# LEAD TOXICOSIS IN BIRDS – A RETROSPECTIVE REVIEW OF 20 CASES

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**ABSTRACT**

Lead toxicosis is a common clinical problem in avian medicine. This paper reviews 20 cases, diagnosed with lead toxicosis, presented to the West Toowoomba Veterinary Surgery in the period 2006-2009. Clinical signs, blood lead levels and clinical pathology were evaluated in light of the clinical outcome in an attempt to determine positive and negative prognostic indicators. No consistent indicators were found.

**ABBREVIATIONS**

| | |
|---|---|
| AST | Aspartate aminotransferase |
| BID | twice daily |
| CK | Creatine kinase |
| IM | intra-muscular |
| Pb | lead |
| PCV | Packed Cell Volume |
| TP | Total protein |
| UA | Uric acid |

**INTRODUCTION**

Lead is a common environmental contaminant, with birds able to access it from a wide range of sources, including lead shot, leaded petrol and oil, galvanised wire, lead-based paints, lead putty, solder, foil from some champagne and wine bottles, some welds on wrought-iron cages, lead weights (e.g. curtain and fishing weights), bells with lead clappers, lead-lighted glass, improperly glazed ceramics, car batteries, bird toys with lead weights, costume jewellery, and even mirror backs. (LaBonde, 1995) The curious and investigative nature of many birds, especially pet parrots, results in the ingestion of lead particles.

Once ingested, lead is solubilised in the ventriculus by the combination of its grinding action and the low pH (2-3.5). Once solubilised, lead is absorbed across the intestinal mucosa and is bound to metallothionein (metal binding protein) for distribution around the body. It can be stored in both bone and soft tissues. When circulating levels exceed a toxic threshold (either through the ingestion of a large amount in a short period of time or through the cumulative effect of chronic ingestion of small amounts), two major target organs are affected: the nervous system and the erythropoietic bone marrow.

Lead exerts its effects in several ways. It competes for calcium at the myo-neurological junction resulting in neuromuscular blockage. It affects neuronal cell adhesion molecules and glucocorticoid

receptors in neurones and glial cells, leading to axonal demyelination. These two effects account for the neurological signs often seen in birds with lead toxicosis (paralysis, paresis, ataxia, seizuring, twitching, gastrointestinal ileus, changes in mentation). Lead also suppresses aminoleuvulinic acid dehydrase (ALAD) and haeme synthetase, leading to an accumulation of aminoleuvulinic acid and protoporphyrin IX in erythrocytes. This in turn inhibits the haeme synthesis cascade and leads to decreased erythrocyte production and increased erythrocyte destruction, resulting in intravascular haemolysis and anaemia. This effect accounts for the lethargy and weakness seen in many patients, and the haemoglobinuria often associated with lead toxicosis in galahs, amazons and Grey parrots (Degernes, 1995; Katavolos, 2008; LaBonde, 1995).

Diagnosis of lead toxicosis is best achieved by measuring blood lead levels. As 99% of circulating lead is in contained in erythrocytes (Katavolos, 2008), blood lead determination is a reliable indicator of toxicosis. There is still debate as to what constitutes a 'non-toxic' blood lead level. It might be better to say that below certain levels, clinical signs are less likely to be seen. What effect these 'non-toxic' levels are having on the patient is difficult to assess. The currently accepted 'non-toxic' blood level is <0.7 umol/L (14.5ug/dL, 0.145 ppm). Levels between 0.7 – 1.7 umol/L (35.2 ug/dL, 0.352 ppm) suggest exposure, while levels >1.7 umol/L confirm lead toxicosis.

Radiology is often recommended as a means for diagnosing lead toxicosis. However the presence of radio-opaque material in the gastrointestinal tract, while suggestive of heavy metal toxicosis, does not confirm that the material is lead. Conversely, the cumulative nature of lead means that an absence of these particles cannot exclude a diagnosis of lead toxicosis.

