# Treatment and Prevention of Avipoxvirus Bill Lesions in North Island Brown Kiwi (*Apteryx mantelli*)

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## Abstract

A juvenile, captive-reared North Island brown kiwi (*Apteryx mantelli*) was presented to Massey University's Wildbase Hospital for a 1.5 x 1 cm raised, dry lesion entrapped under the rhinotheca. The rapid expansion and eruption of the lesion, radiographic evidence of bone lysis of the premaxilla, and positive response to treatment, confirmed a diagnosis of cutaneous avipoxvirus. The bone lysis was found to be a result of pressure necrosis to the premaxilla due to the entrapped nature of the lesion and thus was debrided to allow the active lesion to erupt outwardly. Treatment objectives were to allow the avipoxvirus to run its course whilst preventing secondary infection, providing pain relief and encouraging appropriate keratin growth with intermittent bill debridement and reshaping using a dremel. After five weeks of treatment, the lesion had completely resolved, and the bird was released into the wild a few months later. This report describes the approach to treatment and prevention of avipoxvirus bill lesions in North Island brown kiwi as well as the risk factors that predispose these birds, particularly captive-reared juveniles, to infection.

**Key words:** *Apteryx mantelli*, avipoxvirus, avian pox, brown kiwi, bill lesions, bill debridement, captive rearing.

## Introduction

Avipoxvirus (APV) is a viral disease that belongs to the *Poxviridae* family and is a double stranded DNA virus known to replicate and mature in the cytoplasm of infected cells (Ha *et al*. 2013b). At least three different strains of the virus including A1, A3 and B1 are found in New Zealand with A1 and B1 being the most common (Ha *et al.* 2011). Transmission occurs via direct contact with infected individuals as well as food and water, contact with vectors such as mosquitos, sandflies, ticks and mites, or by exposure to environments that harbour the pathogen (Tripathy and Reed 2008). The virulence of APV amongst wild populations is determined by physical or environmental stressors, the type of viral strain, presence of concurrent infections, route of infection and the age/species of the infected bird (Ha *et al.* 2011). Virus particles survive well outside of the host and infection is host-specific (Ha *et al.* 2013a).

APV has three known clinical presentations in birds. The most common is the cutaneous form which manifests as proliferative dermal nodules or tumours on parts of the bird where there are no feathers such as the bill, face, legs and eyelids (Adams *et al.* 2005). It is thought lesions localise here because of exposure to vectors such as mosquitos (Bean 2017). This form has a low mortality rate with most birds able to recover and develop long-lasting immunity and thus this is a disease that predominantly infects young animals (Bean 2017). Another clinical presentation of APV is the diphtheritic form which impacts the upper respiratory and gastrointestinal tracts and has higher mortality rates (Bolte *et al.* 1999). The third known clinical presentation of APV is the systemic form and is typically only seen in canaries (Van Riper and Forrester 2007).

Avipoxvirus was first reported in kiwi in 2011 (Ha *et al.*2011). Documented cases of APV in kiwi have displayed low mortality rates and even though the kiwi recovered well in these cases, the true virulence of APV and consequent contribution to species decline of kiwi is still yet to be determined (Bean 2017). The main cause of concern is immunosuppression resulting from APV infection and the risk it poses for secondary bacterial and fungal infections (Tripathy and Reed 2008).

## History, Clinical Findings and Treatment

An eight-month-old, captive-reared, male North Island brown kiwi (*Apteryx mantelli*) was referred to Massey University's Wildbase Hospital from a captive-rearing institution for a lesion consistent with cutaneous AVP under the rhinotheca. The kiwi had a known one-month history of this lesion that was discovered during the bird's pre-release clinical examination. It was thought the lesion was a result of trauma and after debridement, the lesion was sealed with a layer of nail varnish to prevent contamination. The staff at the kiwi hatchery were managing the lesion with twice daily cleaning using chlorhexidine (Chlorhex-*C,* 0.5%, Jurox Animal Health, Rutherfort, NSW, Australia) and application of a medical grade manuka honey dressing (PAW Manuka Wound Gel*,* 25 g, Blackmores, Warriewood, NSW, Australia). However, the lesion continued to progressively grow and erupt, at which stage AVP became the top differential diagnosis. The lack of improvement with a prolonged phase of eruption over a few weeks despite symptomatic treatment was a cause of concern amongst staff and the kiwi was referred to Wildbase Hospital.

