitis virus were demonstrated in free-ranging passerine birds in Europe [4].

Severe acute respiratory syndrome

Human severe acute respiratory syndrome (SARS) is due to a coronavirus. The virus that was isolated from palm civets differs from the human SARS by an additional 29-nucleotide sequence [24]. Recent studies showed that domestic poultry (chickens, turkeys, geese, ducks, quail) are unlikely to be a reservoir for SARS and that chickens do not play a role as amplifying hosts for SARS coronavirus [25,26].

Flaviviridae

The most notable flavivirus is West Nile virus. West Nile virus is endemic along parts of the Nile River, but in the late 1990s it was found within the eastern United States and has since spread across the United States.

Crows, jays, raptors, and horses are susceptible species, whereas poultry are considered to be resistant. There are sporadic reports of otherwise healthy psittacine birds dying of West Nile virus [27]. If people or dogs are affected, they usually are older or immunosuppressed.

The West Nile virus is spread by mosquitoes; therefore, control of mosquitoes helps to control this disease greatly. There is speculation that the disease may be transmitted horizontally; this could be true as a result of the high viral load in vascular feather pulp and potential trauma and exposure to that pulp [28].

Clinical signs range from none in resistant species (eg, poultry) to neurologic signs (ataxia, circling, head tilt, seizures) and death in susceptible species. A complete blood cell count usually is normal or a lymphocytosis is present.

A serum antibody ELISA test is available for antemortem testing. Histopathologically, lesions are observed most often in the heart, liver, kidney, and brain. In owls, lesions in the heart were most common and most severe [29]. Recently, it was shown that a reverse transcriptase PCR of vascular feather pulp was significantly more successful at detecting West Nile virus than from liver, spleen, or cloacal samples [28]. Immunohistochemistry also is available for evaluating formalin-fixed tissue.

Treatment consists of supportive care. Recently, in humans, the use of α-interferon seemed to result in better success. This disease already has spread throughout the United States so it is too late to prevent the disease in this country. A conditionally-licensed vaccine is available for use in horses (Fort Dodge Animal Health, Fort Dodge, Iowa); it is being used intramuscularly in birds at the same or a reduced dose [30,31]. Studies are underway to evaluate the antibody response and protectiveness of this vaccine in some species of birds. Also, a DNA plasmid vaccine that is specifically for use in raptors is being developed [32].

Herpesviridae

Herpesviruses are enveloped DNA viruses that usually are host-specific. They cause mild disease in adapted hosts, severe disease in nonadapted hosts, and life-long latent infections with intermittent shedding. Many avian herpesviruses have been described, including Pacheco's disease virus, Amazon tracheitis virus, pigeon herpesvirus, and others.

Pacheco's disease virus

At least three distinct serotypes of Pacheco's disease virus (PDV) occur worldwide. Some psittacine strains can cause clinical signs in pigeons. All psittacine birds can show clinical signs of disease from PDV. Experimental transmission of PDV was successful through the oral, intramuscular, intranasal, and intraocular routes.

Most often, birds are found dead with no premonitory signs. Occasionally, birds may exhibit depression, anorexia, diarrhea, biliverdinuria, or neurologic signs [4]. Mortality can be 18% to 80%. Outbreaks can occur in aviaries with or without recent acquisition of birds. The virus occurs subclinically in many aviaries.

The visualization of hepatic intranuclear inclusion bodies (Cowdry type A bodies) is highly suggestive of PDV. An FA test is available to diagnose PDV from liver samples. Histologically, severe hepatic necrosis is observed. Other diagnostic modalities that are available include EM, cell culture, and viral-specific DNA in situ hybridization. Antibody tests are available, but are of little practical value because antibodies to PDV develop inconsistently; therefore, a bird with no titer could be latently infected and is shedding the virus [4]. Any bird that has an antibody titer to PDV is considered to be latently infected.

The use of acyclovir, 80 mg/kg, by mouth every 8 hours for 10 days, reduces mortality during an outbreak. Herpesvirus vaccines in general, including the PDV vaccine for use in psittacine birds (Psittimune PDV, Biomune, Lenexa, Kansas), prevent severe disease but do not protect the bird from latent infections or shedding [4]. The pigeon and domestic fowl herpesvirus vaccines do not work in psittacine birds because the viruses are serologically different.

