Cloacal Adenocarcinoma and Mycobacteriosis In An Imported Amazon: A Case Report.

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Mycobacteriosis is regarded world wide as a common, yet insidious disease of birds in pet, aviary and zoological collections as well as in the wild (1). The organism is normally found in the soil in low numbers and is shed in the faeces of infected birds. It is a very hardy organism and can exist in soil for years. Infection is by ingestion of infected material. There are more than 50 species of mycobacteria but only a few have so far been found to be significant pathogens in avians. M avium and M genevense are responsible for more than 90% of avian mycobacterial infections. Of these, M genevense has been found to account for up to 80% of avian infections(2).

Mycobacteriosis is highly significant for several reasons:

1. It can cause significant mortality within collections if unchecked
2. It is difficult to diagnose in both diseased and carrier animals
3. It is difficult to eliminate from the environment, especially where birds have access to soil and water
4. Treatment is extremely lengthy, complex and costly
5. It is a significant zoonosis

Clinical Manifestations of Mycobacteriosis

Most mycobacterial infections in pet birds are Per Os, consequently signs of GIT and liver disease are the most common. Following liver and GIT infection, hematogenous spread to bone marrow, lungs, airsacs, kidney and other organs may occur. Less frequently, subcutaneous nodular or diffuse lesions are seen (3). A series of 8 cases of mycobacterial conjunctivitis in cockatiels was recently reported and all but one bird had systemic disease as well (3).
Therefore, due to the often diffuse nature of the infection, clinical signs are many and variable. The most common are listed in approximate order of frequency(3).

- Weight loss is the most common but may not occur until disease is disseminated
- Diarrhoea with cowpat faeces and mucus is common. However, passage of whole seeds and biliverdinuria despite significant liver disease, is uncommon.
- Abdominal distension due to hepatomegaly is common
- Dyspnoea due to organomegaly or respiratory tract infection is seen.
- Shifting lameness due to osteomyelitis can occur
- Subcutaneous or conjunctival lesions that appear pale and soft are typical
- Non specific feather changes such as worn feathers, discoloration and failure to moult are common
- Neurological signs are occasionally seen

Diagnostic Tests

Despite many advances diagnosis, particularly antemortem, still remains the great challenge in mycobacterial infections.

A. Non Specific

Haematology is helpful but often vague and non-specific. Polychromasia, often with amenia is common. Leucocytosis with a heterophilia and monocytosis is common. However, some birds may have no changes(3).

Biochemistry can indicate organ involvement, particularly liver and kidney disease

Radiography can show organomegaly or more specifically granulomas in long bones, lungs or abdomen

B. Specific tests for Mycobacteria

Direct biopsy or endoscopic biopsy can be very useful antemortem if granulomas can be visualised and sampled

Faecal staining with acid-fast stains is simple, quick and cheap. However, it has been estimated that only about 10% of infected birds will have a positive test (4). This is attributed to intermittent shedding and that a significant threshold number of the organisms are required to enable visualisation with the stain.

Faecal culture is regarded as more sensitive but MA can take up to 6 months to grow and so far MG has not been successfully cultured (5)

PCR has become available to test for Mycobacteria in faeces, fresh tissue and paraffin block preparations(1,6). Certainly PCR increases the sensitivity of faecal testing but intermittent shedding is still a significant problem. Availability of samples, especially antemortem is a major limitation for tissue testing. In addition, availability of primers for specific mycobacteria can also limit testing.

Serology has been looked at in various studies. Detection of mycobacterial antibodies using an ELISA and a complement fixation test have been evaluated. They both were able to detect antibodies in response to infection with various serovars of MA, however it was also demonstrated that some infected birds did not produce detectable levels of antibody(1,7). A third serologic assay detecting a mycobacterial antigen called Antigen 85 has been used successfully in hoof stock and primates (1). It was evaluated for use in birds with only moderate success but still seems to have potential (1). At this stage, none of these tests are commercially available.

Zoonotic Potential

Whilst significant, there appears to be marked differences in the zoonotic potential of the different mycobacterial species. M tuberculosis is the most significant human pathogen, followed by M bovis, *in non immuno-compromised patients.* M tuberculosis has only rarely been reported in birds, mainly finches and a few amazons (2).