On presentation at Wildbase, the kiwi was bright, alert and responsive. Initial physical examination revealed the bird was in a good body condition weighing 786g. The kiwi's hydration status was adequate. A 1.5 x 1 cm raised, dry lesion was identified on the distal dorsolateral rhinotheca that appeared to be lifting medially and missing keratin over the wound (Figure 1). No further evidence of proliferative dermal nodules or tumours in regions without feathers was noted. The kiwi was deemed stable and was given 20 ml of oral fluids, a 4 mg/kg dose of butorphanol (*Butorgesic*, 10 mg/ml, Troy Ilium, Glendenning, NSW, Australia) intramuscularly for analgesia and a 24-hour acclimatisation period prior to undergoing further manipulation. Due to the infectious nature of APV, a strict isolation protocol was implemented.

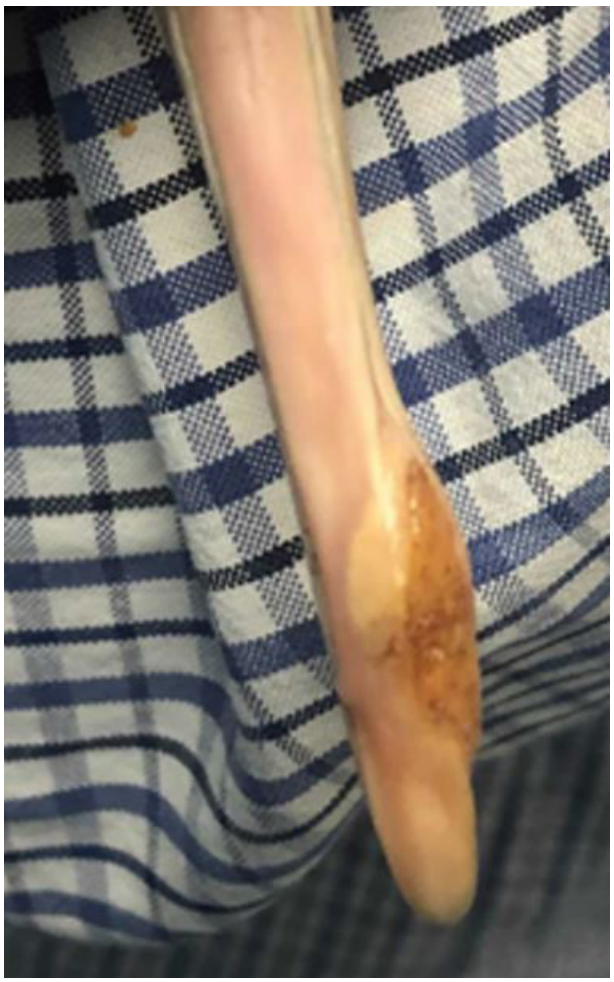

**Figure 1.** The APV bill lesion on the day of presentation at Wildbase hospital. Photo: National Kiwi Hatchery.

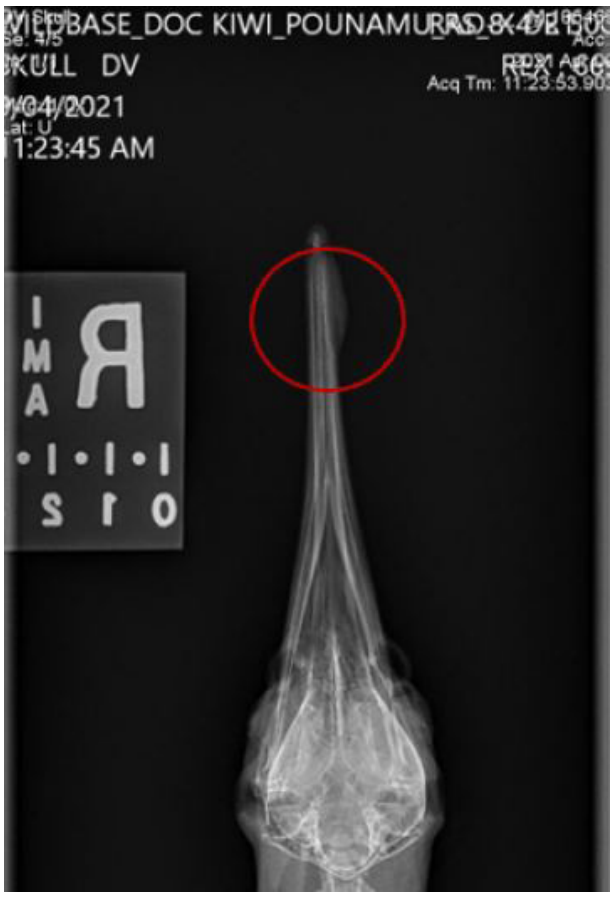


**Figure 2.** The initial dorsoventral radiograph of the head (lesion circled).

On day two post-admission, the kiwi was pre-medicated with 4 mg/kg of butorphanol (*Butorgesic*, 10 mg/ml, Troy Ilium, Glendenning, NSW, Australia) intramuscularly and was induced and maintained under anaesthesia with isoflurane (KB Isoflurane, 100%, Knight Benedikt Animal Health, Seven Hills, NSW, Australia) and oxygen administered via a mask. Ventrodorsal and lateral full body radiographs were taken with no abnormalities detected. Further dorsoventral, lateral and lateral oblique skull radiographs were taken and revealed evidence of bone lysis of the premaxilla beneath the lesion (Figure 2). Whilst the kiwi was still under general anesthesia, an 18-gauge needle was used to probe the lesion and allow closer inspection and a diagnosis of cutaneous avipoxvirus was made. The bone lysis