# Suspected Aflatoxicosis in a Black Capped Lory, Causing a Chronic Hepatopathy

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### *History*

A mature hen, approximately 4 years of age, had been purchased 12 months previously from another breeder. It had not bred with the current owner and it's true breeding history or age was unclear. Six weeks prior to presentation at our hospital, it had been presented to another clinic. The owner felt then, that the bird was "not quite right".

The initial clinical workup including gram stains, CBC and biochemistry was unremarkable except for a higher than expected number of clostridial spores in the faeces. Clinically, the bird appeared to be normal. The Owner requested that the bird be kept "until it was better".

A four-week course of clavulanic acid/amoxil (Clavulox®) was instituted, and the Gram stains returned to normal in this time. The bird was sent home, the Owner felt the bird was improved. Two weeks later, the Owner felt that the bird had relapsed. It was still eating well but seemed a bit breathless and quieter than normal. It was then presented to our hospital.

### *Examination*

On physical examination, the bird weighed 210gm, had 1/5 muscle atrophy of pectoral muscles, increased respiratory efforts and a swollen abdomen. Abdominocentesis revealed a straw-coloured fluid, with a TP of 30gm/L and a specific gravity of 1.025. Gram stain showed no bacteria and Diff quick showed a low cellularity (6 cells/HPF).

**Fluid analysis;** Modified transudate**.**

Abdominal radiographs showed generalised opacity of the abdomen indicative of generalised ascites. It was not possible to discern the shapes of individual organs. The heart appeared to be a normal size and shape.

**CBC and Biochemistry**

| Parameter | Result | Normal value | Flagged |
|---|---|---|---|
| PCV | 32% | 25-45 | |
| TPP | 28g/l | 30-70 | low |
| WCC | $45x10^9$ | 8-20 | High |
| Heterophils | 51% | 40-70 | |
| Lymphocytes | 47% | 30-50 | |
| Monocytes | 0% | 0-3 | |
| Basophils | 1% | 0-3 | |
| Eosinophils | 1% | 0-3 | |
| AST/SGOT | 428iu | 19-159 | High |
| LDH | 1850iu | 277-977 | High |

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***Assessment***

Ascites, most likely due to an inflammatory hepatopathy. Bacterial, mycobacteria, viral, neoplastic congestive and toxic aetiologies were considered at this stage.

***Plan***

Ascitic fluid and faeces were sent away for culture and sensitivity. Assessment of possible mycobacterial infection was requested. No Mycobacteria or other enteric pathogens were isolated from the faeces or fluid samples. The fluid was characterized as a modified transudate with no evidence of infection.

Whilst awaiting results, enrofloxacin (Baytril®) was started at 15mg/kg bid, IM. However, the bird became extremely fractious and stressed by this and a change was made to doxycycline (Psittavet®), at 5mg/kg, IM, every 5 days.

***Outcome***

Throughout this period the birds' condition remained stable and it ate and drank well. In the following week there was no improvement in the ascites. A week after the antibiotics were commenced, the CBC was repeated.

| Parameter | Result | Normal value | Flagged |
|---|---|---|---|
| PCV | 30% | 25-45 | |
| TPP | 27g/l | 30-70 | low |
| WCC | $26x10^9$ | 8-20 | High |
| Heterophils | 36% | 40-70 | |
| Lymphocytes | 51% | 30-50 | |
| Monocytes | 2% | 0-3 | |
| Basophils | 0% | 0-3 | |
| Eosinophils | 11% | 0-3 | High |

***Assessment***

A faecal float was performed and was negative. Whilst the WCC had improved the PCV and protein levels had deteriorated and no improvement in the ascites was seen. A palpable mass could now be felt in the cranial abdomen. The problem still seemed to be active. It was decided to do a laparotomy in order to reach a definitive diagnosis and treatment, if possible.

