Nutritional myopathy in free-living lorikeets

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Introduction

This paper deals with a condition commonly seen in free flying, wild lorikeets in South East Queensland. It is commonly called lorikeet "paralysis" or "cramps". Affected birds are mainly Rainbow lorikeets (*Trichoglossus haematodus*) and Scaly-breasted lorikeets (*Trichoglossus chlorolepidotus*). These birds are usually young or sub-adult and are often found on the ground, being unable to fly or only flutter weakly. Some may show partial wing paralysis. They are often in poor condition and quite thin. The legs are held in extensor rigidity with the toes clenched and they are unable to perch. The name Lorikeet Nutritional Myopathy (LNM) is proposed for this syndrome.

Incidence: Age and Season

Affected birds are usually young, sub-adults. Many are fledglings as evidenced by the dark brown beak. Adults have a bright orange/red beak. Very rarely are birds over 18 months old affected.

The seasonal incidence of LNM reflects the birds breeding pattern. In SE Queensland, the main breeding season for these lorikeets is from August to January, although fledglings have been recorded in all months. The number of affected birds presented reaches a high during this breeding period.

Table 1. Number of sick, injured, orphaned lorikeets presented to Currumbin Sanctuary in 12 months (July 1993 to June 1994)

		Rainbow	Scaly-breasted	
1993	July	25	13	
	August	30	8	
	September	55	14	
	October	56	27	Principal
	November	90	6	Breeding
	December	69	22	Season
1994	January	73	22	
	February	35	25	
	March	74	15	
	April	58	8	
	May	31	8	
	June	22	4	
Total		**618**	**172**	

The age ratio of these birds is approximately 60/40 - adults to juveniles - although as mentioned, the incidence of juveniles increases during and just after the breeding season. The incidence of leg paralysis seen in these juveniles is over 50%, so over 100 affected birds are presented yearly. This is obviously a disease of young, growing birds which are affected at a time of maximum nutritional requirement, when essential nutrients are needed for growth, muscle development, feathering etc.

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Clinical Signs

Most affected birds are quite young (fledglings) and show the typical brown beak of immature lorikeets. As the birds mature, the beak tip shows yellow markings which develop into a red/orange colour which progressively moves up the beak. By 10 to 12 months of age, the birds have an orange/red beak, and by 18 months, the vibrant coral colour of the adults is attained. Rarely are birds over 12 months of age affected.

Typically presented birds are usually noticeably thin with a prominent keel bone - having only 30 - 60% of the normal pectoral muscle mass and are unable to fly - or only flutter weakly. They are usually found on the ground. Many of these birds have concurrent problems - i.e. PBFDS - which in lorikeets, usually involves loss of the primary flight feathers and the tail feathers - making flying even more difficult.

The legs are characteristically held in extensor rigidity with the toes clenched. They are unable to perch. The legs can be manually flexed - sometimes with difficulty. The toes can be opened and the birds will clench an appropriate object but usually cannot unclench and release their grasp. The muscle mass of the legs is usually reduced in size and atrophy is noticeable.

Post Mortem

These birds often have poor feathering. Many show obvious signs of PBFDS with loss of flight and tail feathers. Interestingly, many show signs of feather cysts (as seen in canaries) where the developing feather fails to erupt and grows subcutaneously, promoting a foreign body type reaction, with subsequent cyst formation. They often harbour lice and even, occasionally, keds and generally show signs of a decreased immune response. They often have obvious signs of oral, fungal infections - *Candida* spp; enteric coccidiosis and *Capillaria* infestation.

The skin in most normal healthy birds is opaque due to dermal fat stores. In these birds, transparent skin and a prominent keel bone show weight loss. The characteristic gross pathology is seen in the skeletal muscles. In most normal birds the pectoral and leg muscles are a plump red-brown. Here we see a pallor or a pale streaking which is a reflection of muscle necrosis and cellular inflammatory infiltrate.

Histopathology

The characteristic pathology is seen in the skeletal (striated) muscle and often in the myocardium. In mild cases, sections of skeletal muscle (hind limb) show individual muscle fibres to be smaller than normal and surrounded by a clear zone separating the muscle fibres. These muscle fibres stain more eosinophilic and often have lost their cross-striations.

As the condition advances, the leg muscle sections show multifocal areas of degeneration with increased eosinophilia and margination of nuclei. There is prominence and an increased number of sarcolemmal nuclei and interstitial infiltration of inflammatory cells and early interstitial fibrosis. Similar changes are seen to a lessor degree in the pectoral and cardiac muscles.

In advanced cases, there is widespread degeneration of muscle fibres with fragmentation, eosinophilia and loss of cross striations. There is proliferation of sarcolemmal nuclei with giant cell formation. Many muscle fibres may be mineralised and sections of cardiac muscle show degenerative changes associated with loss of cross-striation, intense eosinophilic reaction and proliferation of sarcolemmal cells.

