Mycobacteriosis in Gouldian Finches

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Mycobacteriosis is becoming a well recognised syndrome in Gouldian finches (*Chloebia gouldiae*) and other small passerines. Most reports in the literature describe individual cases, this presentation will describe an outbreak in an aviary of Gouldian finches.

The aviary was started in 1988 with three pairs of Gouldian finches and two pairs of Blue-faced parrot finches (*Erythrura trichroa).* In 1989 two male gouldians and a pair of St Helena Waxbills (*Estrilda astrild*) were added.

In 1991 the senior author was asked to investigate the high nestling mortality rate in the collection. The hatch to weaning percentage had declined from 48% in 1989 to 11% in 1991. Despite extensive investigation including post mortems aerobic bacteriological culture and histopathology no specific cause could be found for this problem. Management appeared to be good.

In 1992 the adults started developing clinical illness. The first adult presented for post mortem had exceptional hepatomegaly and tissues were submitted to a private laboratory (Anapath) for examination. The histopathological examination revealed mycobacteriosis. Because of public health concerns further samples were submitted to the NSW Department of Agriculture.

Materials and Methods

A total of 17 birds or tissues were submitted to G Redacliff at Elizabeth Macarthur Agricultural Institute, NSW Dept of Agriculture. Ten birds were submitted whole, chilled for gross and follow-up examination. Selected tissues were fixed in 10% neutral buffered formalin. 5µ thick paraffin-embedded sections were prepared by routine methods, stained with haematoxylin and eosin (H & E) and examined by routine bright light microscopy. Selected sections were stained also with Ziehl Neelsen (ZN) stain. Fixed organs only were received from a further 7 birds and were processed as above.

Culture of the organism was attempted on three occasions. No growth was obtained after 3 months' incubation at 37°C or 41°C on a variety of specialist mycobacterial media.

Results

5/10 and 5/7 of the birds submitted whole and fixed respectively were detectably infected with acid fast bacilli (AFB).

The 5 birds examined grossly all had enlarged pale mottled livers extending to the pelvic brim in 3 birds. Spleens were also diffusely enlarged pale and mottled. There were no other consistent gross lesions. One bird had a 1 cm diameter haemorrhagic lump on the kidney and another had a caseous lump at the commissure of the beak. Most birds were in reasonable body condition.

Histological changes were similar in all birds, varying only in degree. Mild presumably early lesions were seen in the livers of 2 birds which died of other causes. One bird died with severe acute interstitial pneumonia of unknown cause. No other significant lesions were seen in the H&E sections of this bird although scattered

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cells with intracytoplasmic AFB were detected in the portal tracts of the liver in ZN sections.

The other bird had a haemorrhagic lymphoid tumour displacing the kidney. In H&E sections there was moderate mainly centriacinar infiltration of the liver by macrophages with abundant pale eosinophilic finely granular cytoplasm. There were also occasional foci of acute necrosis and associated infiltration with small mononuclear cells. ZN stains confirmed the presence of AFB in the macrophages in the liver.

More severely affected birds had confluent sheets of macrophages displacing much of the liver parenchyma. Often only small islands of normal hepatocytes were seen in the centiacinar areas. ZN stains revealed massive infection by AFB in the cytoplasm of these macrophages. The infection was so severe that a dense red mottling was easily seen when the sections were examined with the naked eye. Spleens were consistently involved also. Confluent sheets of macrophages replaced much of the red pulp and the white pulp adjacent to the splenic arterioles. The ellipsoids retained their lymphoid collars although the reticular cells appeared vacuolated. ZN stains showed dense AFB in the macrophages as above and occasional AFB in the cytoplasm of the reticular cells of the ellipsoids. AFB laden macrophages were present in many other tissues. The lamina propria of the duodenum was heavily infiltrated in several birds and clumps of macrophages were readily seen in the lungs. Isolated AFB laden macrophages, small clumps or confluent sheets of macrophages were seen in the heart, gizzard wall, ovary, adrenal, kidney, and brain of some birds. One bird had a single lamellated caseous lesion in the liver. In one very autolysed bird H&E sections were useless for diagnosis but masses of AFB were readily observed in ZN sections.