Clinical pathology is usually non-specific. Haematology often reveals a hypochromic, regenerative anaemia, frequently with erythrocytic ballooning. Many patients have elevated AST, CK and uric acid, reflecting increased protein catabolism and renal dysfunction. In waterfowl, inhibition of D-ALAD (Delta-aminoleuvulinic acid dehydrogenase) to levels <86 IU/L is suggestive of lead toxicosis. Other than D-ALAD, none of these changes can be said to be pathognemonic of lead toxicosis (Degernes, 1995; Katavolos, 2008; LaBonde, 1995).

Treatment for lead toxicosis revolves around three principles: removing lead from the soft tissues and bones through chelation therapy; preventing further absorption of lead from the gastrointestinal tract and exposure to the environmental source of lead; and providing supportive care until the chelation therapy has had an effect.

Calcium EDTA (20-40mg/kg IM) is a commonly used chelating drug. Metal ions (including lead) displace the calcium in calcium EDTA to form a water-soluble complex that is excreted in the urine. The author prefers to treat twice daily till the patient is asymptomatic (usually 3-5 days), then twice weekly till blood lead levels are in the 'normal' range – often up to 6 weeks. Response is usually rapid, with a clinical improvement usually noticeable within 24-36 hours.

Most small particles will pass unassisted through the gastrointestinal tract in 4-5 days. Larger pieces may have to be retrieved endoscopically or by concurrent flush and suction with warmed water or, as a last resort, a proventriculotomy can be performed. Gavaging liquid paraffin or peanut butter is often recommended, but its effect is questionable and its use contra-indicated in patients with gastrointestinal ileus or vomiting.

Supportive care may involve fluid, nutritional and thermal support. A blood transfusion may be required in very anaemic patients.

This paper reviews 20 avian lead toxicosis cases presented to the West Toowoomba Veterinary Surgery in the period 2006-2009. Clinical signs, blood lead levels and clinical pathology were evaluated in light of the clinical outcome in an attempt to determine positive and negative prognostic indicators.

**RESULTS**

The medical records of twenty birds diagnosed with lead poisoning were obtained from the computer files of the West Toowoomba Veterinary Surgery. The sole selection criterion was a blood lead level greater than 1.7 umol/L, as measured on a LeadCare II blood lead analyser (ESA Biosciences, Chelmsford, Massachusetts). The birds included a selection of parrots, poultry and a peacock (see Table 1).

The clinical signs displayed by these birds are shown in Table 2. On average, most birds had been observed as unwell by their owners for 3-4 days prior to presentation. Thirty-five percent were presented for weakness and lethargy; on examination 40% of the total cases showed neurological signs (twitching, paresis, seizures, etc), 10% had gastrointestinal signs (vomiting, diarrhoea) and 20% had signs consistent with renal disease (polyuria, haemoglobinuria). Three birds (15%) were presented for feather damaging behaviour. One bird (5%) displayed no obvious clinical signs.

Of the 20 birds presented, haematology and biochemistry profiles were performed on 15 cases. (The owners of the remaining 5 cases declined further testing.). The results of this testing are summarised in Table 3. Blood lead levels varied from 3.1 umol/L to levels greater than 50umol/L (apparently the highest limit of detection on the LeadCare II system). The PCV was below 40% in 31% of the cases, and elevated above 55% in 13% of cases (presumably due to dehydration). AST was elevated in 33% of the cases, always with a concomitant elevation in CK. UA was elevated in 21% of the cases.

None of the birds were radiographed prior to treatment, although several were radiographed after the diagnosis to ascertain if large particles were present in the ventriculus. In these cases, no particles were visible in radiographs..

All birds except one (No. 12, chicken, which died shortly after admission) were treated with calcium EDTA and supportive care as described above. The outcome of these cases is shown in Table 4. Of the 19 birds that received treatment, 74% survived.

