A retrospective study of circovirus infection in pigeons: nine cases (1986-1993)


Abstract. Circovirus infections were diagnosed in 12 pigeons from the United States, 4 pigeons from Australia, and 1 pigeon from Canada (1986-1993). Circovirus was identified by electron microscopic examination of basophilic botryoid cytoplasmic inclusions that had a histologic appearance similar to that of psittacine beak and feather disease virus inclusions. Inclusions were seen in splenic, bursal, gut-associated, and bronchus-associated lymphoid tissue macrophages and in bursal epithelial cells. Inclusions were composed of paracrystalline arrays of tightly packed, nonenveloped icosahedral virions 14-17 nm in diameter. Histologic changes in the spleens ranged from lymphofollicular hyperplasia with mild discrete lymphocellular necrosis to lymphoid depletion and diffuse histiocytosis. Lesions in the bursa of Fabricius ranged from mild lymphocellular necrosis to severe cystic bursal atrophy. Remaining histologic findings coincided with concurrent bacterial, viral, fungal, and parasitic infections. Immunoperoxidase staining and DNA in situ hybridization demonstrated that pigeon circovirus is distinct from psittacine beak and feather disease virus; however, both viruses apparently share some homologous DNA sequences. Clinical and diagnostic findings indicate that pigeon circovirus may be similar to psittacine beak and feather disease virus with respect to acquired immunodeficiency and subsequent multiple secondary infections.

Psittacine beak and feather disease (PBFD) virus and chicken anemia agent (CAA) are two antigenically distinct, but closely related circoviruses that cause disease in psittacine birds and chickens, respectively. PBFD is characterized by symmetrical feather dystrophy and loss, chronic debilitation, and death. The acute form of the disease occurs less frequently and is characterized by diarrhea and minimal feather changes; death may occur within 1-2 weeks after viral infection. One study reported 69% of all deaths in birds infected with PBFD virus were attributable to secondary infections; death from primary PBFD viral infection was less common. It is therefore speculated that immunosuppression may result from infection with PBFD virus. Additionally, PBFD virus may cause thymic and bursal necrosis or atrophy and has been associated with hypogammaglobulinemia.

CAA causes bone marrow hypoplasia with thymic and bursal atrophy in young chicks. It depresses protective vaccinal immunity and response of splenic lymphocytes to mitogen stimulation. It is thought to be responsible for secondary infections in chickens. Presumptive circovirus-like inclusions were recognized in pigeon bursas as early as 1986 in Canada and 1989 in Australia. Subsequently, circovirus-like infection of pigeons was first recognized and characterized in the United States in California during the fall of 1990. Since then, cases of pigeon circovirus infection have occurred sporadically throughout the state of California and in Australia.

The purpose of this comparative study is to present the clinical and pathologic features of circovirus infection in pigeons. Ultrastructural, antigenic, and DNA studies of this virus are also presented. These studies indicate that pigeon circovirus is antigenically distinct from PBFD virus but does share some homologous DNA sequences. Therefore, pigeon circovirus tentatively should be classified as the newest member of the proposed virus family Circoviridae.

Materials and methods

Criteria for selection of cases

Case records of the California Veterinary Diagnostic Laboratory System (CVDLS) (Davis and San Bernardino branches) were reviewed for disease in pigeons attributable to cir-
covirus infection. Twelve squabs with circovirus infection, representing 4 outbreaks of circovirus infection in pigeons in northern and southern California, were identified from 495 pigeons (2.4%) submitted for necropsy between June 1990 and January 1993. Also included in this study is 1 case of circovirus infection in a pigeon from Canada (1986) and 4 cases from Australia (1989-1993) some of which were previously submitted to 1 of the authors (KSL) to determine the association between splenic and bursal macrophage inclusions and PBFD virus. These 5 cases demonstrate the broad geographic range of the virus and suggest that pigeon circovirus is not a newly emerging pathogen.

Clinical histories

Case 1. A young female racing pigeon from a flock of 50 birds was submitted for necropsy to the CVDLS, Davis branch, in the fall of 1990. Squab mortality was 100%, with 3-5 birds dying per week. Initial clinical signs included anorexia and lethargy, with death occurring within 3-4 days.

Case 2. In May 1992, 2 5-mo-old squabs were submitted to CVDLS for necropsy. A group of 30 squabs were recently purchased and placed in a loft with 45 resident squabs. All birds were vaccinated for pigeon paramyxovirus. Clinical signs of disease initially occurred in birds in the purchased group and then, within 2 wk, sporadically in all birds. At the time of submission, 6 birds were exhibiting clinical signs and 5 squabs had died. Signs of disease included diarrhea, lethargy, rapid weight loss, inability to fly, and death. One week later, 4 more squabs were submitted with mild lethargy.

