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Avian mycobacteriosis in captive brolgas (*Antigone rubicunda*)

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Case report Avian mycobacteriosis is a significant disease of a wide range of bird species worldwide. The most common causative agent, *Mycobacterium avium*, is reported to also infect a range of mammals, including humans. Of 11 brolgas (*Antigone rubicunda*) submitted to the University of Melbourne for postmortem examination over a 10-year period, 7 were diagnosed with mycobacteriosis. All were from a wildlife park and kept in permanent enclosures as part of a breeding program. Most of the brolgas with mycobacteriosis were in poor body condition and had widely disseminated granulomas throughout the body, especially within the liver, spleen and gastrointestinal tract. Respiratory tract involvement was common, with all disseminated cases having pulmonary or air sac granulomas. Rare to moderate numbers of acid-fast organisms were detected in granulomas by histological examination. Where examined by appropriate bacteriological examinations, *M. avium* complex was isolated from affected tissues.

Conclusion This case series is the first known report of mycobacteriosis in brolgas and highlights the pathological changes seen. The complications in maintaining an avian mycobacteriosis-free breeding program and in eradication of the disease from an enclosed wildlife environment are discussed.

Keywords: avian tuberculosis brolgas; mycobacteriosis; *Mycobacterium avium*

Abbreviation MAC, *Mycobacterium avium* complex

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Avian mycobacteriosis, also known as avian tuberculosis, is a well-documented granulomatous disease seen worldwide in a wide variety of bird species,^{1–5} both wild and captive, including zoo collections, wildlife parks, commercial operations and private collections. The mycobacteria most frequently responsible for avian mycobacteriosis are collectively called the *Mycobacterium avium* complex (MAC) and include *M. avium* subsp. *avium*, *M. avium* subsp. *paratuberculosis*, *M. avium* subsp. *silvaticum*, and *M. intracellulare*.⁶ *Mycobacterium avium* is an acid-fast slender bacillus that can survive for up to 4 years in soil.⁷ The longevity of the organism in the environment, together with difficulties in antemortem diagnosis of affected birds, which can remain asymptomatic for prolonged periods, poses significant problems in captive collections, where eradication of the disease is difficult. Sources of human infection are typically household tap water, soils and bathrooms,⁸ rather than livestock and wildlife; therefore, zoonotic transmission is unlikely. However, bacteria within the MAC can infect immunocompromised human patients.⁹

Brolgas (*Antigone rubicunda*) are a species of crane native to Australia and New Guinea. They are a long-lived, large, wetland species that has declined in number, particularly in Victoria, since European settlement.¹⁰ Brolgas feed on a wide range of foods, including wetland and dryland vegetation,¹¹ invertebrate and small vertebrate species such as insects, molluscs, reptiles and birds.¹⁰ Their typical life span in captivity is approximately 30 years.¹² Brolgas in south-east Australia are thought to be geographically isolated from northern flocks, although this may not always have been the case, as a preliminary genetic study indicates there is minimal variation between northern and southern brolgas.¹³ Brolgas in Victoria are currently listed as threatened under the *Flora and Fauna Guarantee Act 1988*.

Avian mycobacteriosis has been reported in at least two other species of crane in North America: the whooping crane (*Grus americana*)¹⁴ and sandhill crane (*Antigone canadensis*).¹⁵ Both of these were individual cases and within free-living birds. The whooping crane had multifocal granulomas extensively throughout the intestines, liver, spleen, kidneys and on the serosal aspect of the trachea. Granulomas in the sandhill crane were limited to the liver.

The purpose of this case series was to describe the pathology seen in seven brolgas with avian mycobacteriosis. The implications of mycobacteriosis to the wildlife park and its breeding program are also discussed. To the authors' knowledge, this case series is the first known report of mycobacteriosis in brolgas.

Case series

Submission history

Over a period of 10 years, 11 brolgas were submitted from a bird and wildlife park in Victoria, Australia, to the Faculty of Veterinary and Agricultural Sciences, the University of Melbourne, for postmortem examination. The birds died or were euthanased because of severe illness. Of the 11 brolgas submitted for postmortem,

7 had findings consistent with mycobacteriosis and these are discussed in this report. All of the brolgas were part of a captive-breeding program at the wildlife park. The brolgas were kept in pairs in large, combination dry/wet, permanent uncovered enclosures. Flying birds and rodents were able to enter the brolga enclosures. Following both the first and final cases of mycobacteriosis at the wildlife park, control measures were recommended to the wildlife park, including soil treatment within the contaminated enclosure with lime, ongoing monitoring of deaths and leaving the enclosure vacant for at least 1 year. In March 2014, just after the last submission, there were 12 brolgas within the breeding program. There have been no further submissions to the University of Melbourne since that time.

