Psittacine Poxvirus Outbreak in New Zealand

Dr Brett Gartrell BVSc (Hons) MACVSc (Avian Health) PhD
IVABS, Massey University
Private Bag 11222, Palmerston North
Email: B.Gartrell@massey.ac.nz

In 2002, two eastern rosellas died and were presented to an Auckland avian veterinarian for post mortem and histopathology. Bollinger bodies were noted in the epithelium of the skin and poxvirus was diagnosed. The Ministry of Agriculture and Forestry (MAF) Biosecurity were notified. The birds belonged to an exporter of wild feral parrots from New Zealand to Europe. The property was placed in quarantine and MAF began an investigation. This investigation was hampered by the owner who changed his story several times in the course of the investigation regarding the provenance of the birds and their movements since purchased. Given the uncertainty in declaring an in-contact flock of budgerigars free of the disease, the birds were seized and after a court case in favour of MAF the birds were destroyed.

The poxvirus was only diagnosed by its histopathological features and its relationship to the psittacine poxvirus seen in continental America and Europe is unknown. No further cases in wild or captive birds in New Zealand have been reported. Considerable ill-feeling was generated in the Auckland avicultural community to MAF's actions. This case highlighted deficiencies in MAFs control of the export business and in wildlife disease surveillance in New Zealand.

Review of Psittacine Poxvirus

Introduction

Poxviruses (Family: Poxviridae) are one of the largest groups of animal viruses.[1,2] They are large DNA viruses which replicate in the cytoplasm of infected cells.[1,2] Avian poxviruses (Genus: *Avipoxvirus*) are not known to infect mammals; however all species of bird are considered susceptible to some strain of pox virus.[1,2] Much of the information on poxviruses is derived from studies on fowl pox.[1,2]

Psittacine pox has not previously been diagnosed in New Zealand or Australia[3,4,5] , but is endemic in Europe, North, South and Central America. [1,2,3,4,5]

Various strains of other poxviruses are already endemic within New Zealand including poxvirus strains that are able to infect shore plover, variable oystercatcher, weka, New Zealand pigeon, thrushes, silvereyes, black robin and North Island robin (Huia Wildlife Disease database, *unpublished*). None of these strains of endemic poxviruses have affected parrots in New Zealand (Huia Wildlife Disease database, *unpublished*).

Psittacine poxvirus is antigenically distinct from fowl, pigeon and quail poxvirus.[1,2,6,7] Psittacine poxvirus has been shown to affect lovebirds, [1,2,8] budgerigars[1,2,6], lories and lorikeets,[10] Amazon parrots,[1,2,5,9,11] macaws, [1,2,12] pionus parrots, [1,2,13] Quaker parakeets, [1,2] and cause mild disease in

chickens.[6,7,9] There is no data on the specific sensitivity of New Zealand parrots to this strain of poxvirus, but all members of the family Psittacidae (parrots) should be considered susceptible.[1,2,3,4]

Clinical syndromes of Avipox infection
Three clinicopathological forms of the disease are recognised.[1,2] Cutaneous, diphtheritic and systemic pox infections may occur and their expression is thought to be influenced by the strain of virus, the route of infection and the affected bird's species, age and condition.[1,2]

The cutaneous form of pox is characterised by discrete nodules that arise on unfeathered skin.[1,2] There is usually only low mortality from this form. The nodules form vesicles that erupt to form scabby erosions.[1,2,5,11,12] These lesions occur on the unfeathered skin around the eyes, beak margins, nares, cere and lower legs and feet.[1,2,5] Secondary infections occur commonly and may delay healing of the lesions.

Diphtheritic pox is characterised by fibrino-necrotic lesions on mucous membranes; commonly in the mouth, trachea and oesophagus.[1,2,5] These birds usually die. On examination the lesions appear as greyish-yellow or brown plaques filled with necrotic tissue. The lesions bleed profusely if damaged or removed.[1,2,5]

Septicaemic pox infections result in acute depression, anorexia and death and are more common in canaries and finches.[1,2]

Poxvirus in parrots
Psittacine poxvirus has been shown to produce both cutaneous and diphtheritic forms and can vary in virulence in different hosts.[1,2,5,11,12] It is often more severe than pox infections in other birds. The earliest clinical signs occur 10-14 days after infection and include serous ocular discharge, blepharitis, rhinitis and conjunctivitis, followed by ulcerations on the eyelid margins.[1,2,5,11,12] Dry crusty lesions appear 12 to 18 days after infection.[1,2,5] Severe upper respiratory tract disease may result from diphtheritic membranes in the oral, pharyngeal, oesophageal or crop mucosa.[1,2,5] Depression, anorexia, diarrhoea, and haematochezia have been seen with poxvirus lesions in the gastrointestinal tract.[1,2] Mortality was 81 % in an outbreak in Amazon parrots in the United States (n = 651/801).[1,11]