Case 3. Four 1-yr-old racing pigeons were submitted to CVDLS in August 1992. Clinical signs included anorexia, lethargy, sneezing, and diarrhea. These were the only birds in a group of 85 that exhibited clinical signs. All birds had been vaccinated for pigeon paramyxovirus at 60 days of age.

Case 4. An 8-mo-old racing pigeon was submitted to CVDLS for necropsy after returning 2 days late from a training flight. This bird and 1 other squab exhibited signs of respiratory distress and weight loss. Racing performance of 80 additional young pigeons was deemed poor by the owner. All birds were vaccinated for pigeon paramyxovirus 1 and pigeon pox virus.

Case 5. A Fantail pigeon was submitted for necropsy to the Department of Veterinary Pathology at the University of Saskatchewan in August 1986. The bird was thin and had urate and fecal pasting around the vent.

Case 6. A 2-mo-old Tippler pigeon with oculonasal mucopurulent discharge and proliferative lesions on the upper and lower beak was submitted for necropsy to Anapath in Australia in August 1989.

Cases 7-9. During 1993, a 4-mo-old racing pigeon (case 7) a 5-mo-old racing pigeon (case 8) and a 6-wk-old Jacobean pigeon (case 9) were submitted for histopathologic examination to Anapath in Australia. The 5-mo-old squab (case 8) came from a group in which 15-20% exhibited signs of respiratory disease.

Diagnostic workup

A full necropsy and diagnostic evaluation was performed on the pigeons in each case. Livers and organs with prominent gross lesions were cultured on blood agar. Intestinal contents were analyzed for pathogenic bacteria and parasites. Tissue impression smears were stained with Gimenez stain and examined for chlamydia organisms in most cases, and in case 1, an organ pool of liver, air sac, and spleen was cultured in a McCoy cell culture system. Inoculated McCoy cells were stained with a fluorescein-labeled monoclonal antibody specific for Chlamydia sp. and examined using ultraviolet light. Air sacs and trachea were cultured on selective media for mycoplasma in cases 1 and 4. Pooled tissues were cultured for viruses in embryonated chicken eggs, chicken embryo kidney cells, and chicken embryo fibroblasts in cases 1 and 4. Serum samples were tested for antibodies to avian paramyxovirus using the hemagglutination inhibition test in cases 2 and 3.

Light microscopy

Tissues from pigeons in each case were fixed in 10% neutral buffered formalin and processed routinely for histologic examination. Select tissues in some cases were also stained using Gomori’s methenamine silver, Brown and Brenn, Bodian, and periodic acid-Schiff techniques.

Electron microscopy

Pooled tissue homogenates of liver, spleen, lung, brain, and intestine from the squab in case 1 were negatively stained with 1% phosphotungstic acid and examined by transmission electron microscopy.

Formalin-fixed splenic or bursal tissues from pigeons of cases 1-4 and 8 were diced into small cubes, postfixed in osmium tetroxide, dehydrated in graded alcohols, and embedded in plastic resin. Ultrathin sections were mounted on copper grids, stained with uranyl acetate and lead citrate, and examined with a Zeiss 10C transmission electron microscope.

Feulgen staining

Feulgen staining was performed on replicate tissue sections from cases 1-3, which contained inclusion-laden macrophages.

Immunohistochemical staining

Immunohistochemical staining was performed on paraffin-embedded tissues from cases 1-3, 5, and 6 following published guidelines in an attempt to determine any antigenic relationship between the pigeon circovirus and PBFD virus. Replicate tissue sections containing viral inclusions, as observed by light microscopy, were subjected to immunohistochemical staining by the avidin-biotin (ABC) immunoperoxidase technique using a commercial kit and PBFD virus-specific polyclonal and monoclonal antibodies. Following immunohistochemical staining, the tissue sections were counterstained with hematoxylin, coverslipped, and examined microscopically. Known positive and negative PBFD control tissues were processed to validate the staining technique. Positive staining for PBFD viral antigen was indicated by the deposition of brown pigment.