Amazon tracheitis virus

Amazon tracheitis virus (ATV) is similar to infectious laryngotracheitis in gallinaceous birds and may be a variant. The ATV was isolated from the trachea of Amazon parrots in Europe. There is a peracute, an acute, and a chronic presentation. The peracute form is associated with severe diphtheritic lesions in the trachea and bronchi. The acute form is associated with tracheal necrosis. The chronic form is associated with conjunctivitis, sinusitis, coughing, rales, and secondary bacterial respiratory infections.

Histologically, hemorrhage and necrosis of the trachea can be observed, as well as a pseudomembranous tracheitis, pharyngitis, ingluvitis, and air sacculitis. Virus isolation and EM can be performed on tracheal exudates. Supportive care and antimicrobials for secondary infections are recommended. It is unknown if infectious laryngotrachitis (ILT) vaccines that were developed for poultry would prevent ATV; however, these modified live vaccines are not recommended in psittacine birds because of the possibility of inducing disease with vaccination [4].

Parakeet herpesvirus

Parakeet herpesvirus has been described in various species in the genus Neophema in the United States and Japan. It is believed to be distinct from ILT because intranuclear inclusion bodies are observed in lungs, rather than trachea. The virus has been associated with severe respiratory disease that involves the lungs and air sacs, central neurologic signs, and death [4]. A mortality rate of 21% (14/67) was reported in *Psitticula* species that were being held at a Japanese quarantine station within 2 weeks of being imported from India [33].

Herpesvirus associated with wartlike skin lesions

A herpesvirus has been demonstrated by EM in wartlike tissue that is observed most often on the toes of cockatoos. Macaws also are affected, but their toe lesions tend to be flat, plaquelike, depigmented areas. These lesions seem to persist for years with no clinical problems; often, long-time cagemates are unaffected which suggests a low infection rate [4].

Herpesvirus of European budgerigars

This herpesvirus has been described only in budgerigars in Europe. It is considered to be serologically different from other herpesviruses of psittacine birds, but serologically related to pigeon herpesvirus. This virus may cause feather abnormalities, but mainly is known for causing early embryonic death (ie, dead in shell). It is believed to be egg transmitted [4].

Herpesvirus associated with papillomatosis (wartlike gastrointestinal lesions)

Papillomatous lesions in the gastrointestinal (GI) tract should not be confused with warts that are caused by papillomavirus (see Papillomavirnae, under Papovaviride section). Grossly, the papillomatous cloacal masses look wartlike, but histologically, they lack the long rete pegs that are associated with true papilloma lesions [4]. Recently, internal and external papillomatosis has been associated with psittacid herpesvirus–1 (PHV-1) [34,35].

Species that are affected most commonly include the Amazon parrots and macaws. Amazon parrots are prone to developing concomitant bile duct or GI tract carcinoma that recently was associated with PHV-1, genotype 3 [35]. Also an African gray parrot, an eclectus, and a cockatiel were diagnosed with PHV-1–associated liver or GI carcinoma [35]. Papillomatosis has not been reported in free-ranging birds.

Clinical signs of papillomatosis include wartlike masses anywhere along the GI tract—most commonly in the cloaca and oropharynx. Birds may exhibit weight loss, signs of straining to defecate, soiled vent, or blood in stool. Some masses cause GI obstruction with associated clinical signs. Because the virus is latent, birds that have been treated previously may have recurrence of lesions and signs with stress. Amazon parrots that have bile duct carcinoma may exhibit biliverdinuria and lethargy and bile acid levels may be high [36].

Diagnosis is suggestive based on gross appearance, location, and the tissue turning white after application of (5% acetic acid) vinegar. Definitive diagnosis is based on histology. Treatment involves removing the wartlike growth. In the author's experience, it is best to apply silver nitrate to the lesion—or half of the lesion if it circumferentially involves the cloaca—every week under anesthesia until gone. Butorphanol also is administered at 2 mg/kg, intramuscularly, one time before the procedure. The lesions can recur with stress.

Orthomyxoviridae

Influenza virus is in the Orthomyxoviridae family and consists of 3 types—A, B, and C; only type A is found in birds. There are two influenza A subtypes based on surface proteins, "H" for hemagglutinin and "N" for neuraminidase. There are 15 known H subtypes and 9 known N subtypes. All subtypes are found in birds, but only H1, H2, H3, N1, and N2 are found commonly in humans. All avian virulent strains to date have been H5 or H7 subtype, but most H5 or H7 isolates have been of low virulence. For example, an H5 or H7 automatically is called a "highly pathogenic" strain, but live chicken pathogenicity testing can later find the virus to be nonpathogenic in chickens (eg, 2003 Texas H5N2 outbreak). Further information on avian influenza can be obtained at http://www.oie.org and http://www.cdc.gov.

