UPDATE ON LORIKEET PARALYSIS SYNDROME

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Lorikeet paralysis syndrome (LPS), also known as clenched foot syndrome, has been recognized in Eastern Australia since the 1970s. Free flying rainbow lorikeets (*Trichoglossus haematodus*) present with unilateral or more commonly bilateral, flexed hocks and clenched feet and are unable to perch. They are usually resting on their hocks. Some birds may arrive collapsed and die within 1-3 days. Other birds continue to live and adapt to resting on their hocks. Only low numbers are known to recover completely and return to the wild. In inner-city Sydney, juveniles, 12-16 weeks of age, appear to be most commonly presented.

MATERIALS AND METHODS

Nine free-flying rainbow lorikeets were presented with signs of LPS. Eight were from the inner-city area of Sydney and one was found 90 km southwest of Sydney (Camden, NSW). Two of the nine birds were treated for heavy metal poisoning using intramuscular injections of CaEDTA (Calsenate® 200mg/ml Parnell Lab (Australia) PTY LTD) at 20 mg/kg and subcutaneous fluids q12h for 3-5 days. One of these two also received meloxicam (Metacam® 1.5mg/ml Boehringer Ingelheim (Australia) PTY LTD) orally at 0.15 mg/kg q12h. A third lorikeet was given a single injection of CaEDTA.

Blood lead concentrations were determined for al lorikeets from the inner-city using Leadcare II blood lead in-house analyser (ESA Biosciences, Chelmford, Massuchusetts). Six juvenile lorikeets from the inner-city (including two that had been treated with CaEDTA) and the lorikeet from Camden was euthanased by intravenous pentobarbitone sodium (Lethabarb® 325mg/ml Virbac (Australia) PTY LTD) and tissues were fixed in neutral-buffered formalin, embedded in paraffin and 4 µm sections were routinely stained with Haematoxylin and Eosin. In addition to soft tissues, the vertebral column and a leg of these birds was demineralised and serially sectioned. Kidney sections from two lorikeets that had not been treated were stained with a Zeihl-Neilsen stain to look for the presence of acid-fast inclusion bodies consistent with lead deposition in the kidney. Lead and zinc concentrations were determined on formalin-fixed liver samples from four birds that had not been treated with calcium EDTA.

RESULTS

Blood lead and tissue heavy metal concentrations

Blood lead concentrations ranged from 7.4 µg/dl to greater than 70 µg /dl. Liver concentrations in three birds were less than 10 µmol/ kg weight. One bird had a lead concentration of 11µmole/kg wet weight. Liver zinc levels ranged from 0.23 to 0.8 mmol/kg and were considered to be within the

normal range as compared to those found in poultry.

Histologic findings:

Metastatic mineralization. Moderate to severe multifocal metastatic mineralization with associated necrosis of the proximal tubules was present in one lorikeet. Three birds also had a moderate to marked zonal mineralization of the koilin. A moderate lymphoplasmocytic inflammation of the myoventriculus was present in one bird with the ventricular lesions, although the relationship between the mineralization and the inflammatory lesion was unclear. A mild to moderate multifocal mineralization was also found in the interatrial sepatae in the lung of a fourth bird.

Lesions caused by or potentially caused by viruses: A severe acute and diffuse necrotizing enteritis, pancreatitis, and nephritis were present in one bird that died spontaneously. Many cells within these lesions exhibited karyomegaly and the enlarged nuclei contained a pan nuclear amphophilic to eosinophilic dense inclusion. These inclusions are consistent with those caused by an adenovirus. Basophilic globule cells (Psittacine Beak and Feather Disease Virus inclusions) were present in the bursa of one of two birds that had bursal tissue submitted. Another lorikeet had a hypoplastic spleen, a lesion consistent with Psittacine Beak and Feather Disease Virus infection, but the bursa from this bird was not available to examine histologically. Diffuse papillomatous changes were found in multiple sections of the jejunum in two birds. These lesions closely resembled the mucosal papillomas associated with Psittacid Herpesvirus 1.

Lesions of the muscle and nervous system. The lorikeet from Camden had a moderate subacute diffuse degenerative myopathy of the muscles of the syrinx and a section of skeletal muscle. A prominent degenerative encephalomyelopathy was found in one bird. Lesions were most prominent in the midbrain at the base of the cerebellar peduncles and in the grey matter of the lumbar spinal cord. These lesions were associated with the loss of neurons, gliosis and in some spinal sections neuraxonal degeneration. Neuraxonal degeneration of the spinal cord was also seen in two other lorikeets to a lesser extent.

Acid fast staining: Two kidney sections were examined for the presence of acid fast inclusions consistent with heavy metal poisoning. Acid fast inclusions were not found.