DNA in situ hybridization

Replicate paraffin-embedded, inclusion-laden tissues from pigeons of cases 1-3 were subjected to DNA in situ hybrid-
ization following published guidelines to determine the presence of homologous nucleic acid sequences in pigeon circovirus and PBFD virus. Replicate tissue sections were dewaxed in limolene/xylene, rehydrated in graded alcohols to deionized water, treated in pepsin solution, and rinsed. Tissue sections were then hybridized to digoxigenin-labeled DNA probes. The first probe (FN8) was a 30-base, 3'-end-labeled oligonucleotide that is routinely used for detection of PBFD viral DNA in clinical specimens. Probe FN8 was designed specifically to recognize unique PBFD viral nucleic acid sequences. The second probe (probe 99) was a 2-kilobase pair double-stranded DNA probe made by polymerase chain reaction amplification of the PBFD viral genome with digoxigenin-11-dUTP included in the reaction mixture and was used to recognize homology between PBFD virus and pigeon circovirus nucleic acid sequences. Following stringency washes, antidigoxigenin antibody conjugated to alkaline phosphatase was placed on the tissue sections. Color development was achieved with nitroblue tetrazolium dye. Sections were counterstained with fast green, coverslipped, and examined microscopically. Appropriate positive and negative control tissues from psittacine birds with and without PBFD were used to validate the hybridization procedure.

Results

Individual cases

Case 1. The pigeon in case 1 had a caseogranulomatous bronchopneumonia from which a nonpathogenic Pasteurella sp. was isolated. Additionally, bronchial epithelial hyperplasia and syncytial cell formation were suggestive of herpesvirus infection. Chlamydia psittaci was isolated from an organ pool. Splenic lymphofollicular hyperplasia was pronounced and accompanied by discrete lymphocellular necrosis. Scattered macrophages at the periphery of splenic follicles contained large globular basophilic cytoplasmic inclusions. Additional histologic findings included prominent Kupffer cell phagocytosis; perivascular lymphocytic cuffs around arterioles, arteries, and large veins in the pancreas; and a mild enteritis with enterocyte necrosis associated with adherent coccobacilli. Parasitic infections included intestinal trichomoniasis and infection of proventricular glands by Tetramer sp.

Case 2. Microscopic tissue lesions in 1 of 2 birds initially submitted in case 2 included acute, multifocal, random hepatocellular necrosis with basophilic intranuclear inclusions (identified as adenovirus infections by electron microscopy) and periportal hepatitis. Similar inclusions were observed within enterocytes in association with enterocyte necrosis and crypt dilatation with detritus. Other crypts appeared hyperplastic. The spleen was characterized by diffuse histiocytosis. Discrete lymphocellular necrosis occurred in rare follicular structures that were mostly infiltrated with macrophages containing phagocytic debris. Rare macrophages in these follicles contained basophilic cytoplasmic “botryoid” inclusions. Similar inclusions were numerous in the bursa of Fabricius, which exhibited cystic bursal atrophy, discrete lymphocellular necrosis, and histiocytic follicular infiltrates.

Histopathologic findings in the second bird included nonsuppurative periportal hepatitis, nephritis, and enteritis. Enteritis was associated with trichomonad infection of the intestinal crypts. Marked lymphofollicular hyperplasia was observed in the gut-associated lymphoid tissue and spleen. Macrophages in splenic follicles and gut-associated lymphoid tissue contained amorphous basophilic inclusions, basophilic needle-like crystalline inclusions, or botryoid inclusions. Inclusion-laden macrophages were numerous in most follicles.

Histopathologic findings in the remaining 4 birds included marked splenic lymphofollicular hyperplasia with variable discrete lymphocellular necrosis (Fig. 1). Basophilic cytoplasmic inclusions were seen within splenic macrophages in 3 of 4 birds. There was variable lympholysis in bursal follicles and gut-associated lymphoid tissue in all birds; basophilic cytoplasmic inclusions were observed within macrophages and bursal epithelium in 3 of 4 birds. All birds had lymphocytic or lymphohistiocytic infiltrates in various organs, including pancreas, adrenal glands, thyroid glands, liver, kidneys, testes, myocardium, crop, or adipose tissue.

Analysis of intestinal contents revealed large numbers of trichomonads in some birds. Paramyxovirus type 1 titers were negative at 1:4 dilution for 5 of 5 birds tested (hemagglutination inhibition test).