identified on radiographs was most likely a result of pressure necrosis from the lesion being entrapped as has been previously reported in kiwi with avipoxvirus (Bean 2017). To allow the active lesion to erupt outwardly and relieve the pressure necrosis, the keratin cover was debrided and a small strip of adhesive Ioban™ (3M, St. Paul, MN) was applied over the lesion to provide a protective barrier. The kiwi recovered successfully with no complications during the anesthesia. Repeat dorsoventral, lateral and lateral oblique skull radiographs were taken on day 27 post-admission and the bone lysis of the premaxilla identified in the first set of radiographs was no longer evident.

On day seven of hospitilisation, a blood sample was obtained from the medial metatarsal vein. Biochemical analysis revealed a mild hypoproteinaemia (TPP 46 g/l, reference range 54-62 g/l) with a normal haematocrit (0.38, reference range 0.38-0.54), likely a result of dehydration (Morgan 2008). The white cell count was markedly elevated (55.7 x $10^9$ cells/l, normal reference range is 8.7 – 14.5 x $10^9$ cells/l, Morgan 2008) indicating a marked leukocytosis consistent with an active immune response. Examination of the blood smear also revealed cytoplasmic blebbing and dark discoloration of lymphocyte cytoplasms. Haematological testing was repeated at days 22, 27 and 34 post-admission and all parameters were within normal limits.

On day eight of hospitilisation diarrhoea was noted. A faecal exam including $ZnSO_4$ flotation was conducted at a commercial diagnostic laboratory but failed to identify the cause. The diarrhoea resolved without treatment within 24 hours.

Initial treatment goals included:

- Maintenance of hydration status with 15 ml of oral fluids (Compounded Sodium Lactate, Baxter Healthcare PTY LTD, Toongabbie, NSW, Australia) twice a day administered with a rubber crop tube.