The disease was described first in the 1890s and was isolated first in 1955. All species of birds are considered to be susceptible to influenza A viruses, but most infections result in no to mild clinical signs. In 1997, influenza A was shown to pass from chickens to humans in an outbreak in Hong Kong in which 18 people became sick and 6 died who had contact with infected birds; this outbreak was controlled by killing 1.5 million chickens. An H5N1 subtype that was isolated from humans in Thailand and Vietnam was sequenced genetically and all genes were of bird origin. In 2003, H5N1 subtype was confirmed in turkey poults in Cambodia, China (Hong Kong had a single positive peregrine falcon), Indonesia, Japan, Laos, South Korea, Thailand and Vietnam. Also in 2003, influenza A H7N7 was isolated from poultry workers in The Netherlands. More than 80 human cases were reported and one person, a veterinarian, died.

Wild birds are the natural hosts for influenza A, especially waterfowl, and generally do not exhibit illness from infection. Domestic birds, particularly chickens, are susceptible to high mortality with influenza A. Animal to human transmission has occurred, but is unusual. The chance that influenza A will develop human genes and transmit from human to human is real and may happen in the near future. Because the virus is enveloped it is susceptible to common disinfectants.

The many different strains of influenza A create a wide variety of clinical signs. Poultry tend to have respiratory disease and high mortality. Influenza A has been recovered from psittacine birds that were asymptomatic and those that showed signs of severe respiratory disease [4].

Many tests are available, including the AGID test, ELISA, hemagglutination inhibition, and reverse transcriptase (RT)-PCR. Not all birds develop demonstrable antibodies on the ELISA.

Supportive care is recommended for treatment in humans. Birds with H5 or H7 are required to be depopulated. The 2003 influenza A H5N1 that was isolated from people in Hong Kong was sequenced genetically and was found to be resistant to two antiviral drugs, amantidine and rimantidine; however, it was sensitive to oseltamavir and zanamavir. Currently, prevention consists of testing and culling after involving the federal government.

Papovaviridae

The Papovaviridae family of nonenveloped viruses consists of two subfamilies—Papillomavirinae and Polyomavirinae. Papillomavirinae causes true warts in birds, whereas Polyomavirinae causes polyomavirus in birds.

Papillomavirinae

Papillomaviruses are highly host specific and cause benign skin masses on the face and head of African gray parrots and the legs and feet of passerine

birds in the Fringillidae (ie, finches) family. Papillomatous masses that are found in the cloacal region or GI tract are not caused by a papillomavirus (see herpesvirus) [37]. Masses that are caused by papillomavirus are characterized histologically by long, thin folds of hyperkeratotic epidermis that is accompanied by acanthotic parakeratosis (rete pegs) [4]. The papillomavirus that was isolated from African gray parrots was related antigenically to bovine papillomavirus type 1 [38]. Differential diagnosis is based on demonstration of intranuclear inclusion bodies with 45-nm to 50-nm viral particles by way of EM.

Polyomavirinae

Polyomaviruses are host specific and cause subclinical disease in mammals; however, in psittacine and other birds they cause severe clinical disease. The first polyomavirus that was described in any animal was in budgerigars in 1981 [39,40].

The disease in budgerigars is different than what is described for other psittacine birds and is called budgerigar fledgling disease. Budgerigars exhibit poorly developing feathers, especially the contour feathers; abdominal distension; subcutaneous hemorrhage; and the young can die acutely with or without neurologic signs, such as ataxia and tremors.

Psittacine birds, other than budgerigars, usually develop subclinical disease because their mature, healthy immune system prevents the acute form of the disease. These birds shed polyomavirus intermittently for the remainder of the their life. Immature nonbudgerigar psittacine birds can develop peracute, acute, or chronic forms of the disease that usually depends on age at exposure. Peracute death occurs in young birds. Acute disease also occurs in young birds with an approximate mortality rate of 27% to 41% and includes 12 to 48 hours of depression, anorexia, delayed crop emptying, regurgitation, diarrhea, dehydration, subcutaneous hemorrhage, dyspnea, and polyuria. The subcutaneous hemorrhages are seen most easily over the crop, carpi, or cranium. One outbreak that was described is typical of an acute event; 31% of birds between 28 and 48 days of age died within a 6-week period.

