Update on Papovavirus Infection in Fledgling Psittaciformes

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PAPOVAVIRUS IN FLEDGLING BUDGERIGARS

Initial Outbreaks

In 1981 high mortality (death) rates of fledgling budgerigars *(Melopsittacus undulatus)* were reported both in Ontario (1) and in Texas and Georgia aviaries (5). Mortality occurred in birds approximately 1 day to 3 weeks old (1,5). Affected birds exhibited distended abdomens and reddening of the skin (5). Feather abnormalities were also observed by Bernier *et al.* (1). Changes included 1) lack of down feathers on the back and abdomen, 2) lack of filoplumes on the head and neck, and 3) retarded growth of the tail and contour feathers. Many fledgling birds were found with full crops and appeared to die acutely (5). Those birds that survived continued to suffer from feather growth problems (6).

Clinical Signs and *Histology*

Post mortem characteristics included: hydropericardium (fluid around the heart), enlarged heart and liver with areas of necrosis (dying cells), congested kidneys and lungs, and fluid in the abdomen. The virus produces basophilic (blue-staining) bodies within the nucleus of cells stained and examined with the microscope. These *"inclusion"* bodies are composed of virus particles from 50-55 nanometers (one-billionth of a meter) in diameter (1,5). These virus particles were later characterized as a papovavirus (2) (genus: polyomavirus) (14) and referred to as budgerigar fledgling disease virus (BFDV) (2).

Later Outbreaks

Another outbreak of BFD in Southern California occurred with an 80% loss of fledgling budgerigars. In addition to the classical acute death of fledgling birds exhibiting no clinical signs, 10% had nervous tremors of the head, neck, and limbs. Incoordination and irregular muscle movement (ataxia) were also observed 1-2 days prior to death. These birds were also dehydrated and emaciated, in contrast to those birds that died acutely. Again, microscopic examination of cells revealed intranuclear *inclusion bodies* in the *cerebellar* portion of the brain. These virus particles were identified as papovavirus (15).

A papovavirus was also isolated in Japan from budgerigars lacking flight and tail feathers at the age of 3 to 4 weeks during March to May 1981 (10). No death occurred in the budgerigars less than 2 weeks old, as found in typical BFD; and there were no signs of feather abnormality in the adult breeding birds. Virus was detected in the skin and kidneys of affected birds, and particularly in the outer cells of the feather follicle and kidney tubules. It had been previously suggested that "French molt" is a nonfatal form of BFDV (1,5). This work from Japan describing nonfatal feather abnormalities associated with a papovavirus in young budgerigars supported this. Whether mortality is a part of this disease may depend on the age of the bird at the time of infection (10). A papovavirus was also isolated from birds with "French molt" in Germany. The same virus induced mortality in fledgling budgerigars (13).

Transmission

Bozeman *et al.* (2) recovered virus from apparently normal young budgerigars kept in infected aviaries, suggesting the possibility of *carriers.* The carrier state would make it very difficult to break the transmission cycle unless some method was devised to recognize the carriers. These carriers would be capable of transferring the virus to healthy birds.

Since the virus is in the blood of the fledging bird, the mechanism of bird to bird transmission may occur by a number of methods: 1) regurgitation of infected crop tissue; 2) shedding of virus from feather follicle areas which then would be carried by air currents within an aviary; 3) cloacal excretion of virus from kidney tubules; and 4) respiratory exhalation of virus (7). *Horizontal transmission* has been demonstrated from adult carriers to young budgerigars (6). Transmission from an infected female breeder through the egg (vertical transmission) is also possible. On the other hand, maternal *antibody* transmitted to the egg yolk of an embryo may help protect the fledgling budgerigar in early life (unpub. data).

Preventive Management

Depopulation of infected birds (potential *carriers*), a thorough cleaning of facilities, and restocking with serologically negative breeders have been suggested for elimination of the disease from an aviary (6). A less drastic alternative in management is breaking the continuous breeding cycle by resting the breeders for at least 2 months. After removal and disposal of nest boxes, the breeders are first kept in the infected facility for 1 month and then moved to a clean facility for another month. During this month the old facility is completely disinfected and fumigated with formaldehyde gas. Breeders are moved back a minimum of three days after fumigation and allowed to breed in new disinfected nest boxes. This should decrease the amount of virus in feces and slow the spread of the virus to succeeding clutches (6).

PAPOVAVIRUS IN FLEDGLING NON-BUDGERIGAR PARROTS

Background

During the months of May and June of 1982 a papovavirus was involved in a disease outbreak affecting handfed fledgling parrots. It was referred to as a papovavirus-like-fledgling-disease (PVLFD). Two nurseries were involved within one aviary or breeding farm. Nursery 1 demonstrated a 41% mortality (15 of 36 birds) including a hyacinth macaw, a blue and gold macaw, a scarlet X green wing macaw hybrid, yellow naped amazons, a double yellowheaded amazon, and 8 sun conures. The second nursery had a 31% mortality (6 of 19) that included 3 sun conures and 3 white crowned pionus parrots (3).