Table 1. Distribution of microscopic lesions

	Metastatic Mineralization	Adenovirus	PBFDV*	Papillomas	CNS Disease	Muscle Degeneratio
Camden	Lung	No	Yes	No	Yes	Yes
Sydney 1	Ventriculus	No	No	Yes	Brain and Spinal Cord	No
Sydney 2	Kidney and Ventriculus	No	No	No	No	No
Sydney 3	Ventriculus	Yes	No	Yes	No	No
Sydney 4	No	No	No	No	Yes – very mild	No
Sydney 5	No	No	No	No	Yes - focal	No
Sydney 6	No	No	Suspicious lesions	No	No	No

DISCUSSION

Lorikeets commonly present to avian practitioners across Eastern Australia with clenched feet. They may appear bright but are unable to perch and walk on their hocks. In some cases birds present with clenched claws and moribund, a number deteriorate and do not survive. Reports in the literature suggest that effected birds can either be adults or juveniles, but in the authors' experience in the Sydney area, most are juveniles. Treatment regimes advocated by over the past 25 years for this syndrome have included vitamin E injections, vitamin B complex injections, CaEDTA injections, meloxicam orally and by injection, and corticosteroid injections. These medications have variably been combined with bicycling the legs as a crude form of physiotherapy. No overall successful response to any treatment has been recorded. The aetiology of this syndrome is not known, but may have more than one cause. Microscopically poliomalacia, a non-suppurative polioencephalomyelitis, lesions consistent with vitamin E/selenium deficiency and trauma have been reported (Hartley, 1995). The lorikeets in this study presented with a diverse array of lesions and confirmed diseases. Significantly, only the lorikeet from Camden had brain and spinal cord lesions similar to those commonly associated with LPS in other studies. No lesions specific for lead poisoning were seen, but microscopic lesions are not consistently seen in birds with lead poisoning. The most common lesion in these birds was metastatic mineralization of the gastrointestinal tract, lungs or kidneys. Possible caused for this lesion include high calcium diets, DDT exposure, and ingestion of toxic levels of vitamin D analogs (rodenticides) and cadmium toxicity. Vitamin D poisoning can also cause metastatic mineralization, but birds are relatively refractory to it.

All eight tested birds in this study were positive for exposure to lead using the Leadcare II test with blood concentrations of 10 µg/dl or greater and three lorikeets had marked markedly elevated lead levels (>70 µg/dl) as measured by this test. Lead poisoning is recognised as a cause of peripheral nerve dysfunction. Pet birds, including lorikeets, with lead poisoning, may show clinical signs of clenched foot and a similar sign called foot or wrist drop is seen in humans with lead poisoning (Thomson, 2006). Lead poisoning is considered clinically more significant in children as compared to adults (Bellinger, 2004). Lead poisoning occurs commonly in birds around the world and has been documented in flying foxes in Queensland (Skerrat, 1998). McOrist (1986) suspected that lead toxicity to be one of the causes of LPS in lorikeets from Sydney. However, blood lead levels were not

measured in these birds, and the liver lead levels were found to be low (McOrist, 1986).

The question then arises were the birds in this study actually experiencing lead poisoning or was there another cause of the signs? High concentrations of blood lead should be accompanied by detectable or high concentrations of lead in the liver (Ochiai, 1992). Yet this was not found to be the case in this study. The manufacturers of the Leadcare II test were contacted and indicated that cadmium, at high levels may cause the Leadcare II test to read positive, raising the possibility that these birds might have been exposed to cadmium or possibly some other cross-reactive contaminant. In mammals, metastatic mineralization of the kidney is a characteristic lesion associated with cadmium toxicity and thus our findings of metastatic mineralization in 4 of 7 birds in this study provides additional evidence that cadmium toxicity could be playing a role in LPS.

The significance of the other lesions and infectious agents found in these birds relative to their presenting signs remains unknown and may be entirely coincidental. One lorikeet had multifocal muscle degeneration which can be caused by vitamin E or selenium deficiency, but can also be the result of many other diseases. Vitamin E or selenium deficiency was the suspected cause of a myopathy seen in lorikeets at the Currumbin Sanctuary in South-East Queensland (Hartley, 1995). These birds had focal degenerative lesions were seen in cardiac and skeletal muscle.

Lesions of known or suspected virus aetiology were found in four lorikeets in this study. Given that these were caused by two and possibly as many as three different viruses, it is very unlikely that they are related to the LPS. Changes characteristic of adenovirus infection were seen in one bird. If adenovirus infection is confirmed with by other means such as electron microscopy, this is the first wild lorikeet in Australia to be described with this infection. Psittacine beak and feather disease was confirmed in one bird and suspected in another. Circovirus is not uncommon in free-flying lorikeets and is unlikely to be the major factor in LPS. Papillomatous changes were seen in the gastro-intestinal tract of two birds. These are the types of lesions normally seen in neotropical parrots with Psittacid Herpesvirus infections (Darrel, 2005). However, the lesions are more commonly found in the cloaca and the oral cavity. It is yet to be determined how common the papillary lesions seen in these birds' intestines are or what their distribution is. Future studies to probe fresh intestinal tissue for herpesvirus have been planned.

FUTURE LORIKEET PARALYSIS STUDY

A detailed protocol has been developed to pursue the evidence that have been developed in the early stages of this investigation. In addition to a complete and more extensive histological examination of tissues from lorikeets with LPS, serum biochemistry profiles will be done to look for evidence of muscle degeneration, electrolyte disturbances and evidence of kidney dysfunction. Tissues, including bone, will be screened for an array of heavy metals and if they are not found, screening for other toxins will be considered. Fresh tissues will be saved to screening for herpesviruses and all birds will be tested for infection with the beak and feather disease virus. Lastly, brain and spinal cord sections from birds with an array of lesions will be submitted for testing for bornavirus antigens.

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