M avium and M genevense are responsible for more than 90% of avian mycobacterial infections. Of these, M genevense has been found to account for up to 80% of avian infections (2). Both of these mycobacteria are rarely implicated in infections in people with normal immune systems. However, M avium has been shown to occur in 30-80% of AIDS patients and M genevense is now being detected as well (2).

Treatment

Of most concern is that *M avium* is refractory to treatment with many drugs used in the treatment of Mycobacteriosis. Therefore, even if treatment is not attempted, all people in contact with the bird or involved in cleaning of infected areas need to be made aware of the risk and take proper precautions. Treatment of pet birds has recently been shown to be successful (5) and in my opinion this is a mixed blessing. Treatment requires multiple drug therapy for 1 year and significant follow up testing. Issues of informed consent, lack of compliance and other potentially disastrous legal problems need to be clearly addressed by all parties involved.

Current status of Avian Mycobacterial Disease in Australia
Incidence of disease obtained from several pathology registries around Australia showed that of total bird cases seen, mycobacterial infections were seen in ?% of cases. In histopathology cases mycobacteria were seen in ?% of cases.

Of these ? % were M avium, ?% were M genevense and ?% other.

Available tests from commercial labs in Australia for Mycobacterial infections in birds are

Acid fast staining of faeces and tissue samples
Faecal Culture
PCR of Faeces
PCR typing of mycobacterial species based on isolation of bacteria from culture or tissue samples

Case report

A 3yo male, captive bred but imported Amazon parrot was presented with weight loss, and frank blood in droppings and variable appetite. The bird had been on the premises for 18 months and had been sharing a flight with a hen of the same species for the last 12 months. The flights were suspended and newly built but shared a wall with adjacent flights.

Initial work up showed an unwell looking bird, underweight, frank blood in faeces. Owner has also noted some red contour feathers developing on chest/neck.
Haematology; PCV 36%, WCC 35 (10-13), Het 90% Lymp 10%
Biochem all normal including bile acids, except Amylase 600 (300-450 iu)
Focal Gram normal and focal floatation normal
Urinalysis shows USG 1.005, dipstick NSF but urine appears viscous.
No significant findings on cloacal or oral exam.
Radiographs do not indicate gross evidence of metal particles.
Started baytril and calsenate pending Zn levels

1 week later**, some** improvement, slight weight gain, still has intermittent frank blood in **faeces**.
Zn levels normal
Repeat haematology WCC still 30, marked heterophilia.
Chlamydia antibody (Immunocomb) suspect positive (1-2 on Combscale).
Increase dose of baytril and reassess in a week.

Bird seemed to improve in demeanour and appetite over next 2 weeks and blood disappeared from droppings but failed to gain weight. A PBFD test was done and was negative.

Then 4 weeks after initial presentation, bird started to deteriorate again, blood in droppings, sick bird look, loss of appetite and appeared to be close to death. The WCC was still about 30 with a marked heterophilia. PCV was 30 and had dropped from 36. Biochemistry was still normal. Cultures for Salmonella were performed and were negative.

Started injectable doxycycline (Psittavet) weekly, which nearly killed the bird in the 24 hours after the first dose. However, over the next 7 weeks the bird gradually improved. The bleeding became much less frequent and seemed to improve if the client administered 150mg oral calcium borogluconate daily.

After 7 weeks of treatment the WCC was 12, Het 75, Lymp 22, mono 3, however biochemical abnormalities were appearing, AST, LDH and uric acid were all becoming significantly elevated. This was partly attributed to chronic Psittavet administration but CK was normal despite repeated injections. The bird was significantly improved but still markedly underweight and far from being called well or cured.

1 week later (after 12 weeks of illness/treatment) the bird significantly deteriorated, with severe haemorrhage from cloaca and died after 2 days.

On gross post-mortem a 0.5cm solid mass was found in the lateral wall of the cloaca, the kidneys were friable and swollen on the same side and other organs appeared normal. Histopathology done by Dr Shane Raidal showed an adenocarcinoma of the cloaca and colon and multifocal granulomatous enteritis with ZN positive bacteria.

In conclusion, ultimately this bird died of cancer. In retrospect many of the clinical signs suggest that it also suffered from clinical Mycobacteriosis, hence the response to long term Doxycycline. It developed many of the typical symptoms of Mycobacteriosis: weight loss, leucocytosis, mucoid faeces, feather color changes and general chronic debility. The diagnosis becomes even more relevant considering the value of the rest of the collection and the fact that birds are both bought and sold on a regular basis.

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