Another aviary had an outbreak of this fledgling disease in both conures and macaws during the summer of 1983. It too was associated with a papova-like virus and caused the death of 14 out of their 45 birds, aged 4-6 weeks (12,13).

A papovavirus has been detected in

tissue by *fluorescent antibody techniques* during 1982-1984 in 17 cases of domestically bred nestlings of Austral, Asian, and Neotropical parrot species submitted to Cornell University. These included both hand-fed and parent-reared nestlings. The virus was referred to as generalized parrot papovavirus infection (GPPI). 8,9).

A papova-like virus infection has occurred in Western Australia in young fully-fledged adult lovebirds *(Agapornis spp.)* (16).

Histology

This fledgling disease of parrots has been associated with the BFD papovavirus by the detection of the characteristic *basophilic* intranuclear *inclusion bodies* (3,8,9,11,12,16) in the cells of affected birds. These inclusion bodies contain virus particles ranging in size from 42-49 *nanometers* (16). Studies in the serum of these birds show that the virus is related to BFDV (17). These previously described disease conditions in various species of birds have been referred to as: 1) a papova-like virus (12,13,16), 2) a papovavirus-like fledgling disease (PVLFD) (3), and 3) generalized parrot papovavirus infection (GPPI) (8,9). These conditions are probably caused by the same or closely related papovavirus. However, positive identification of the virus depends on future isolation and thorough characterization.

Clinical Symptoms

Clubb and Davis (3) listed a number of symptoms which were placed into two categories: peracute (rapid) death or chronic debilitation with renal failure.

First acute stage:

1. Reduction in daily weight gain.
2. Prolonged crop emptying time.
3. Vomiting and reverse peristaltic waves in crop.
4. Depression and glassy appearance of eyes.
5. Anorexia and dehydration.
6. Hemorrhage at site of feather removal.
7. Death within 24 hours.

Chronic state:

1. Subnormal weight.
2. Maldigestion and slow gut transit time.
3. Passage of large amounts of urine (polyuria).
4. Secondary yeast infection (candidiasis).
5. Abnormal feathering.
6. Depression.
7. Failure to self feed at a normal age.

Clinical recovery was predominately the case after reaching adult size.

Jacobson *et al.* (12) described the acute stage leading to death:

1. Weak with difficult breathing (dyspnea).
2. Distended crop.
3. Areas of hemorrhage under the skin.
4. Lethargy and loose droppings and *crop stasis.*
5. Death within 26 hours.

Pass (16), in Australia, also described an acute systemic illness leading to sudden death in young but fully fledged lovebirds. No feather abnormalities were observed.

***Post Mortem* Characteristics**

Abnormalities were observed in the spleen, kidneys, liver, intestine, myocardium, and skeletal muscles. Graham (8) found that skin and feather follicle involvement were the exception rather than the rule. There were typical BFDV basophilic intranuclear inclusion bodies. More detailed descriptions of the histopathological findings have been reported (3,8,9,12,16).

Serology

A serological test *(FAVN)* was developed by P.D. Lukert to detect anti-BFDV antibodies. This test would determine whether a bird had been exposed to the disease. The test was first used to detect these antibodies in flocks of budgerigars and individually infected birds (4,6) and was later used to detect anti-BFDV antibodies in surviving birds from the psittacine aviary which suffered fledgling mortality in May-June 1982. All fledgling birds that survived the acute stage of the disease contained anti-BFDV antibodies. Nest mortality in sun conures *(Aratinga solstitialis)* that had been imported from Guyana 7.5 months prior to the outbreak, suggested that serological tests should be performed on other birds from the same imported lot. Tested birds had no contact with the infected breeding farm prior to serum submissions, yet 80% (4 out of 5) were positive for antibody to BFDV. These birds were also associated with the second outbreak of a papovavirus in fledgling psittacines in the summer of 1983 (3).

Approximately a year and a half later, the incidence of *seropositive* birds in the breeding farm which had the first outbreak was determined again: 33% (35 out of 106) were found to be seropositive. Interestingly, many of these seropositive birds raised normal offspring in the following 2 years after the outbreak (3).

