Post Mortem Findings in Pigeons From Australian Lofts Infected with Pigeon Circovirus

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Introduction

Pigeon circovirus (PiCV) is a single-stranded, nonenveloped virus that is in the same genus as the other circoviruses that infect birds, including the Beak and Feather Disease Virus of parrots (Todd et al. 2008). This virus has been found in Europe, Asia, North America, China, and Australia and most likely has a world-wide distribution (Raue et al. 2005, Scullion and Scullion 2007, Woods et al. 1994). There is moderate variation in the virus's genetic structure, but the pathological significance of this remains unknown (Todd et al. 2008).

The virus infects young pigeons and can be found in a number of tissues, including the liver, spleen, kidney, respiratory tissues, and cloaca. It is highly lymphotrophic and heavy concentrations are found in the Bursa of Fabricius, spleen, gut-associated lymphoid tissues (GALT), and bronchus-associated lymphoid tissues (BALT). It has been said that this virus has an affinity for both B and T lymphocytes, but lesions in the thymus have not been reported. There are no specific gross lesions indicative of virus infection, however, atrophy of the bursa and a small spleen are often found in infected pigeons (Duchatel et al. 2006, Smyth et al. 2001).

Microscopic diagnosis of infection requires the detection of cells containing characteristic large cytoplasmic basophilic inclusion bodies (Basophilic Globule Cells). These inclusions are most commonly found in the bursa and are less likely to be found in the spleen and other lymphoid tissues. Immunohistochemical and PCR-based studies have shown that the virus can be present in lymphoid tissue and many other tissues in the absence of inclusions. Nonspecific lesions also associated with the virus include lymphoid necrosis, and lymphoid depletion of the bursa and spleen. Lymphoid proliferation in the spleen, GALT and BALT has also been suggested to be the result of PiCV infection (Smyth et al. 2001).

PiCV can be detected in the blood, oral cavity, and cloaca of young birds. Blood and cloacal concentrations peak in the weeks after weaning. Cloacal virus can be detected after blood concentrations fall below detectable levels. Young birds that survive infection are believed to be infected for life. Virus can be found in many organs in adult birds, including the spleen and liver, but the majority of these birds will be negative for virus in the blood and cloaca, making screening for infection in adult birds impossible at this point (Duchatel et al. 2005, Duchatel et al. 2006, Todd et al. 2006).

Once the virus is introduced into a flock, it is believed to spread rapidly in the young birds and may infect up to 100% of them. Viral DNA has been found in embryos, but the importance of vertical transmission in maintaining this disease in a loft remains unknown. There is evidence there is a very high prevalence of PiCV in racing pigeon lofts in Ireland, Britain, and Europe (Todd et al. 2006).

Because this virus attacks lymphocytes and at least some macrophage-like cells, it is assumed to impact the function of a pigeon's immune system. PiCV is believed to play an important role in Young Bird Sickness

(YBS), which is associated with considerable morbidity and mortality in young racing pigeons. YBS has variable manifestations, but could be characterized as disease outbreaks by common infectious agents of pigeons that are more severe and involve more of the flock than would be expected where the pathogens acting alone. Disease causing agents associated with YBS include viruses, bacterial, fungi, and parasites (Table 1). The clinical manifestations of pigeons with YBS will largely depend on the secondary agent causing disease and can range from respiratory signs, signs of gastrointestinal disease, to nonspecific signs such as weight loss and poor racing performance (Raue et al. 2005, Scullion and Scullion 2007).

It is the purpose of this report to describe the post mortem findings of young pigeons infected with PiCV from 4 Australian lofts.

Materials and Methods

Tissues were obtained opportunistically from tissue samples submitted for histopathology or whole birds submitted for necropsy. In most cases, an extensive set of tissues including trachea, lung, air sac, multiple levels of the digestive tract, liver, kidney, gonad, brain, spleen and bursa were available for histological examination.

PCR confirmation of infection in live birds was done by submission of samples to Molecular Diagnostic Services (Googang, NSW). Samples were submitted when there was a suspicion of PiCV in pigeon lofts.