This scenario is similar to White Muscle Disease - Selenium/Vitamin E. deficiency which is characterised by discrete multifocal areas of muscle degeneration with fibrous replacement of affected muscle fibres.

Early in the disease, affected muscle fibres become swollen, lose their cross striations and often exhibit nuclear degeneration. Mononuclear inflammatory leucocytes may migrate into and around the affected muscle fibres as they undergo necrosis

In several cases - both natural deaths and sacrificed birds, the brain, spinal cord and peripheral nerves (sciatic) were dissected from fresh specimens, placed in 10% neutral buffered formalin and submitted for histopathology - in all cases no pathological changes could be found in any areas of neurological tissue. This contrasts with cases reported by Dr. W. Hartley of Taronga Zoo Wildlife Pathology Register. He found lorikeets showing signs of leg paralysis from the Sydney area, when examined histologically, showed a spongy appearance of the ventral horns containing the motor neurones of the spinal column with demyelination and death of many neurones. This form of paralysis is suspected to be viral, related and associated perhaps with a B-vitamin deficiency component. The form of lorikeet paralysis seen in SE Queensland appears to have no neurological component and seems to be a myopathy caused by a nutritional deficiency - hypovitaminosis E.

Biochemistry

In the light of the histopathological findings, it was decided to look at the normal blood values of these lorikeets. The normal blood parameters of many Australian native birds are unknown and many laboratories processing avian blood, use poultry values as their standard.

The blood values of normal wild flying lorikeets were measured. Fifteen Rainbow and seven Scaly-breasted lorikeets were caught - the average ratio of occurrence of these birds at Currumbin Sanctuary. Within 2 hours of capture the birds were anaesthetised using a face mask and 2% halothane/oxygen and bled from the right external jugular vein. Approximately 0.5 to 1 mL of blood was collected and placed in sterile blood tubes. This blood was refrigerated and then sent for analysis.

Table 2

Blood Parameters measured in 15 free-flying wild Rainbow lorikeets (*Trichoglossus haematodus*)

Parameter	Mean	S.D.	Range
PCV (L/L)	0.51	0.045	0.38 - 0.54
AST (u/L)	359	93.0	196 - 389
ALT (u/L)	218	104	75 - 446
CPK (u/L)	776	178	403 - 1034
T.Bili (ymol/L)	35.6	19.7	6.5 - 49.2
T.Protein (g/L)	30.3	5.62	23.41
Albumin (g/L)	17	3.0	15 - 24
Creatine	0.03	0.013	0.01 - 0.05
Uric acid (umol/L)	170	86	100 - 310

Table 3

Blood Parameters measured in 7 free-flying wild Scaly-breasted lorikeets (*Trichoglossus chlorolepidotus*)

Parameters	Mean	S.D.	Range
PCV (L/L)	0.51	0.04	0.48 -0.59
AST (u/L)	358	152	64 - 543
ALT (u/L)	196	151	28 - 474
CPK (u/L)	1087	279	570-1404
T. Bili (umol/L)	23.9	7.8	11.1 -37.5
T. Protein (g/l)	30	5.0	23 - 35
Albumin (g/L)	15.8	1.9	13 - 18
Creatine	0.04	0.021	0.02 -0.08
Uric Acid (umol/L)	170	97	100 - 310

To check that the effects of capture and handling did not significantly affect these blood values, 6 Rainbow and 6 Scaly-breasted lorikeets were retained in captivity for 2 weeks. They were housed in a 4m x 3m x 2m high cage, partially sheltered. They were provided with daily fresh water, a nectar mix of honey/water/complan, medium parrot seed, chopped greens (apple, carrot, celery, sweet corn and sprouted wheat) and fresh browse. After 2 weeks blood samples were collected as before and no statistical difference was found in any blood parameters.

These blood values provided a useful data base for lorikeets and serum CPK was chosen as the best indicator of muscle damage. Birds affected with nutritional myopathy showed far greater changes in CPK values when compared with birds suffering traumatic muscle injuries - i.e fractured wings and legs.

Table 4

Representative samples of serum CPK values in Lorikeets with traumatic injuries. (Blood was collected within 24 hours of presentation.)

Injury	Species	CPK(u/l)	Normal CPK (U/L)
Fx. R. Wing	Rainbow.	3382	776
Fx. R. Wing and beak	Rainbow.	922	776
Fx. L. Wing & L. leg	Rainbow	1624	776
Fx. L. Wing	S'Breasted	2623	1087
Fx.beak&head trauma	S'Breasted	6124	1087

It appears that with traumatic muscle injuries CPK levels may rise by 5 times normal. In contrast lorikeets suffering myopathy, show far greater elevations in serum CPK(see table)

Table 5

Serum CPK levels in lorikeets showing varying degrees of leg paralysis.(blood collected within 24 hours of presentation)

Condition	Species	CPK(u/L)	Normal CPK (u/L)
Mild	Rainbow	17497	776 (x20)
Moderate	Rainbow.	38254	776 (x50)
Severe	Rainbow.	72610	776 (x94)
Advanced	Rainbow.	1,157,400	776(x1500)
Mild	S'Breasted	17864	1087 (x16)
Severe	S'Breasted	36,620	1087 (x33)

Lorikeets affected with myopathy consistently showed CPK elevations of at least 20 times to 100's of times normal values.