There were no appreciable differences between the birds that survived and those that died in the average blood lead levels and PCV. All of the birds that died had elevated CK, but so did 50% of those that lived. UA was elevated in 20% of those that died and 13% of those that lived. These differences are summarised in Table 5.

**DISCUSSION**

Lead toxicosis is a common problem in avian practice; when the high level of environmental lead contamination in modern society is combined with the inquisitive nature of the parrot, lead poisoning is the inevitable consequence.

The clinical signs frequently described in the literature were observed in many, but not all, of the birds in this review. While neurological signs are commonly associated with lead poisoning, only 40% of these cases showed neurological signs. (It is possible, however, that the birds presented for weakness

and lethargy were displaying altered mentation, making the incidence of neurological signs higher than 40 %.) Polyuria and haemoglobinuria are frequently described, but only 20% of these birds displayed signs consistent with renal disease. Again, vomiting and diarrhoea are often mentioned, but only 10% of these birds displayed these signs. These findings suggest that, while neurological, renal and gastrointestinal signs should lead the clinician to consider lead toxicosis, their absence does not rule it out.

There was no correlation between blood lead levels and the severity of the disease and eventual outcome. In other words, a low level of blood is not a positive prognostic indicator, nor is a high level a negative indicator.

While clinical pathology can give the clinician information on the patient's health status, it does not help to make a diagnosis of lead toxicosis, nor does it provide any prognostic indicators. The presence of a regenerative anaemia is suggestive of lead toxicosis but was present in only 31% of these cases.

Although not used consistently in the diagnostic evaluation of these birds, radiology does not appear to assist in the early diagnosis of lead toxicosis. An absence of radio-opaque particles in the gastrointestinal tract does not exclude lead toxicosis, but radiology may be of value in determining treatment requirements once a diagnosis has been made.

Based on this review, lead toxicosis can be treated successfully in approximately 75% of cases. Nevertheless, 25% will die despite treatment, and this needs to be communicated to clients before treatment commences. Clinical signs, blood lead levels and clinical pathology do not consistently indicate the severity of the disease or its outcome.

**REFERENCES**

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LaBonde J. (1995) Toxicity in pet avian patients. *Sem Avian Exotic Pet Med* **4**: 23-31

| Species | Number Seen |
|---|---|
| Eclectus Parrot (*E roratus polychloros*) | 1 |
| Galah (*Eolophus roseicapillis*) | 7 |
| Chicken (*Gallus gallus*) | 2 |
| Peafowl (*Pavo cristatus*) | 1 |
| Duck (*Anas domesticus*) | 1 |
| Sun Conure (*Aratinga solstitialis)* | 1 |
| Sulphur-Crested Cockatoo (*Cacatua galerita galerita*) | 1 |
| Yellow Tailed Black Cockatoo (*Calyptorhynchus funereus*) | 1 |
| Rainbow Lorikeet (*Trichoglossus haematodus*) | 1 |
| Budgerigar (*Melopsittacus undulatus*) | 1 |
| Cockatiel (*Nymphicus hollandicus*) | 3 |

**Table 1:** Species presented to the West Toowoomba Veterinary Surgery with lead toxicosis, 2006-2009

| | Species | Weak | Neurological | Gastrointestinal | Renal | Feather | No signs |
|---|---|---|---|---|---|---|---|
| 1 | Eclectus | x | x | | | | |
| 2 | Galah | x | | | | | |
| 3 | Galah | | | x | | | |
| 4 | Galah | | | | x | x | |
| 5 | Galah | | | | x | | |
| 6 | Galah | | | | | x | |
| 7 | Galah | x | x | | | | |
| 8 | Galah | | | | | | x |
| 9 | Peacock | | x | | | | |
| 10 | Chicken | | x | | | | |
| 11 | Duck | | x | | | | |
| 12 | Chicken | x | x | | | | |
| 13 | Sun conure | | x | | | | |
| 14 | Sulphur-Crested cockatoo | | | | x | | |
| 15 | Yellow-Tailed Black cockatoo | x | | | | | |
| 16 | Rainbow Lori | | x | | | | |
| 17 | Budgerigar | | | | | x | |
| 18 | Cockatiel | x | | | | | |
| 19 | Cockatiel | | | x | x | | |
| 20 | Cockatiel | x | | x | | | |
| **Totals** | | 35% | 40% | 10% | 20% | 15% | 5% |