- Provision of pain relief with 4 mg/kg of butorphanol (Butorgesic, 10 mg/ml, Troy Ilium, Glendenning, NSW, Australia) given intramuscularly twice a day for the first five days before transitioning to 10 mg/kg of tramadol hydrochloride (Tramadol oral suspension, 10 mg/ml, Biomed LTD, Auckland, New Zealand) administered orally twice a day for a further 6 days.
- Control of inflammation with a 3-week course of 1 mg/kg of meloxicam (Metacam oral suspension, 1.5 mg/ml, Boehringer Ingelheim Animal Health NZ LTD, Auckland, New Zealand) administered orally twice a day.
- Prevention of secondary infection with a one-month course of 125 mg/kg of amoxycillin/clavulanic Acid (Curam oral suspension, 250+62.5 mg/5 ml, Sandoz/Novartis NZ LTD, Auckland, New Zealand) administered orally twice a day and topical application of povidone iodine (Betadine, 10%, Sanofi-Aventis NZ LTD, Auckland, New Zealand) to the bill lesion twice a day.

Throughout hospitilisation, the bird remained bright, alert and responsive. The bird's nutrition status was maintained with a mince diet formulated for captive kiwi (80g per day) that was initially hand fed but once the bird overcame the stress associated with hospitilisation and pain was better controlled, food was consistently freely consumed. As a result, the bird's weight gradually increased from 786g to 1016g during the 37-day long hospitalisation period.

Over the 37 days in hospital, the bill lesion gradually healed with new keratin forming along the proximal border. Self-debridement of the remaining crust of the lesion through natural probing behaviours facilitated by probing boxes filled with soil and forest litter allowed some return to bill uniformity. However, abnormal growth of the surrounding keratin (Figure 3) meant that a bill reshaping procedure under general anesthesia was indicated by day 31 post-admission to encourage normal growth and prevent entrapment of foreign material under lifting keratin. The bird was anaethetised as previously described, with the addition of intubation for anaesthesia maintenance. A tapered grinding dremel (9.5 mm aluminum oxide grinding stone) was used at low speed to debride the firm keratin flaps and smooth the boarders of the lesion. Areas of soft keratin were cleaned with aqueous chlorhexidine gluconate (Chlorhex-C, 0.5%, Jurox Animal Health, Rutherford, NSW, Australia) on a cotton bud. No complications were experienced during anesthesia and the kiwi recovered well.

During the remainder of the kiwi's time in hospital, the healing of keratin over the lesion was closely visually monitored and kept clean with iodine as previously described. Discharge date was dictated by replacement of soft keratin with hard keratin in conjunction with a normal hematological profile.

The kiwi was discharged on day 37 post-admission. The lesion on the bird's beak responded extremely well to symptomatic treatment and mechanical debridement and was completely resolved at this time. The kiwi was considered no longer infectious at discharge, and the immunity to APV resulting from active infection should provide long-term protection and prevent him from contracting the disease again in the future (Bean, 2017). All medications had been stopped at the time of discharge, but the kiwi hatchery staff were instructed to monitor the appearance of his bill closely and to get in contact with any concerns of abnormal bill growth or signs of inappetence. The kiwi was released into the wild a few months later (Figure 4 shows the appearance of the bill at the site of the lesion immediately prior to release).