Chronic disease is associated with weight loss, intermittent anorexia, polyuria, renal failure, poor feather growth and signs of immunosuppression, such as secondary bacterial or fungal infections. It seems that the chronic disease occurs in immunosuppressed adults that cannot overcome the clinical signs of infection.

Transmission of polyomavirus is through exposure to excretion and secretions, especially urine. Vertical transmission has been proven in budgerigars and is suspected in other species. The incubation period is believed to be between 2 and 14 days.

Gross necropsy findings include ascites, hepatosplenomegaly, renomegaly, pale cardiac or skeletal muscle tissue, feather dystrophy, and

subcutaneous hemorrhages. Histologic findings include hepatic necrosis, karyomegaly in hepatic and splenic tissue, bursal lymphoid depletion, and membranous glomerulopathy [4]. Viral intranuclear inclusion bodies can be detected in feather follicles and renal tissue. A DNA probe (PCR) test is available to detect viral DNA in tissue or feces. Antibody tests are available; a positive result denotes that exposure has occurred and that the bird probably sheds virus intermittently. Many birds in aviaries are affected subclinically and are a constant source of infection for other birds in the aviary and are a particular danger for young birds; therefore, all birds should be vaccinated.

Psittacine birds should be vaccinated with the commercially available, fully licensed vaccine Psittimune (Biomune). This inactivated vaccine is administered subcutaneously and has been proven to be safe and effective [41,42]. Generally, young birds are vaccinated starting at 5 weeks of age and are protected optimally at 2 weeks after the second vaccine. Birds that weigh 200 g or more receive 0.5 mL, whereas birds that weigh less than 200 g receive 0.25 mL of vaccine.

There is no treatment for the disease. The prognosis is grave if clinical signs are present. Because it is a nonenveloped virus it is stable in the environment and difficult to destroy. A synthetic phenol, sodium hypochlorite (bleach), stabilized chlorine dioxide, and 70% ethanol have shown some efficacy in destroying the virus. A swab of the environment can be tested with a DNA probe to evaluate the disinfection process.

Polyomavirus in passerine birds

This disease was described first in Canada in 1986; 51% of 2- to 3-day-old chicks died acutely after 0 to 48 hours of nonspecific illness. Acute mortality can be observed in passerine birds of any age. Those that survive can develop feather dystrophy and deformed beaks. Gross necropsy findings include hepatomegaly with paleness and mottling, splenomegaly with congestion, and subserosal intestinal hemorrhage [4]. Intranuclear inclusion bodies can be observed in the spleen, bone marrow, intestine, kidney, heart, and liver. DNA probe tests that detect polyomavirus in psittacine birds do not detect polyomavirus in passerine birds; therefore, one must request a specific passerine bird polyomavirus DNA probe test from the laboratory [43,44].

Paramyxoviridae

Newcastle's disease virus

Newcastle's disease is caused by a paramyxovirus of which there are four pathotypes—lentogenic, mesogenic, neurotropic velogenic, and viscerotropic velogenic. The last also is known as exotic Newcastle's disease, which is a foreign animal disease in the United States. Exotic Newcastle's disease is

highly pathogenic in poultry, but psittacine birds and pigeons are more resistant to infection. Free-ranging pigeons can serve as mechanical vectors. The nonexotic forms of Newcastle's disease used to be common before the use of vaccines.

The disease is spread by direct contact with viral particles from aerosolization of respiratory secretions or feces or from food, water, or litter that is contaminated with feces. Recovered birds are believed to shed the virus indefinitely. The incubation period ranges from 3 to 28 days, but the average is 5 days in chickens.

The clinical signs vary with the pathotype but include depression, diarrhea, anorexia, ruffled feathers, oculonasal discharge, conjunctivitis, dyspnea, ataxia, muscle tremors, paralysis, and death. In people, Newcastle's disease causes a mild, acute granular conjunctivitis, general malaise, and sinusitis that resolve within 7 to 20 days.