***Laparotomy findings***

A diffuse peritonitis was seen. The liver was a pale tan colour and massively enlarged. The right lobe was almost spherical, the left lobe was a normal shape but a large mass of amorphous tissue was attached to the visceral side. The spleen was pale and enlarged and the reproductive and gastrointestinal tracts were grossly normal. Neoplasia or cirrhosis were suspected. The bird was euthanased.

***Histopathology results***

The liver architecture is markedly distorted by proliferating bile ductules accompanied by moderate to heavy infiltrates of plasma cells, heterophils and lymphocytes which extend into the liver parenchyma. The hepatocytes are pale and swollen and karyomegaly is seen. There are occasional small granulomas in the parenchyma

**Anatomic diagnosis**; severe bile ductule proliferation with chronic active portal hepatitis. Diffuse vacuolar change, karyomegaly and occasional granuloma formation.

The spleen shows severe acute and chronic splenitis with mild macrophage hyperplasia.

**Comment**: The lesions in the liver are consistent with a chronic hepatopathy and probable areas of secondary bacterial infection. Chronic aflatoxicosis or some other type of toxic insult to the liver would be the most likely cause of this disease.

The splenic changes are suggestive of chronic active infection.

# A Brief Review of Aflatoxins

## Definition

Aflatoxins are mycotoxins produced by *Aspergillus* spp. in or on crops or food. The name is derived from *A. flavis*, the first fungus identified as producing the toxin. *A. parasiticus*, *A. nominus* and *A. niger* are also known to produce aflatoxins. There are 4 major aflatoxins **B1, B2, G1, G2**, and two metabolic products**, M1** and **M2**, that are of significance as direct contaminants of food and feed. However, aflatoxin B1 is usually predominant and is the most toxic. Interestingly, *A. fumigatus*, the main cause of mycotic infections, does not seem to produce aflatoxins.

### Occurrence

The factors influencing fungal growth and toxin production are complex and not precisely understood. Aflatoxins often occur in crops in the field after prolonged drought and high temperatures. Post-harvest contamination can occur if crop drying is delayed or storage conditions are too moist and/or warm. Insect or rodent infestation can facilitate mold invasion.

The products with the highest risk of aflatoxin contamination are corn, peanuts, cottonseed and wheat. Other nuts, oilseeds, grains and dairy products have also been contaminated. Aflatoxins are very heat stable and can survive heating to 100°C. *Aspergillus* spp will produce toxin slowly at 10°C and most rapidly at 35°C. A humidity of 85% is ideal for fungal growth and toxin production. Significant toxin production can occur in 24 hours under optimal conditions.

### Aflatoxicosis

Aflatoxicosis is primarily a hepatic disease, manifesting as acute necrosis, cirrhosis and carcinoma. Histologically, extensive bile duct proliferation and periportal fibrosis are seen. Aflatoxin B1 is a very potent carcinogen in primates, birds, fish and rodents. Susceptibility varies with age, sex and species, but the young tend to be the most susceptible. Other clinical signs seen in birds include embryonic death, decreased egg production, shell deformities, immunosuppression, GIT disease, feather abnormalities, bone problems, bleeding and anemia.

### Control and Management of Aflatoxins

Aflatoxins are regarded as unavoidable contaminants of food and feed despite good manufacturing practice. The accepted level for human food is 20 parts per billion, except for milk which is 0.5ppb. Various methods of testing are used including several types of chromatography and immunochemical methods (TLC, LC, RIA, ELISA and ICA).

Detoxification is also used as testing provides no guarantees and contamination is common. Chemical treatments via ammoniation or use of sodium bisulfite appear to be the most useful methods of degrading aflatoxins in feed.

An alternative method is to add inorganic sorbent materials called *Chemisorbents* to the diet of animals, which bind and inactivate aflatoxin in gastronintestinal tract.

### Prevention of Aflatoxicosis

Purchase good quality, clean, dry seed and nuts. Imported products, especially from third world countries, should be viewed with suspicion as they are subject to little or no regulation. Use supplies of feed rapidly and store in cool, dry, pest proof containers.

### References

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