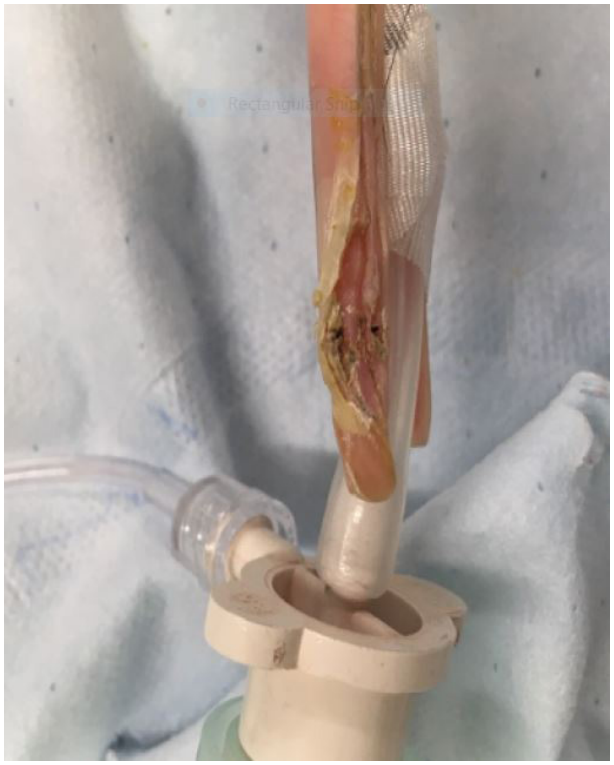

**Figure 3.** The bill pox lesion before reshaping with dremel mid-hospitilisation.

**Figure 4.** The healed pox lesion on the bill prior to release.

## Discussion

Initially, the top differential diagnoses for the bill lesion were trauma, cutaneous AVP or neoplasia such as squamous cell carcinoma. The initial appearance of the lesion looked like bruising consistent with trauma but the progressive expansion and eruption of the wound despite treatment was characteristic of a cutaneous AVP lesion (Bean 2017). Traditionally, APV has been diagnosed via gross and/or histologic examination of the lesions. Recent studies have used polymerase chain reaction (PCR) assays for diagnosis through the detection of the APV 4b core protein gene (Ha *et al.* 2013a).

In this case, based on characteristics of gross lesions, radiographic findings and haematological changes, the most likely diagnosis was an active AVP bill lesion with consequent pressure necrosis/reactive premaxillary bone caused by application of nail varnish, which prevented eruption of the lesion. Ideally a histologic sample would have been obtained during the initial debridement whilst at Wildbase, but definitive gross findings meant that this was decided against. Neoplasia such as squamous cell carcinoma was ruled out due to the positive response to treatment.

The bill of a kiwi holds a crucial role in navigation and foraging and thus, in order to survive in the wild, it must maintain functionality (Cunningham *et al.* 2013). The bill, however, is also delicate and due to the probing motion kiwi use to carry out these functions, the bill is very prone to injury (Cunningham *et al.* 2013). Originally it was thought that kiwi localised their prey using olfaction, but more recent studies have shown that kiwi have mechanoreceptors located throughout their bill and at the distal tip in a concentrated sensory pad which pick up the vibrations of prey moving in the soil (Cunningham *et al.* 2013). Olfaction instead appears to have a significant role in maintaining territories and communication between individuals (Castro *et al.* 2010).

When evaluating the kiwi bill histologically, hard keratin plates are divided by regions of soft keratin with increased density of sensory tissue in between (Cunningham *et al.* 2013). Normal wear and patterns of keratin growth are not well understood but may have significant clinical implications. Any bill injury or lesion deeper than 0.5 mm in the keratin layer has the potential to expose bone and thus the risk of osteomyelitis is high (Cunningham *et al.* 2013). Furthermore, the nasal cavity runs the full length of the bill meaning a lesion or injury at any level has the potential to disrupt respiration and olfaction.

In this case, the pox lesion resulted in a disruption to keratin growth and thus the functionality of the kiwi's bill was at risk. Intermittent reshaping of the beak and debridement of the lesion with a dremel prevented entrapment of organic material, encouraged correct keratin growth and prevented further pressure necrosis impacting underlying structures. The healing process was extensive with the kiwi spending 37 days in hospital but by the time of discharge, the lesion had fully resolved. Had treatment not been successful in keeping the bill clean, complications such as osteomyelitis could have developed. However, in the event of osteomyelitis, bill tip amputation would not be a viable option due to the importance of the sensory pad at the distal tip of the bill and implications associated with exposure of the nasal cavity thus preventing the kiwi's ability to feed properly and carry out normal behaviours. Had the lesion deteriorated to this point, euthanasia would have been the best option.

It is likely that the initial sealing of the lesion with nail varnish could have contributed to the trapped and inward growing nature of this lesion and consequent lysis of the premaxilla. This highlights the importance of the supportive care approach to cutaneous APV bill lesions in kiwi with minimal intervention and allowing for eruption of the lesion as part of the healing process.

