Diagnosis and Clinical Management of a Little Spotted Kiwi (*Apteryx owenii*) with Karaka Berry (*Corynocarpus laevigatus*) Toxicity

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Background

The Little Spotted Kiwi (*Apteryx owenii*) is one of 6 species of Kiwi in New Zealand. It is the smallest of all the kiwi and, along with all 6 species, is threatened with an estimated 1400 birds remaining mainly on offshore islands such as Kapiti as well as a few smaller offshore islands (Robertson 2003) They feed mainly on earthworms, insect larvae, spiders and beetles but will also eat fallen fruit and seeds (Heather and Robertson 2006).

The karaka tree (*Corynocarpus laevigatus*) is native to New Zealand and is abundant in the North Island, especially lowland and coastal regions, and is limited to areas such as Nelson, Marlborough and Banks Peninsula in the South Island (Parton et al 2006). The kernel of the karaka fruit contains the toxin karakin, which also contains 3-nitropropanoic acid (3-NPA) (Shaw and Billing 2006). Further investigation of the fruit and foliage of karaka has revealed that karakin is accompanied by four other esters of 3-NPA; cibarian, coronarian, corynocarpin and corollin. There are also a further six unidentified nitro compounds (Majak and Benn 1994). Systemically, nitropropionic acid irreversibly inactivates succinate dehydrogenase, thereby blocking ATP formation (Anderson et al 2005). The clinical signs of Karakin toxicity are weakness, hind leg paralysis, and convulsions (Shaw and Billing 2006). The concentration of karakin observed in the kernels differs according to location and state of maturity, with factors such as climate, season and soil type possibly playing a role. Differences in severity of symptoms seen are likely due to these differences in concentration as well as the quantity of seeds eaten (Bell 1974). It is important to note that the flesh of the fruit is not toxic - only the seed kernel. It is an important food source for birds with Kererū (NZ wood pigeon - *Hemiphaga novaeseelandiae*) its most likely main dispersal agent. The toxic kernel was also an important food source for pre-European Maori who developed a method of steaming and steeping the kernel to remove the poison before grinding them to make bread (Sawyer et al 2003).

History

A 10-year-old, female Little Spotted Kiwi (LSK) was found recumbent on Kapiti island in March 2008. She was unable to stand or walk, and unable to hold her head upright. Supportive care was initiated by the Department of Conservation (DoC) rangers prior to sending the Kiwi to Raumati Vet clinic on the mainland. Supportive care was continued at Raumati Vets and consisted of IV Hartmanns with 4% Dextrose, Baytril,
Trimethoprim Sulphonamides, Thiamine, and Calcium gluconate. She was tube fed 3 times per day with Hills A/D. Radiographs and biochemistry and haematology were all within normal limits. The Kiwi was sent to the New Zealand Wildlife Health Centre at Massey University 5 days after being found recumbent on the island.

Case Report

Presenting neurological abnormalities included depression, torticollis, inability to stand, and bilateral absence of conscious proprioception.

Supportive care was continued with the inclusion of Augmentin 15mg/Kg (0.25ml) IV BID and twice daily force feeding of 50g Captive kiwi mix. The bird was place in an ICU unit at room temperature and monitored closely for signs of regurgitation and aspiration.

A blood sample was sent to New Zealand Veterinary Pathology (NZVP) for biochemistry and in-house white cell count was performed. Results were as follows; white blood cell count of $18.0 \times 10^3/L$ (ref: 10.3-18.1), with heterophilia, $14.8 \times 10^3/L$ (ref: 2.7-10.1) and eosinophilia, $0.7 \times 10^3/L$ (ref: 0.0-0.3). Increased CK 5154U/L and increased AST 304U/L most likely due to handling and lateral recumbency. All other biochemistry was within normal limits.