Case 3. In 3 of 4 birds in case 3, serologic testing for avian paramyxovirus (by hemagglutination inhibition) was positive at 1:160 dilution for 1 bird and negative at a 1:4 dilution for the other 2 birds. Histologic findings included neuronal intracytoplasmic eosinophilic inclusions (paramyxovirus infection) in 2 birds, 1 of which also had trichomoniasis with severe necropurulent ingluvitis and esophagitis, moderate portal pleocellular hepatitis, and serofibrinous bronchitis and airsacculitis. One of the remaining 2 birds had a focal nonsuppurative encephalitis, prominent nodular lymphoid hyperplasia with discrete lymphocellular necrosis in the splenic white pulp, and numerous cells laden with dense basophilic cytoplasmic inclusions in the bursa of Fabricius. The last bird had a severe fibrinous airsacculitis, pericarditis, and peritonitis, with intralesional Gimenez-positive chlamydial organisms. Bursal tissue contained scattered cystic follicles containing mucus and detritus. Cells containing basophilic botryoid cytoplasmic inclusions were numerous in the bursal follicles and in gut-associated lymphoid tissue. Additionally, numerous Cowdry-type A intranuclear inclusions were present within hepatocytes and were accompanied by periportal and pa-
Figure 1. Photomicrograph of the spleen from a pigeon (case 2) with lymphofollicular hyperplasia infected with circovirus. There is mild discrete lymphocellular necrosis within splenic follicles. HE. Inset. Higher magnification of splenic follicle demonstrating discrete lymphocellular necrosis. HE.

renchymal infiltrates of lymphocytes, macrophages, and heterophils.

Case 4. Significant histologic findings in the pigeon in case 4 included proliferative dermatitis and bronchitis with intracytoplasmic Bollinger bodies, which are pathognomonic lesions of poxvirus infection. In addition, there was multifocal pyogranulomatous bronchitis, peribronchitis, pneumonia, and ulcerative tracheitis caused by secondary invasion by Aspergillus sp. The bursa was characterized by severe cystic atrophy with scattered cells in remnants of bursal follicles containing botryoid cytoplasmic inclusions (Fig. 2). Rare inclusions were also seen in macrophages in the spleen, along with diffuse histiocytosis and prominent sheathed arterioles. The liver had severe bile ductule proliferation in areas of extensive necrosis and accumulation of macrophages, which contained cell debris and hemosiderin. Kupffer cells in the remaining parenchyma were prominent and also contained hemosiderin. Nonsuppurative phlebitis of the portal and central veins was observed.

Case 5. Salient histologic findings in tissues from the pigeon in case 5 included airsacculitis, diffuse hepatocellular necrosis with eosinophilic intranuclear inclusions, and bursal lymphocellular necrosis and depletion. Many follicles contained cells with irregular basophilic cytoplasmic inclusions and eosinophilic intranuclear inclusions within the medullary epithelium. Splenic lymphoid depletion and diffuse histiocytosis was observed. Macrophages were distended with eosinophilic debris and gray-brown pigment. Escherichia coli was cultured from the air sacs and liver. Ova of Capillaria sp. were identified on fecal examination.

Case 6. Histologic lesions in the pigeon in case 6 included severe diffuse tracheitis, rhinitis, and sinusitis. Pseudomonas aeruginosa and E. coli were cultured from the nasal exudate. Poxvirus inclusions were associated with proliferative dermatitis. Additional histologic lesions included diffuse periportal hepatitis, multifocal purulolymphocytic interstitial nephritis, splenic lymphofollicular hyperplasia with mild histiocytosis, and lymphofollicular bronchitis. Splenic macrophages contained basophilic intracytoplasmic inclusions. Chlamydial organisms were identified in Gimenez-stained splenic impression smears. In the bursa of Fabricius, there was lymphoid hyperplasia and
occasional intracytoplasmic inclusion bodies in central bursal cells.

The owner of this pigeon subsequently had repeated problems with outbreaks of salmonellosis and enteritis associated with pure heavy growth of *E. coli*. Necropsy findings in a bird that subsequently died included lesions in the upper respiratory tract and oral cavity similar to those described above. Moderate lymphoid depletion was observed in the bursa and spleen, but no viral inclusions were seen.

**Case 7.** *Escherichia coli* was cultured from the liver and intestine of the bird in case 7. Histologic findings included cystic bursal atrophy with severe lymphoid depletion, splenic lymphoid depletion, and lympho-cellular necrosis in the splenic and gut-associated lymphoid tissue. Severe multifocal hepatic necrosis was associated with basophilic intranuclear inclusions. Additional lesions included multifocal lymphocytic interstitial nephritis and multifocal enteritis with crypt hyperplasia and villus blunting. Previously described cytoplasmic inclusions were observed in macrophages in the spleen and bronchus-associated lymphoid tissue and in cells in the bursa of Fabricius.