Incubation period
The recorded incubation period varies from 4 days to over a month.[1,2] Following exposure most birds develop lesions in 7 to 14 days.[1,2] Outbreaks in large flocks may continue for several months.[1,2]

Carriers
There has been suggestion that some birds (including lovebirds) may recover from infection but develop intermittent shedding of virus from feathers, skin or the gastrointestinal tract.[1,2] Persistent infections of up to thirteen months duration have been documented in chickens.[6,7]

Transmission of poxvirus
Poxviruses are extremely resilient in the external environment.[1,2] Transmission occurs by direct contact with an infected bird or through indirect contamination with infected objects or insect vectors.[1,2] Poxviruses cannot penetrate intact epithelium and must enter through an abrasion or puncture. All blood sucking insects can act as primary mechanical vectors.[1,2] In New Zealand, sandflies (*Culicoides spp.)* are the most likely insect vector, and are presumed to have played a role

in an outbreak of poxvirus in shore plovers at Mt Bruce (Alley M and Gartrell BD, *unpublished data*). Respiratory inhalation and egg transmission of poxvirus have been suggested but not proven.[1,2]

Pathological features
Demonstration of intracytoplasmic eosinophilic inclusion bodies (Bollinger bodies) is pathognomonic for the disease, and have been seen in association with lesions in the skin, mucosa of sinuses, trachea, crop, and oesophagus.[1,2,5,6,10] Necrosis and inflammation may obscure these inclusions.[2] Other histologic lesions include myocardial and hepatic necrosis, airsacculitis, pneumonia, peritonitis and accumulation of necrotic debris on the gastrointestinal tract.[1,2]

Development of immunity
Poxvirus infections stimulate the production of antibodies that provide immunity from that strain of virus for six to twelve months.[1,6]

Diagnosis
A presumptive diagnosis of poxvirus can be based on clinical signs; however differential diagnoses include trauma, *Trichophyton*, *Cnemidocoptes* mites, papillomavirus and bacterial infections.[1,2,12] Diphtheritic lesions can be confused with those produced by hypovitaminosis A, candidiasis, aspergillosis, or trichomoniasis.[1,2,12] The presence of poxvirus is confirmed by the demonstration of Bollinger bodies by histopathology or demonstration of the viral particles by electron microscopy.[1,2] Poxvirus is easily propagated in cell cultures.[12] DNA probes designed for chickens will not reliably detect psittacine poxvirus, as they are antigenically distinct.[9]

Risks to New Zealand parrots: wild birds, poultry and aviculture industries

Based on the information received, the strain of poxvirus seen in the rosellas was most likely a diphtheritic strain of psittacine poxvirus with high morbidity and mortality. DNA identification of the virus would have aided in determining its country of origin. The risk of this disease becoming endemic in New Zealand was considered to be high, based upon the resilient nature of the poxvirus in the environment, and its ability to spread via insects and other mechanical vectors.

The most severe threat to New Zealand posed by this disease was the potential effect on native parrots. All species of New Zealand parrots are potentially susceptible to this strain of virus. A combination of insect vectors and introduced wild parrots which could act as a reservoir for infection means the disease could quickly spread across both the North and South Islands even if the movement of captive parrots was stopped. The most likely scenario is that this disease would cause high mortality when it is introduced to a naive population. Some birds would survive the initial infection and develop immunity over time. However, the population of the critically endangered kakapo is approximately 80 birds and a viable breeding nucleus may not survive the initial disease effects. Other populations of endemic parrots such as the kaka, kea and kakariki are also likely to be adversely affected. Breeding stocks of the endangered Antipodean Island parakeet kept on the mainland of New Zealand would also be at risk.

The risk to the poultry industry if the poxvirus became endemic in New Zealand is unknown. Psittacine poxvirus strains have been shown to cause disease in chickens that was not prevented by vaccination with fowl pox or pigeon pox vaccination.[7]

The aviculture industry within New Zealand could also have been severely damaged by the introduction of this disease. Most aviculturalists in New Zealand maintain their birds in outside

aviaries and would therefore be at risk. Individual financial losses in lost trade for the domestic and export market could be high.