Avian serum can be sent to the San Bernardino County Laboratory in California for antibody testing for paramyxovirus (PMV)-1, -2, and -3. Many tests are available, including the HI test, ELISA, AGID, and virus isolation. Quarantine stations were developed to keep exotic Newcastle's disease out of the United States; therefore, any bird that enters U.S. quarantine is tested serologically.

Pet birds that have a nonexotic form of Newcastle's disease should be offered supportive care. Any bird, pet, that is positive for exotic Newcastle's disease in the United States is to be reported to federal authorities and destroyed.

Vaccines are available for control of nonexotic forms of Newcastle's disease in the United States. Exotic Newcastle's disease is controlled by the federal government by test and cull methods. A vaccine is available in Europe for exotic Newcastle's disease, but its use is not allowed in the United States because the tactic to control the disease in this country is to test and cull any positive birds.

Paramyxovirus (PMV)-2

Passerine birds that are infected with PMV-2 show mild, self-limiting disease, whereas psittacine birds, especially African gray parrots, that are infected with the same virus have severe difficulty breathing, diarrhea, and high mortality [4].

Paramyxovirus (PMV)-3

PMV-3 has been isolated from a large variety of psittacine and passerine birds and from clinically normal and dead birds. In *Neophema* spp the morbidity is high and the mortality is low. All ages are affected, but neonates have the most severe symptoms. Clinical signs include torticolis and ataxia. Gross necropsy findings include pulmonary edema and congestion, hepatomegaly, and pancreatic atrophy [4].

Nestling cockatiels had high mortality after clinical signs of opisthotonus, leg paralysis, and dyspnea. Gross necropsy findings included cardiomegaly and pericardial effusion. Virus was recovered form the brain and heart. Moluccan cockatoos that had PMV-3 presented with ataxia and death. Intracytoplasmic inclusion bodies were seen in the brain. Finches that had PMV-3 exhibited conjunctivitis that was followed by anorexia, diarrhea, and dyspnea. Only two of five finches that were infected experimentally died in one study [4].

Paramyxovirus (PMV)-5

PMV-5 has been isolated from budgerigars in Japan that exhibited acute clinical signs including depression, diarrhea, torticolis, dyspnea, and high (90%) mortality. Young birds seem to be more susceptible than adults [4].

Paramyxovirus (PMV) of columbiformes

This pigeon strain of PMV causes severe neurologic disease in pigeons, but only mild disease in chickens. Young pigeons suffer a high mortality rate of up to 90%. An inactivated subcutaneous vaccine against pigeon PMV-1 is used extensively in Europe. It is recommended to vaccinate pigeons at 3 to 4 weeks of age and again at 4 months before breeding; also, all new pigeons should be vaccinated [4].

Poxviridae

Poxviruses are the largest of viruses and the genus Avipoxviruses are found worldwide in greater than 20 families of birds. There are many species of Avipoxvirus, such as psittacine pox, canary pox, pigeon pox, falcon pox and fowl pox. Each species of pox has varied host specificity, but typically the most severe clinical signs are seen in its natural host.

The virus is transmitted by way of mosquito or by way of mechanical means through broken skin. Mites and other blood-feeding insects have been implicated in the spread of disease. The incidence of disease increases with rainfall and mosquito population. Mosquitoes were shown to remain infective for weeks to months. The virus is extremely durable in the environment; it is killed by 1% KOH, 2% NaOH, or 5% phenol, or heating to 50°C for 30 minutes or 60°C for 8 minutes [4].

Ten to 14 days postinfection, birds can show blepharitis, ocular discharge, rhinitis, and conjunctivitis that is associated with raised papules. Later, ulcerations of the lid margins occur that dry to crusty lesions, scabs, and eventually, scars. Persistent infections of 13 months or longer have been reported in chickens. Stress can activate a latent infection.

Clinical signs can be divided into ''dry'' pox that consists of cutaneous papular lesions, and ''wet'' pox that consists of mucosal papular lesions of the oropharynx. Occasionally, birds may display neurologic signs.

Diagnosis is based on typical clinical signs and histologic finding of Bollinger bodies (intracytoplasmic inclusion bodies) in skin or mucosal cells, and is considered to be pathognomonic.

Treatment consists of providing supportive care. Scabs should be left to heal naturally to lessen scarring. Vaccines have been created for chickens, pigeons, turkeys, canaries, quail, waterfowl, falcons, and Amazon parrots. The fowl pox vaccine may provide some protection in pigeons, waterfowl, and falcons. It is recommended to vaccinate before the breeding or mosquito season. Maximum protection occurs 3 to 4 weeks after vaccination.