A second unrelated lot of imported sun conures was also tested for anti-BFDV. These birds had no other bird

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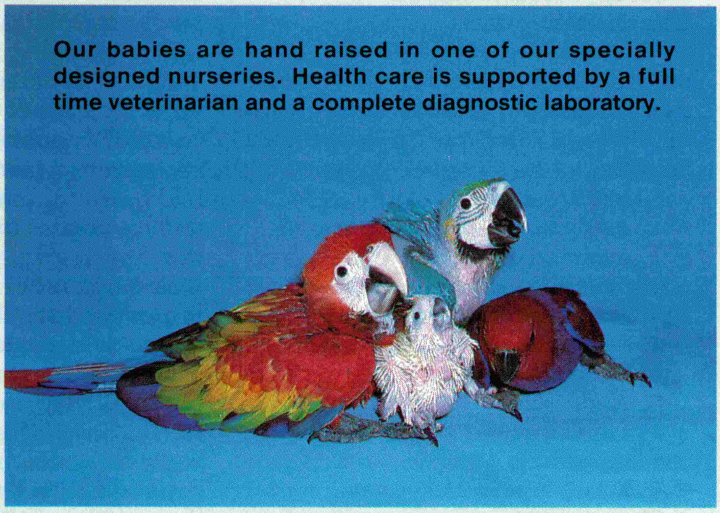

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contact within the U.S. and 23% (3 out of 17) were seropositive, suggesting that exposure to a papovavirus serologically similar to BFDV may occur in either quarantine or Guayana (3).

During 1983-1984, 3 geographically separate aviaries previously infected with a papovavirus serologically similar to BFDV were evaluated for the presence of antibody to the virus. The percent seropositive to BFDV for each aviary was 45% in Aviary 1; 25% in Aviary 2; and 10% in Aviary 3. A variety of species from these aviaries were seropositive, including macaws, conures, amazons, cockatoos, and "miscellaneous" (17). It therefore appears that a wide range of psittacine species are susceptible to a papovavirus similar to BFDV.

Three groups of seropositive birds, consisting of cockatoos and macaws, showed a decrease in serum antibody from one sampling period to the next. This suggests that antibody titers wane very rapidly after initial exposure to the virus. However, it also opens the question as to whether these birds maintain an infected state and become carriers, or eliminate the virus completely (17).

Perspectives in Preventive Management

It is important that psittacine birds which are known to have been in contact with this virus be tested for the presence of antibody over a period of at least a year. The virus is especially apt to become active in carriers during times of stress such as severe weather conditions and breeding. An increase in amount of antibody would suggest the recurrence of the virus from a *latent* (carrier) state. Viruses capable of *latency* may be reactivated during breeding and then spread to the fledgling birds.

On the other hand, these birds may completely eliminate the virus over a long enough time period after an initial exposure. This would remove the virus naturally from the environment (breeding facility) as the susceptible fledgling hosts are removed (3). Davis *et al.* (6) suggested that control of the disease in budgerigars may be accomplished by interrupting the year round breeding cycle in addition to complete cleaning and disinfection. Seasonal breeding is the normal occurrence in larger psittacine birds which may allow for a natural control (3). If the virus is completely eliminated from the adult bird and the breeding cycle is interrupted, then the breeders may become immune to future exposures (3). This remains speculative until further work is accomplished. It is very possible that some birds may become carriers while others completely rid themselves of the virus.

Until more is known concerning the nature of the disease, the suggested means of control in an exposed flock is the disruption of breeding cycles with complete disinfection of cages and nest boxes. In addition, new fledgling birds should not be introduced into a nursery infected with the disease. *Seronegative* parents should also not be introduced to infected nurseries or aviaries.

The best preventive medicine suggested by Davis *et al.* (1983) for budgerigar breeding facilities is maintaining a totally closed (isolated) flock. This avoids the introduction of infection by carriers of the virus. Where restocking is necessary, introduction of *seronegative* birds to BFDV should be practiced. Visitation by personnel between avaires should be avoided. Similar preventive care may also be applied to larger psittacine breeding facilities.

Serological Testing

The FAVN test is available through the Poultry Disease Research Center (PDRC). The serum must be taken and sent by your veterinarian to: Dr. Steve Thayer or Dr. R.B. Davis, Poultry Disease Research Center, 953 College Station Road, Athens, Georgia 30605.

A minimum of 200 microliters (0.2 cc) maintained cooled or frozen is required for the test. Prior contact with PDRC by your veterinarian before shipment is required.

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Glossary

antibody: a globulin produced in an animal against a foreign intruder.
basophilic: stains with basic dyes.
carriers: an individual that harbors the virus without clinical symptoms.
cerebellum: part of brain concerned with the coordination of muscles and maintenance of bodily equilibrium.
crop stasis: stoppage of food flow through the crop.
FAVN: fluorescent antibody neutralization test.
fluorescent antibody techniques: virus specific antibody labelled with a fluorescent dye; used to detect virus in tissue.
histology: microscopic study of the structure of animal (or plant) tissue.
horizontal transmission: bird to bird transmission.
inclusion bodies (intranuclear): virus particles enclosed within the nucleus of a cell.
latency: state of apparent inactivity.
nanometers: one billionth (10^{-9}) part of a meter.
post mortem: after death.
serology: study of serum components (i.e. antibodies).
seropositive: specific antibody to the virus present in bird.
seronegative: no specific antibody to the virus present in bird.

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