Results

***Flock 1*:** A single pigeon from this flock of racing pigeons was submitted in mid December. Exudative lesions of the oral mucosa had been reported grossly. PiCV infection was based on finding numerous basophilic globule cells (BGCs) in the bursa. Most were located at the junction of the cortex and medulla of each follicle. An increased rate of lymphoid necrosis was observed.

This bird also had significant lesions that were consistent with pigeon herpesvirus infection. There was a severe multifocal to focally extensive necrosis of the exocrine pancreas. Multifocal necrosis of the crop epithelium and ulceration of the oral mucosa was also observed. Pannuclear eosinophilic inclusion bodies were present in many cells in these lesions. A heavy superficial growth of bacteria and yeasts were seen on the surface of the oral lesions.

***Flock 2*:** The owner first requested help when 5% of his 200 young racing pigeons were found to be thin and pale. Birds in this flock where densely housed and there was heavy faecal contamination of the food and water. Two birds were submitted alive. Both were emaciated with marked pectoral muscle atrophy. Each had a leucocytosis, heterophilia, and monocytosis. Both were hypoprotenemic and one bird was severely anaemic. Both had *Capillaria* sp. infections of the intestines. The anaemic bird had an intussusception associated with a mass of worms and had bled into its intestinal tract. Both had a severe ulcerative ingluvitis. Bacteria, yeasts, and an abundance of trichomonads were found on impression smears of the crop. The birds were wormed and treated for the trichomoniasis by the owner. Subsequently, several birds were observed to develop central nervous system signs.

One bird with central nervous signs and two other thin birds were necropsied. Circovirus infection was confirmed in all three birds with the identification of BGCs in the bursa. Some degree of lymphoid depletion and increased lymphoid necrosis was also present in each bursa. Basophilic globule cells were also found in the submucosa of a tertiary bronchus and the submucosa at the junction of the proventriculus and ventriculus in one bird.

Concurrent diseases included a severe diffuse chronic air sacculitis, peritonitis and pericarditis with intralesional *Chlamydophila psittaci* in one bird. A second bird had a moderate chronic active

multifocal hepatic necrosis consistent with the liver being showered by haematogenous bacteria, a severe *Capillaria* infestation, and a moderate to severe lymphoplasmacytic meningoencephalitis. The cause of the brain lesions was not determined, but was suspected to be bacterial. The last bird had multifocal hepatic necrosis with intralesional *Chlamydophila psittaci*, lymphoid deletion of the spleen, and a mild diffuse acute enteritis associated with a heavy bacterial overgrowth of the small bowel. All three had a severe chronic ulcerative ingluvitis containing a heavy growth of bacteria and yeasts. Two birds had a marked lymphoid depletion of the spleen. Four of the 5 birds necropsied had serpentine deviations of the keel, suggesting that they had suffered from metabolic bone disease as squabs.

Instructions were given to improve hygiene, redesign the food and water containers to prevent faecal contamination and to decrease crowding. All birds were treated orally with fluconazole for two days and they were treated for 30 days with enrofloxacin in the drinking water. A "few" birds died in the following week and then mortality stopped.

***Flock 3*:** Flock 3 contained Australian Show Pen Homer Pigeons. Deaths were occurring in birds aged 4 to 10 weeks in mid February. Some birds were ataxic. Elevated trichomonad and coccidian levels were reported. The tissues submitted were from a bird that had a mucopurlent air sacculitis and a caseous nodule adjacent to the trachea.

Microscopically, BGCs were present in small numbers in the bursa. The bird had a moderate to severe diffuse heterophilic air saculitis, a multifocal to extensive suppurative meningitis and a mild diffuse lymphoplasmacytic tracheitis. There was a mild to moderate depletion of lymphocytes in the spleen. The cause of the air sacculitis and encephalitis was not determined, but based on the heterophilic response was believed to be bacterial. The nodule adjacent to the trachea was a partially encapsulated area of cellulitis containing a monomorphic population of round bacteria. Rare coccidia were seen in the intestines.