Psittacine pox

This disease used to be common in recently imported Amazon parrots—especially blue-fronted Amazon parrots, macaws, and pionus—but is seen rarely today. Occasionally, an older imported bird will present with old pox scars on the eyelids, nostrils, and face. Corneal ulcers or crystallization may occur with or without a uveitis, that also can lead to later scarring [45]. In one outbreak of yellow-headed Amazon parrots, 28% died that developed the "wet" or mucosal form of pox that can cause pneumonia or air sacculitis [46].

Agapornis pox

Lovebirds have their own species of pox—Agapornis pox—which causes morbidity and mortality rates of approximately 75%. Lovebirds develop areas of dry, darkened skin, but not papules.

Canary pox

In canaries, aggression brings about wounds near the carpi; therefore, pox nodules often are seen there. Mortality rates can reach 70% or more as the result of a septicemic form that occurs commonly in canaries [4]. Also, lung tumors can form later in canaries. It is recommended to vaccinate with Poximune C (Biomune) at fledging stage (>4 weeks) and 1 month before breeding season. This is a wing web vaccine. Some canary breeders vaccinate every 6 months. Vaccinating healthy canaries during an outbreak is acceptable if care is taken not to become a mechanical vector while vaccinating.

Pigeon pox

Mortality in squab may reach 50%. Cutaneous tumors can form later in Columbiforme birds. The available vaccine is attenuated and is applied to racing pigeons by plucking feathers on the leg and brushing the vaccine on the open follicles. This should produce a swelling and a yellowish-brown discoloration in the area in 5 to 7 days [4].

Poxvirus of wild passerine birds

Many veterinarians who practice avian medicine take in wildlife. It is important to realize that passerine pox may enter your practice by way of the commonly infected and commonly presented wild house finch.

Reoviridae

The Reoviridae family consists of three genera—Orthoreovirus, Orbivirus, and Rotavirus—all of which have been described in birds. Orthoreoviruses are described more commonly in birds than the other genera of viruses; at least 11 antigenically distinct serotypes have been described in psittacine birds, pigeons, domestic fowl, geese, and raptors. Orthoreoviruses have been studied best in chickens and cause tenosynovitis. In other birds, the clinical signs are varied and can be vague and complicated by concomitant bacterial or fungal infections. Transmission mainly is by way of the fecal oral route, but also occurs from exposure to respiratory sections. The virus is stable in the environment and is difficult to destroy.

In psittacine birds, the liver is the primary organ that is targeted; clinical signs include anorexia, depression, weight loss, subcutaneous hemorrhage, diarrhea, ascites, dyspnea, nasal discharge, ataxia, paralysis, uveitis, hypopyon, and edema of the head and legs [4]. African gray parrots and cockatoos that were infected experimentally died or showed no clinical signs [47]. In another study, antibodies to reovirus were detected in 4.1% of psittacine birds in a U.S. quarantine facility over a 7-year period [4]. Recently, reovirus was described in African gray parrots and budgerigars; many had concomitant Pacheco's disease virus or fungal infections and lymphoid depletion [48,49]. Reovirus probably is more prevalent than currently is believed; it is likely that more information will be forthcoming as it is studied further.

Diagnosis is based on EM and is confirmed by virus isolation. Because the virus can be present with no clinical signs and concomitant disease is common, the presence that is indicated by EM is not enough to reach a definitive diagnosis. Intracytoplasmic inclusion bodies may be observed and many other tests have been used in poultry, including AGID, virus neutralization, ELISA, and immunofluorescent antibody (IFA). Virus has been detected in bursa, spleen, pancreas, and even in circulating cells [49]. The vaccine that is available for use in chickens is not recommended for use in psittacine birds because the chicken and psittacine reoviruses are not related antigenically [4].

Pigeon reovirus

Reportedly, 8% to 16% of homing pigeons in Europe had reovirus antibodies [4]. Clinical signs in pigeons include diarrhea and dyspnea and signs that are consistent with hepatitis.

Retroviridae

Retroviruses cause lymphoid tumors, including erythroid leukosis and myleoblastosis, in chickens. Clinical lesions that are similar to those described in chickens have been described in many other species of birds, including canaries, psittacine birds, and pigeons; however, a definitive causal relationship between retroviruses and lesions in these birds has not been proven [4]. Canaries that have lymphoid leukosis often have masses about the head and neck.