Kiwi bill injuries are thought to be extremely painful, so analgesia is an important component of a treatment plan (Bean 2017). The best way to monitor pain in kiwi is regular evaluation of mentation and appetite and in this case neither posed a cause for concern indicating analgesic interventions were likely adequate (Bean 2017). However, it is important to take into account the impact of his preservation reflex on observations and interpretation.

Treating wildlife for long periods raises the question of welfare of the individual and likelihood of successful release after normal behaviours are curtailed in hospital. In this case, there was a tradeoff between complete healing of the lesion and levels of stress observed during treatment. Intermittent episodes of regurgitation and difficult handling were both points of concern. However, the absence of inappetence or weight loss resulted in a decision to monitor the kiwi closely and keep him in hospital until replacement of soft keratin with hard keratin was observed, in conjunction with a normal haematological profile. These decisions should be made on a case-by-case basis and if the prognosis and viability for release is poor, euthanasia should be considered.

Conservation efforts surrounding North Island brown kiwi are widespread and intensive. They include the hatch and captive-rearing of chicks in Operation Nest Egg facilities, the use of "kiwi crèches" and the release of birds into Kohanga sites, along with the translocation of wild birds to predator free islands and reserves (Colbourne *et al.* 2005). A huge amount of time, funds and skill goes into these efforts and they can be physiologically costly to the birds involved. APV has the potential to significantly impact these efforts if not approached and managed correctly, especially in captive-rearing facilities with high stocking densities and consequent viral shedding into the environment (Bean 2017). Pox lesions on the bill are most common in kiwi being reared in captive facilities due to the high stocking densities and consequent shedding of the virus into the environment (Bean 2017). The probing nature of the foraging habits of kiwi puts them in direct contact with this pathogen in soil (Bean 2017). Furthermore, captive-rearing facilities generally house young, naïve populations of kiwi that have no immunity against APV (Bean 2017). In a perfect world, antibody screening for the virus should

be implemented as a routine pre-translocation diagnostic test for kiwi, especially those being introduced into a naïve population (Ha *et al.* 2013b). As a baseline, kiwi should be routinely quarantined and monitored for development of characteristic APV lesions for approximately 30 days prior to release into a new population (Ha *et al.* 2013b).

Avipoxvirus preventative management changes should focus around reducing stocking rates of kiwi rearing facilities (and thus the risk of transmission between individuals); good hygiene and regular soil changes of enclosures in captive facilities to reduce the risk of environmental contamination; and minimising the potential for vector-borne transmission by mosquito proofing enclosures, eliminating sources of standing water and treating against ectoparasites. Recent studies have shown commercial fowlpox vaccines to be effective at providing protection against two wild strains of APV in passerines (Ha *et al.* 2013b). Demonstration of immunity to APV in kiwi after exposure to the vaccine is yet to be investigated and highlights the need for further research.

Effects of APV infection in wild birds are not as well documented as they are in domestic birds, but it is a known contributor to biodiversity loss, especially in regions with immunogenically naïve populations such as the Galapagos Islands, Hawaii and New Zealand (Atkinson and LaPointe 2009). Known effects of APV include but are not limited to, starvation; reduced reproductive rates; impaired ability to fly in flighted species; reduced hatching rates of chicks; immunosuppression; diminished foraging habits; and poor fitness (Ha *et al.* 2011). APV is increasingly becoming identified in both wild and domestic birds across the North and South Islands of New Zealand and has been documented as causing devastation to the black robin (*Petroica traversi)* and shore plover (*Thinornis novaeseelandiae)* populations (Ha *et al.* 2013b). Although mortality rates do not appear to be as high in North Island brown kiwi (Bean 2017), the widespread impacts of APV on kiwi populations is still very much unknown. The need to understand the prevalence of APV in wild kiwi populations and its clinical effects on infected birds, especially surrounding the growth and healing of bill keratin, along with development of treatment and prevention strategies is an important next step in the preservation of these species as the effects of climate change and concurrent vector-borne disease spread become more prevalent.

**Acknowledgements**

Special thanks to the team at Wildbase hospital and the National Kiwi Hatchery Rotorua, for their guidance and support.

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