**Table 2**: Clinical signs

| | Species | Blood lead (Umol/L) | PCV (%) | AST | CK | UA | TP |
|---|---|---|---|---|---|---|---|
| 1 | Eclectus | 24.8 | 43 | N | N | N | N |
| 2 | Galah | H | 40 | - | - | - | - |
| 3 | Galah | 37.1 | 50 | N | N | N | N |
| 4 | Galah | 3.5 | 45 | N | N | H | N |
| 5 | Galah | 23.6 | 28 | H | H | H | H |
| 6 | Galah | 18.8 | 57 | N | N | N | N |
| 7 | Galah | H | 30 | N | N | N | N |
| 8 | Galah | 4 | - | H | H | N | L |
| 9 | Peacock | 4.6 | 45 | N | H | N | H |
| 10 | Chicken | H | - | - | - | - | - |
| 11 | Duck | 3.1 | 45 | N | H | N | N |
| 12 | Chicken | 10.4 | - | - | - | - | - |
| 13 | Sun Conure | H | 26 | N | H | N | N |
| 14 | SCC* | 11.8 | 45 | N | H | N | N |
| 15 | YTBC* | 11.7 | 32 | H | H | N | N |
| 16 | Rainbow Lori | H | 45 | H | H | H | L |
| 17 | Budgerigar | 4.1 | 65 | H | H | N | N |
| 18 | Cockatiel | H | 48 | N | N | - | N |
| 19 | Cockatiel | 45.8 | 30 | - | - | - | - |
| 20 | Cockatiel | 3.8 | - | - | - | - | - |
| **NORMAL** | | | 9/16 | 10/15 | 6/15 | 11/14 | 11/15 |
| **HIGH** | | | 2/16 | 5/15 | 9/15 | 3/14 | 2/15 |
| **LOW** | | | 5/16 | - | - | - | 2/15 |

Table 3: Blood lead levels, haematology and biochemistry results.
(H = high; L = low, N = normal)

SCC = Sulphur-crested cockatoo
YTBC = Yellow-tailed black cockatoo

| | Species | Survived | Died |
|---|---|---|---|
| 1 | Eclectus | x | |
| 2 | Galah | x | |
| 3 | Galah | x | |
| 4 | Galah | x | |
| 5 | Galah | | x |
| 6 | Galah | x | |
| 7 | Galah | x | |
| 8 | Galah | x | |
| 9 | Peacock | x | |
| 10 | Chicken | x | |
| 11 | Duck | x | |
| 12 | Chicken | | Died |
| 13 | Sun Conure | | x |
| 14 | Sulphur –Crested | | x |
| 15 | Yellow-tailed Black | x | |
| 16 | Rainbow Lori | | x |
| 17 | Budgerigar | x | |
| 18 | Cockatiel | x | |
| 19 | Cockatiel | x | |
| 20 | Cockatiel | | x |
| **Totals** | | 14/19 | 5/19 |

**Table 4**: Outcome of 20 cases of lead toxicosis

| Outcome | Pb (average; Umol/L) | PCV (average; %) | AST | CK | UA | TP |
|---|---|---|---|---|---|---|
| **Lived** | 25.9 | 40 | 75% Normal | 50% High | 81% Normal | 75% Normal |
| **Died** | 22 | 41.8 | 60% High | 100% High | 80% Normal | 80% Normal |

**Table 5.** Differences between groups of birds that lived and died