Discussion

Mycobacteriosis is a chronic infectious disease of humans, other mammals, birds, reptiles and fish caused by *Mycobacterium sp*. Members of the genus *Mycobacterium* are strictly aerobic, non spore- forming curved or straight gram positive rods. They are acid-fast in that they resist the decolourisation effect of acid-alcohol after they are stained with hot carbol fuchsin.

There are three main types of Mycobacteria that have zoonotic potential; *M tuberculosis*, *M bovis* and *M avium* complex. *M avium* is often grouped with other closely related organisms (*M avium*; *M intracellulare*; *M scrofulaceum*) to give the MAIS Complex. The MAIS is classified into a number of serotypes 1-21. Serotypes 1, 2 and 3 are the normal pathogens in birds.

The high lipid content of the cell wall allows the organism to live in soil or faeces for long periods of time. It also confers resistance to acids, disinfectants, antibodies and desiccation.

Griner (1978) first reported atypical mycobacteriosis in six species of passerines. This was at San Diego Zoo and none of these were in Gouldian Finches.

Montali and Nichols (1987) first reported atypical mycobacteriosis in Gouldian finches. They speculated that atypical mycobacteriosis has adapted to Gouldian finches producing high morbidity and low mortality. Mortalities may be precipitated by stressful conditions such as overcrowding or transport. This corresponds to the findings in this series where the increase in mortalities corresponded to an increase in population density.

Riddell (1987) describes avian tuberculosis in psittacine birds and some other birds as a massive infiltration of epithelioid cells in the liver without necrosis or encapsulation.

Rae and Rosskopf (1992) reported six separate cases of mycobacteriosis in five private collections of Gouldian finches. All had mild to severe histiocytic hepatitis, four had splenitis. There were three with pneumonia and some had enteritis, myocarditis, proventriculitis, ventriculitis, thyroiditis, ingluveitis and tracheitis associated with AFB. There were no caseous lesions.

Reece (1992) described similar diffuse granulomatous lung lesions in a gouldian finch. These descriptions concur with the findings in this series of finches and it is important to recognise that this is the usual pathology of tuberculosis in passeriformes. In the galliformes and anseriformes tuberculosis takes the more usual form with caseating granulomas and giant cells. The finch described by Reece 1992 had only lung lesions,

suggesting inhalation as the route of infection. In the present outbreak the earliest and the most severe pathology was in the liver, with severe changes also in the spleen and duodenum, suggesting an oral route of infection.

It is important to recognise this form of mycobacteriosis in passeriformes and to be aware that if only H&E sections are examined then early lesions might be missed. Mycobacteriosis should be considered in any chronically sick bird.

The high morbidity and mortality in this collection suggests a special susceptibility. This may be a result of inbreeding in this particular population or of a special susceptibility of the species as a whole. This has special importance in view of the concerns held for the species in the wild.

More research into these findings is needed to determine;

1. whether as Montali and Nichols speculate, this is a new type of bacteria.
2. whether it is a new host response.
3. or perhaps whether there is some immune deficiency in gouldian finches which predispose towards infection with an ubiquitous organism.

The inability to culture the particular mycobacteria in this series makes these questions even more intriguing.

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References

Griner LA (1978). Atypical tissue reaction to *Mycobacterium avium* in passeriformes and coraciiformes. In Montali RJ (Ed): Mycobacterial Infections in Zoo Animals. Washington DC Smithsonian Institute Press. pp 205-207.

Montali RJ, Nichols D and Jarnagin JL (1987). Mycobacteriosis in Gouldian Finches. *AAV Today*. **1**: 54.

Rae MA and Rosskopf WJ Jr (1992). Mycobacteriosis in Passerines. In *Proceedings of the Association of Avian Veterinarians*. New Orleans, Louisiana. pp 234-242.

Reece RL *et al* (1992). Some unusual diseases in the birds of Australia. *Veterinary Record*. **130**: 178-185.

Riddell C (1987). Liver. In Avian Histopathology. American Association of Avian Pathologists, University of Pennsylvania, Kennet Square, Pennsylvania. p 62.