Treatment

Affected birds were housed in hospital cages. Warmth (in the form or heat lamps or human humidicribs) was provided where necessary. The birds were offered a diet of fresh water, nectar mix - honey/water/complan, chopped greens and fresh native flowers (grevillia, melaleuca etc.) where available.

The nectar mix was supplemented with Vitamin E. powder (White E - Vetsearch) approximately 80gm/litre, providing 5 i.u. Vit. E/mL of Nectar. The nectar mix consists of 1 part honey and 9 parts water made up to 1 litre. To this is added 60gms. Complan (Boots Company (Aust) Pty.Ltd.) This mix is prepared fresh daily

Limited physiotherapy, in the form of limb massage and extending and flexing the legs was performed.

Concurrent problems were also treated. These commonly included :-

Intestinal coccidiosis	Toltrazuril (Baycox - Bayer: 25g/l) at 1mL/litre for 5 days
Capillaria spp.	Netobimin (Hapavet - Vetafarm 35 mg/g for 5 days) or Avermectin B (Avomec Injection for Cattle - MSO Ag Vet) at 200ug/kg *per os*, repeated in 1 week

Birds which showed signs of severe weight loss were supplemented with PolyAid (Vetafarm) via crop needle.

Injectable vitamin E (Hardock's Vit. E. - International Animal Health Products - 400 mg Alpha Tocopherol/mL) at dose of 0.1 mL I/M repeated weekly, was given to several birds. These birds showed no greater improvement or improved prognosis compared to birds receiving the oral supplement. Recent work by Mainka *et al* on alpha tocopherol in Swainson's Hawks may explain this. They found I/M injections of Vit. E. resulted in no statistically significant changes in circulating alpha tocopherol concentrations up to 7 days following administration. Oral supplementation of Vit. E. with food may also result in slower absorption because of effects of dilution and digestion or to Vit. E. destruction in the digestive tract. Dietary fat may increase the absorption of Vit. E. from the gut - i.e. Vit. E. with fat and without food appears to give a faster and more efficient increase in circulating Vit. E. With this in mind, the Sanctuary is now orally dosing birds with a Vit. E./Canola oil mixture and withholding food for 2 hours post dosing.

Selenium/Vit. E injections were tried with no better results than injectable Vit. E alone. Some birds developed injection reactions and abscesses at the injection site when Selenium/Vit. E. was used.

Prognosis was directly related to serum CPK levels, the higher the level, the worse the prognosis and the longer the recovery period. A CPK level of approx 70,000 u/l appeared to be a cut off point. Below this level, birds usually recovered in about 2 to 3 weeks. Above this level, very few ever recovered, suffering from irreversible muscle damage and subsequent fibrous replacement of necrotic muscle fibres and scar tissue development. The legs remaining in permanent extensor rigidity with locked joints and clenched toes.

Discussion

S.E Queensland is presently undergoing a development boom, with the Brisbane - Gold Coast corridor showing the fastest growth of almost any region in Australia. The end result of this is a massive loss of native habitat as huge areas are cleared for development, housing etc. It's the usual story of habitat destruction impinging on the well being of native fauna.

The lorikeet's natural diet is primarily pollen and nectar. They also eat blossoms,berries and other fruit, ome seeds and insects and their larvae. T shrinking native resources are unable to provide the large lorikeet population with their natural diet.

During the breeding season, each pair usually lays 2 eggs, therefore the population has the potential to double at this time. With their natural food supplies dwindling and barely able to support the adult population, parents feeding chicks rely more and more on backyard bird feeders. The backyard feeding of lorikeets is very popular in S.E. Queensland with thousands of people providing honey water or sugar water mixes to free flying lorikeets. Apart from the problems of hygiene and disease transmission, the biggest worry is that these diets are not nutritionally complete. Sugar water or honey water approximates the nectar component of the diet, containing mainly carbohydrates and low amounts of Vit. B. complex. The pollen component provides most of the protein, minerals and vitamins, especially oil solubles like Vit. E. Chicks raised on a predominantly honey water diet will show severe growth and developmental problems due to deficiencies of essential nutrients. The complan contained in the nectar mix provides all of these required nutrients. There are also numerous proprietary brands of both wet and dry lorikeet mixes available commercially.

The solution appears obvious - PLANT MORE NATIVE TREES!!! This would benefit ALL wildlife, not just lorikeets. Also it is necessary to educate the public to understand that the birds need a nutritionally complete diet and that honey water mixes are deficient.

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