**Case 8.** Lymphofollicular hyperplasia in the spleen and bursa and lymphoid infiltrates in the liver, kidney, pancreas, and lung were the salient histologic features in tissues from the pigeon in case 8. Inclusion-laden cells were seen in the spleen, bursa of Fabricius, and gut-associated lymphoid tissue. Rhinitis and pharyngitis were associated with heavy bacterial growth. Chronic proliferative enteritis and nonsuppurative airsacculitis were also observed.

**Case 9.** In the pigeon of case 9, histologic lesions included lymphocellular necrosis and mild cystic change in the bursa, lymphofollicular hyperplasia in splenic and gut-associated lymphoid tissue, and lymphocellular necrosis in the gut-associated lymphoid tissue. Inclusions occurred in the bursa and in gut-associated lymphoid tissue. Additional lesions included nonsuppurative portal hepatitis with bile ductule proliferation, chronic hyperplastic enteritis, lymphocytic pancreatitis with acinar apoptosis, chronic airsacculitis, focal necrotizing esophagitis, and thyroid follicular hyperplasia.

**Circovirus**

Table 1 lists concurrent infections associated with circovirus infection. Table 2 summarizes the histologic changes associated with primary circovirus infection. Virus particles, 14-17 nm in diameter, were identified
when negatively stained pooled tissue homogenates from the pigeon in case 1 were examined by transmission electron microscopy. Cytoplasmic inclusions in macrophages and epithelial cells in ultrathin tissue sections of spleen and bursa, respectively, from cases 1-4 and 8 were dense, non-membrane-bound paracrystalline arrays or semicircles of tightly packed icosahedral, nonenveloped virus particles (Fig. 3). Particle size and morphology were consistent with previously described circoviruses. 

Cytoplasmic inclusions stained magenta with Feulgen staining, indicating that DNA was present. Immuno-histochemical staining with virus-specific antibodies for PBFD virus stained inclusion-laden macrophages brown in positive control tissue (bursa of Fabricius) from a psittacine bird with PBFD. In contrast, tissues from all pigeons and the negative control failed to stain.

Pigeon tissue sections exposed to the short DNA probe (FN8) did not hybridize and were devoid of staining. In contrast, control tissue from a bird with PBFD exhibited strong DNA hybridization with probe FN8 as indicated by blue-black staining of inclusions. Pigeon tissues exposed to the long DNA probe (probe 99) demonstrated weak to moderate hybridization,

Table 1. Concurrent infections in pigeons with circovirus infection.

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Concurrent infections</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td><em>Pasteurella</em> bronchopneumonia, herpesvirus bronchopneumonia (presumptive), chlamydiosis, attaching-effacing enteric cocccobacilli, intestinal trichomoniasis, <em>Tetramerces</em> sp. infection, proventriculus</td>
</tr>
<tr>
<td>2</td>
<td>adenoviral hepatitis and enteritis, intestinal trichomoniasis</td>
</tr>
<tr>
<td>3</td>
<td>respiratory aspergillosis, poxvirus dermatitis and bronchitis</td>
</tr>
<tr>
<td>4</td>
<td>paramyxovirus-1 encephalitis, trichomoniasis and ingluvitis/esophagitis, chlamydiosis, viral hepatitis (adenovirus or herpesvirus)</td>
</tr>
<tr>
<td>5</td>
<td><em>E. coli</em> airsacculitis, viral hepatitis (adenovirus or herpesvirus), intestinal capillaritis</td>
</tr>
<tr>
<td>6</td>
<td>bacterial rhinitis, sinusitis, and tracheitis (<em>E. coli, Pseudomonas aeruginosa</em>), poxviral dermatitis, chlamydiosis</td>
</tr>
<tr>
<td>7</td>
<td>viral hepatitis (adenovirus or herpesvirus)</td>
</tr>
<tr>
<td>8</td>
<td>bacterial rhinitis</td>
</tr>
<tr>
<td>9</td>
<td>etiologies undetermined</td>
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Table 2. Histologic changes in tissues from 17 pigeons infected with circovirus that are considered primary for circovirus infection.