Control of Poxvirus

Control considerations

Poxviruses are stable in the environment and can persist for years in dried organic material.[1,2] The virus is resistant to many disinfectants but can be inactivated with:

- 1 % potassium hydroxide,
- heating to 50 degrees Celsius for 30 minutes,
- heating to 60 degrees Celsius for 8 minutes,
- steam application,
- 2 % sodium hydroxide, or
- 5 % phenol.[1,2]

Isolation of affected birds is recommended. [1,2,13] Birds are considered to be at their greatest infectivity when dried scabby lesions are present.[13] The spread of virus by mechanical spread via people and objects should be considered. Supportive care of affected birds and treatment of secondary infections is normally attempted. [1,2,12,13] However to eradicate this disease all infected birds and in contact birds should be euthanased and destroyed.

Vaccination can only be attempted using a homologous vaccine.[4,6,9,13,14] Fowl pox and pigeon pox vaccines will not produce protective immunity to the psittacine poxvirus, as it is antigenically distinct.[1,2,6,14] The use of an autologous vaccine may be considered, however the vaccination of at risk flocks has resulted in the nosocomial spread of poxvirus spread in outbreaks in the United States.[1,2,11,12] Immunity from attenuated live vaccines takes 10 to 14 days to develop.[1,6] Inactivated vaccines produce only partial immunity, which may protect a bird from severe disease but not prevent infection.[1,15] Further, strains of the attenuated poxvirus have the capacity to recombine with other poxviruses to create new strains.[1,2,12] For all these reasons, vaccination is not recommended as a control strategy.[1,2,4]

Vector control should consist of disinfection or destruction of fomites. The risk of spread by insect vectors may need to be addressed by judicious spraying with insecticides.[15] It was fortunate that this outbreak has occurred in winter when there is less insect activity.

For these reasons it was suggested that a restriction on the movements of captive parrots be put in place until after the outbreak was controlled. However, control was accomplished in this case by initially quarantining the property, with subsequent destruction of in contact birds and disinfection of the premises.

References

Ritchie BW. Poxviridae. In: Avian Viruses: Function and Control. Winger's Publishing, Inc., Lake Worth, FL. Pp 285-311, 1995

Gerlach H. Viruses. In: Ritchie BW, Harrison GJ, Harrison LR (eds). Avian Medicine: Principles and Applications. Wingers publishing, Lake Worth, Florida. Pp 862-948, 1994.

Macwhirter P, Ambrose S. Introduction of exotic avian diseases through legal importation. Eclectus 3 December 1997.
http://www.tasweb.com.au/bapa/eclectus.htm

Doyle K. Avian quarantine. Australian Veterinary Journal 75, 9 1997.

Jakoby J, Korbel R, Schneeganss D, Kosters J. Parrot pox in Amazon parrots in an import unit. (in German). Tierarztliche Praxis 18 (3) 255-258, 1990.

Winterfield RW, Reed W. Avian pox: infection and immunity with quail, psittacine, fowl and pigeon pox viruses. Poultry Science 64 (1), 65-70, 1985.

Boosinger TR, Winterfield RW, Feldman DS, Dhillon AS. Psittacine pox virus: virus isolation and identification, transmission and cross-challenge studies in parrots and chickens. Avian Diseases 26 (2) 437-444, 1982.

Tsai SS, Chang TC, Yang SF, Chi YC, Cher RS, Chien MS and Itakura C. Unusual lesions associated with avian poxvirus infection in rosy-faced lovebirds (Agapornis roseicollis). Avian Pathology 26 (1) March 1997

Olmos PR, Martinez BL, Lopez JE, Martinez LP. Pathogenicity and antigenicity in fowls of a suspected isolate of psittacine pox virus (from Amazona stivae). (in Spanish). Veterinaria – Mexico 17 (2) 104-109, 1986.

Gerlach H, Ramis AJ, Enders F, Casares M, Truyen U: Avian pox in lories (Neopsittacus sp.): a case report. International Virtual Conferences in Veterinary Medicine: Diseases of Psittacine Birds, 1998.
http://www.vet.uga.edu/ivcvm/1998/gerlach2/gerlach2.htm

McDonald SE, Lowenstine LJ, Ardans AA. Avian pox in blue-fronted amazon parrots. Journal of the American Veterinary Medical Association 179 (11), 1218-1222, 1981.

Fudge AM. Psittacine vaccines. Veterinarian Clinics of North America – Small Animal Practice 21 (6) 1273-1279, 1991.

Winterfield RW, Clubb SL, Schrader D. Immunisation against psittacine pox. Avian Diseases 29 (3) 886-890, 1985.

Wernery U. Viral diseases. In: Samour J (ed). Avian Medicine. Mosby, London. Pp 264-274, 2000.