Flock 4: This flock of racing pigeons experienced weight loss, marked depression and an exudative ulcerative stomatitis in 6 week-old birds during the month of April. BGCs were found in the bursa where they predominated in the medullae of the follicles. There was an associated increase in necrosis of the lymphocytes and there was a moderate infiltration of heterophils. Lymphoid and histiocytic depletion was marked in the spleen, but periportal lymphoid nodules were present in the liver and perivascular lymphoid cuffs were common in the kidney. The oral mucosa was ulcerated and the surface of the ulcers contained a heavy growth of Gram positive cocci. Eosinophilic pannuclear inclusion bodies (Herpesvirus inclusions presumptive) were seen in the epithelial cells. There was multifocal pancreatic necrosis with some cells containing inclusions similar to those seen in the oral mucosa. There was a diffuse air sacculitis and serositis with intralesional hyphae which had morphology consistent with an *Aspergillus* sp.

A summary of the infectious diseases concurrently found in the birds from these 4 flocks experiencing PiCV infections can be found in Table 2.

PCR detection of PiCV

Twenty additional lofts were found to have birds infected with PiCV during the 2007-2008 breeding season based on the detection of PiCV DNA by PCR. The majority of the positive birds were less than 4 months old. Birds from lofts infected with PiCV were never positive when they were 8 months of age or older.

Discussion

PiCV has a worldwide distribution and lesions consistent with this virus infection have been recognized in pigeons in Australia since 1986 (Woods et al 1993). In other countries, this virus is believed to contribute to

YBS (Raue et al 2005), Scullion and Scullion (2007). YBS is a multifactorial illness, apparently caused by a number of pathogens occurring individually or concurrently with the common denominator being that all birds also have an infection with PiCV. The apparent immunosuppressive effect of PiCV infection is believed to result in an increase susceptibility to other pathogens and possibly an increased severity of disease caused by them. The purpose of this study was to approximate the extent of PiCV infection in a select group of Australian pigeon lofts, describe the concurrent diseases found in these pigeons and compare these findings to those described in birds with YBS in other parts of the world.

Although this cannot be considered a controlled study, it is clear that PiCV is present in a significant number of pigeon lofts in Australia. It is also clear that it can occur concurrently with other infectious agents and these agents are many of those that have been reported in descriptions of YBS.

It is easy to argue that the PiCV may have nothing to do with these other illnesses. If it is as widespread in Australia, as it is in parts of Ireland, Britain, and Europe where the majority of young birds will be infected with PiCV (Todd et al. 2006), then it will be almost inevitable that PiCV would be found in almost any young sick pigeon. On the other hand, the development and severity of diseases seen in these birds caused by opportunistic pathogens such as *Candida*, *Trichomonas* and *Aspergillus* coupled with the common finding of lymphoid depletion of the spleen and bursal necrosis is very consistent with the hypothesis that these diseases develop because of PiCV-induced immunosuppression. In the end, it seems to the authors that management issues such as crowding and hygiene, the ubiquitous nature of these pathogens in racing pigeons, the exposure to pathogens during the racing season and the impact of PiCV infection on the immune system will all play a role in the development of YBS and should be considered when attempting its control.

There have been no reports of what to expect in a loft with PiCV infection in subsequent breeding seasons. One of the author's (C.W.) impressions is that the consequences of infection will be less severe in the following breeding seasons.

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Table 1. Infectious Agents Found in PiCV-infected Pigeons as Reported in the Literature.

Viruses	**Fungi**
Adenovirus Columbid herpesvirus Poxvirus	*Candida* sp. – ingluvitis *Aspergillus* sp. – respiratory disease
Bacteria	**Parasites**
Escherichia coli – septicaemia, enteritis and air sacculitis *Klebsiella pneumoniae* – Other bacteria – ingluvitis *C psittaci* – respiratory and systemic disease *Salmonella* sp. – enteritis, septicaemia	*Trichomonas* – ingluvitis, enteritis *Spironucleus* (*Hexamita*) - enteritis Coccidia – enteritis Nematodes – enteritis, proventriculitis

Table 2. Infectious Agents Found in PiCV-infected Pigeons in this Study.

Virus	**Fungi**
Columbid herpesvirus	*Candida* sp. – ingluvitis *Aspergillus* sp. – respiratory disease
Bacteria	**Parasites**
Undefined bacteria – ingluvitis, hepatitis, enteritis, cellulitis, meningitis and encephalitis. *C psittaci* – respiratory and systemic disease	*Trichomonas* – ingluvitis Coccidia – enteritis *Capillaria* - enteritis