A complete avian neurological exam was performed with the following abnormal findings; weakness and ataxia (recumbency), torticollis, incoordination, loss of balance (could not stand), bilateral proprioceptive deficits, bilateral loss of menace and PLR, loss of muscle tone, bilateral loss of cutaneous sensation on all parts of leg, no withdrawal in right leg and bilateral knuckling of digits. She also has regular episodes of tonic (seizure-like) contractions where she thrashes around the ICU.

Radiographs were repeated at Massey but showed no significant findings. An ultrasound was performed the same day and showed a 2cm x 1cm foreign object in the gizzard. Emergency endoscopy was performed to examine the contents of the gizzard. Three karaka seeds were found and removed from the gizzard.

There has been one case reported by Shaw and Billing (2006) where they described possible karaka berry toxicoses in a number of North Island Brown Kiwi that were exposed to karaka berries but there was no evidence that the kiwi had actually eaten the berries. Clinical signs of karaka toxicoses were used to diagnose the toxicity in these cases. This paper, combined with the clinical signs and subsequent removal of karaka seeds from the gizzard of the LS kiwi lead us to the diagnosis of karaka seed toxicity.

Treatment of karaka seed toxicoses is symptomatic and supportive after removal of the seeds. In the cases reported by Shaw and Billing some of the Kiwi took up to 6 weeks to recover, so we used that as our benchmark for the LSK.

Supportive care at this stage included twice daily force feeding, IV fluid therapy (Hartmanns + 2.5% glucose), twice daily physiotherapy to try and prevent excessive muscle wasting as well as regularly turning her over in the ICU.

The removal of the karaka seeds had a remarkable effect on the Kiwi’s demeanour; she became much brighter and alert but showed no improvement in neurological signs the first 3 weeks in hospital and was still lateral recumbent with occasional tonic contractions. A CT scan was performed during this time to rule
out other causes of her symptoms but no abnormalities were detected.

She started to show some improvement in neurological signs about 5 weeks after being found on Kapiti. She was able to stand unassisted for a few seconds and subjectively her legs appeared to be getting stronger. She would still twitch and tremor when sitting upright but the signs of torticollis were markedly reduced. The IV fluids were discontinued and oral fluids started.

Over the next week (week 6) there was a tremendous improvement in the amount of time she was able to spend in an upright position unassisted. She still showed signs of central neurological deficits but was able to right herself well. Over the next 2 weeks she was able to maintain balance and remain upright for increasing periods of time.

About 8 weeks after presenting at Massey (27/4) she was shifted to a larger cage as her demeanour and amount of activity was much improved. She was still coping fine with force feeding but also started to forage for earthworms and eat small amounts of the captive kiwi diet that was left out for her. She was able to stand for extended periods of time but would still knuckle and fall forward when attempting to walk.

Over the following weeks she showed continual improvement in head carriage, posture and gait. It was noted that she would attempt to forage but seemed unable to eat the earthworms offered, possibly due to in co-ordination and inability to throw her head back to eat. 2 months after presenting at Massey she was moved to a large enclosure to allow her room to forage and move around. She would eat between 6-12g of Kiwi mince offered overnight.

She started showing increasing co-ordination when walking around the enclosure but would still fall over after being handled and replaced in the room. Daily physiotherapy was discontinued as she was no longer recumbent and the stress involved outweighed the advantages of physiotherapy. Because she was moving around her enclosure so well and maintaining weight, force feeding was decreased to once daily.

She continued to improve slowly but steadily in our care so it was decided to send her to Mt. Bruce, a Department of Conservation run sanctuary for native wildlife. Here she would have access to an outdoor, natural, predator-proof enclosure where she could be monitored by DoC staff and assessments made on her suitability for release back on Kapiti Island.

She has continued to improve while at Mt. Bruce although she still shows signs of in co-ordination after handling and will fall forward when replaced back in her enclosure.

She has a way to go before we can even consider release but the marked improvement from when she presented in lateral recumbency with severe neurological deficits to an almost normal kiwi are very promising.

References


Figure 1. A Little Spotted Kiwi

Figure 2. Ripe Karaka Fruit