<table>
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<th>Tissue</th>
<th>Histopathologic change</th>
<th>No. birds affected</th>
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<td>Spleen</td>
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<tr>
<td></td>
<td>cytoplasmic inclusions in macrophages</td>
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<td></td>
<td>diffuse histiocyctosis</td>
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<tr>
<td>Bursa</td>
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</tr>
<tr>
<td></td>
<td>and epithelial cells</td>
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<td></td>
<td>discrete lymphocellular necrosis</td>
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<tr>
<td></td>
<td>lymphoid depletion/atrophy/cystic change</td>
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<tr>
<td></td>
<td>lymphoid hyperplasia</td>
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<td>GALT*</td>
<td>discrete lymphocellular necrosis</td>
<td>6</td>
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<td></td>
<td>histiocytic cytoplasmic inclusions</td>
<td>4</td>
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<td></td>
<td>lymphofollicular hyperplasia</td>
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<td>BALT+</td>
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<td>discrete lymphocellular necrosis</td>
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<td>Myocardium</td>
<td>lymphocytic infiltrates</td>
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* Gut-associated lymphoid tissue.
+ Bronchus-associated lymphoid tissue.

Figure 4. Photomicrograph of DNA in situ hybridization of bursal tissue from a pigeon with circovirus infection (left) and an African gray parrot with PBFD (right). Dark staining indicates homologous DNA sequences in circovirus-infected pigeon tissue. Digoxigenin-antidigoxigenin alkaline phosphatase label with fast green counterstain.
suggesting that homologous nucleic acid sequences exist in PBFD virus and pigeon circovirus. (Fig. 4).

Discussion

In the pigeons of this report, primary and secondary lymphoid tissues exhibited a broad range of changes from lymphofoillcular hyperplasia to lymphoid depletion. The most common histologic change seen was lymphofoillcular hyperplasia, which was observed most frequently in the spleen and less frequently in bronchus- and gut-associated lymphoid tissue and rarely in the bursa of Fabricius. The lesion most likely reflects immune response to infection with numerous agents. Most squabs had several concurrent infections, which is suggestive of an ineffective immune response.

Various degrees of lymphocellular necrosis were observed in lymphoid tissue from most birds. Lymphoid depletion seen in some birds may have been a result of more extensive lymphocellular necrosis. Viral infection resulting in lymphocellular necrosis could directly impair humoral and cellular immunity. Severe bursal atrophy was apparent in 5 squabs, and the diffuse histiocytosis with lymphoid depletion in the spleen of these same birds indicate probable severe impairment of immune function.

Viral infection of the monocyte-macrophage system may interfere with functional aspects of this system, such as microbicidal activity or antigen processing, as is postulated in psittacine birds with PBFD. The age of the bird at the time of infection probably determines the effect of the virus on the immune system, as is observed with PBFD and CAA. Psittacine birds with PBFD may exhibit hypogammaglobulinemia and a similar incidence of secondary infections that are usually contained by humoral or cell-mediated immune functions. The experimental studies in chickens infected with CAA have demonstrated depressed herpesvirus vaccinal immunity.

Pigeons in cases 3 and 4, developed paramyxovirus and poxvirus infections, respectively, despite having been previously vaccinated. In addition, squabs in case 2 had been vaccinated against paramyxovirus-13 weeks prior to submission for necropsy; however, all 5 birds tested had no detectable antibodies to this virus. Vaccine failure or improper vaccination procedure cannot be discounted in these cases, but it is unlikely to have occurred in all 3 cases.

Light and electron microscopic characteristics of the pigeon circovirus were similar to those of PBFD virus. Ultrastructural studies demonstrated that the pigeon circovirus formed paracrystalline arrays and semicircles comprised of virions 14-17 nm in diameter, similar to the PBFD virus, and, as in PBFD virus, pigeon circovirus arrays of virions were readily recognized by light microscopy as basophilic botryoid cytoplasmic inclusions within macrophages. CAA virions are typically 18-22 nm in diameter. Inclusion-laden macrophages were observed frequently in the spleen and gut- and bronchus-associated lymphoid tissue. In addition, cytoplasmic inclusions were observed within bursal epithelial cells, which has not been reported previously in psittacine birds with PBFD.

Pigeon circovirus is a novel virus that is distinct from but closely related to PBFD. Immunohistochemical staining indicates that pigeon circovirus is antigenically distinct from PBFD virus. However, the 2 viruses are closely related based on homologous DNA sequences, as demonstrated by DNA in situ hybridization. These findings support the classification of this pigeon virus as the newest member of the proposed family Circoviridae.

Pigeon circovirus, like PBFD virus, may be associated with acquired immunodeficiency and a plethora of secondary infections. Definitive information on the pathogenicity of this novel circovirus in pigeons and its effects on the immune system can only be obtained by reproduction of the disease and by subsequent immune function studies.

References