Retroviruses can be transmitted vertically and horizontally. Diagnostic modalities include complement fixation (CF) and ELISA to detect virus, and VN and ELISA to detect antibody. The virus is unstable outside of the host and is destroyed easily by common disinfectants. There is no vaccine available; therefore, hygiene is of the utmost importance.

Rhabdoviridae

All warm-blooded animals, including birds, are susceptible to rabies virus. Avian infections are rare, but have been reported in chickens, waterfowl, and raptors. A passive hemagglutination test in wild birds was seropositive in 23.1% of 65 predatory birds and 2.9% of 278 nonpredatory birds. Although no case of avian-to-human transmission of the rabies virus has been documented, pre-exposure rabies prophylaxis and preventing trauma while handling raptors is recommended [4].

Togaviridae

The Togaviridae family consists of three genera. Only the alphaviruses are described in birds, including Eastern equine encephalomyelitis (EEE) and Western equine encephalomyelitis (WEE).

Birds are a known reservoir for EEE and WEE; many species of birds are involved. The virus is transmitted through a mosquito bite. In pheasants, it was shown to be transmitted through the broken skin that is created by pecking.

The mosquitoes that are known to transmit EEE virus in the United States and Canada include *Culiseta melanura*, *Aedes* spp, and *Coquillettidia* spp. *Culiseta melanura* feeds mainly on birds and rarely on people, whereas *A sollicitans* and *A vexans* feed on birds and people, but are less likely to be infected with the virus. Bird mites are known to be potential vectors.

The mosquitoes that are known to transmit WEE virus include *Culex tarsalis* in the western United States and Canada and *Culiseta melanura* in the eastern United States. People are an accidental host to the virus. If infected with EEE, the mortality rate is up to 80%; if infected with WEE, the mortality rate ranges from 5% to 15%. In Canada, a direct connection was shown between the prevalence of the virus in wild birds and the number

of human cases. During the 1962 outbreak in Saskatchewan, Canada, the WEE virus was isolated from 22% of the wild bird population.

Birds may show no clinical signs or ataxia, trembling, weakness, paralysis, and death. People show varying degrees of neurologic signs and death. Antemortem diagnosis is difficult. Histopathologic lesions of lymphoplasmacytic encephalitis is suggestive. There is no specific treatment for EEE and WEE other than supportive care [4].

Proventricular dilatation disease

The causative organism of this disease has been identified as an 89-nm virus that is unclassified. The route of transmission is by way of fecal to oral and seems to affect birds of many orders, including psittacine birds.

Clinical signs include severe, chronic weight loss; regurgitation; delayed crop emptying; ravenous appetite; undigested food in stool; and neurologic signs (eg, falling off perch). The virus paralyzes the nerves in the proventriculus and the bird essentially starves to death—despite a good appetite—because of its inability to process food. Other nerves or organs can be affected alone or in conjunction with the proventriculus.

Suggestive diagnostic testing includes radiographs that demonstrate proventricular dilitation and whole, undigested food particles or seeds in the feces. Many diseases can cause proventricular dilation, including disease from parasites, yeast, megabacterium, mycobacterium, foreign body, neoplasia, and lead and zinc toxicosis.

Definitive diagnostic testing includes crop biopsy that demonstrates a ''lymphoplasmocytic ganglioneuritis.'' EM that demonstrates a 89-nm virus in the feces is highly suggestive, but the virus is labile and does not withstand overnight shipping.

Birds usually die within 2 years of developing clinical signs. Recently, treatment with cyclo-oxygenase (COX)-2 inhibitors significantly improved clinical signs; however, the mechanism against the virus is unknown [50]. Prevention consists of avoiding exposure to known infected birds.

Summary

Many viruses definitively cause disease in our companion birds, whereas other viruses have been implicated or associated with typical clinical signs. Some families of viruses that have been discovered in mammals have not been associated with disease in birds. It is imperative to perform a necropsy on any birds that die—whether a pet, aviary, or display bird, and despite the fact that other diseases may be present—because viruses can occur concurrently, especially when immunosuppression is present. Also, it is imperative to use available vaccines to decrease and control the incidence of these diseases, as has occurred in the canine and feline pet populations.

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