Pathological features

Each bird underwent routine postmortem examination. Five of the seven birds had poor body condition with concave pectoral muscles (pectoral muscle score approximately 2/5; Table 1). Consistent findings in the seven birds were variably sized, slightly raised, cream-coloured nodules, particularly within the liver, spleen and alimentary system. The nodules exhibited necrotic centres surrounded by a thin, fibrous capsule and ranged in size up to approximately 35 mm in diameter.

The subcutis, kidney, oviduct and bone marrow were variably involved, with multiple nodules (Table 1). Hepatic nodules were present in all seven birds (Figure 1) and there were multifocal splenic and alimentary nodules in six of them.

Six of the seven birds had respiratory tract involvement. Of these, birds 1 and 6 had scattered nodules of less than 5 mm diameter within the wall of the abdominal air sacs. Three birds (nos 2, 3, 5) had multiple, variably sized pulmonary nodules. Bird 6 had one visible nodule in the lungs; bird 1 and bird 7 had no visible nodules in the lungs. Three birds (nos 1, 2, 6) had cloacal nodules and prolapse of the cloaca (Figure 2).

Bird 4, a juvenile bird with only a single nodule, had a large blood clot within the peritoneal cavity, ascites and pulmonary oedema.

Histopathological features

Tissues were fixed in 10% neutral-buffered formalin, embedded in paraffin, sectioned and stained with haematoxylin and eosin. Selected tissues were stained with Ziehl-Neelsen and periodic acid-Schiff.

Histologically, the cream-coloured nodules were granulomas and similar in all organs. The granulomas consisted of central necrotic material surrounded by epithelioid macrophages and multinucleate giant cells enclosed by a cuff of lymphocytes, plasma cells and low numbers of heterophils (Figures 3, 4). Smaller nodules consisted of epithelioid macrophages with no necrotic core. The granulomas were often encapsulated by fibrous tissue and frequently coalesced and effaced the hepatic and splenic parenchyma. In other tissues, the granulomas were individualised. Gastrointestinal lesions were distributed within the lamina propria, submucosa and/or muscular layers.

In each case, liver tissue samples and a variable selection of other tissues containing granulomas were stained with Zeihl-Neelsen stain. Within the epithelioid macrophages and necrotic centres of the granulomas, rare to low numbers of acid-fast, slender bacilli suggestive of *Mycobacterium* spp. were seen (Figure 5). In birds 1, 524/06, 4 and 5, rare acid-fast bacilli (as few as 10 organisms within an entire section of tissue) were detected. Low to moderate numbers of acid-fast bacilli were found in birds 3, 6 and 7.

Bird 3 had mycotic pneumonia in addition to acid-fast organisms within granulomas. Lung sections showed multiple small to large infarcts with a heavy growth of fungal hyphae radiating out from the vascular walls. The hyphae stained intensely pink with periodic acid-Schiff and showed branching septate filaments. The fungal infection was localised to the lungs, whereas the infection with acid-fast organisms was widely disseminated throughout the body.

Bacteriology

Liver tissue was cultured from all birds except bird 4. Additionally, intestine from bird 1, bone marrow, intestine and proventriculus from bird 2, bone marrow and intestine from bird 3 and lung and proventriculus from bird 5 were collected for mycobacterial culture. For bird 4, swabs of liver and pericardium were taken for routine culture and resulted in no colonies on conventional media.

Collected tissues were homogenised and decontaminated at 1 : 10 w/v in 3% v/v NaOH for 30 min, then neutralised with HCl. The homogenised tissues were centrifuged and the pellet used to inoculate Lowenstein-Jensen agar slopes containing pyruvate and glycerol (Oxoid, Basingstoke, UK). The cultures were incubated at 35°C for up to 8 weeks, until typical mycobacterial colonies were visible to the naked eye. An aliquot of cultures from birds 1, 2, 3, 5 and 6 were submitted to the Victorian Infectious Diseases Reference Laboratory (North Melbourne, VIC, Aust) for further identification using interspersed repetitive-unit-variable-number tandem-repeat typing.¹⁶

Bacterial colonies grown on the surface of the slope from tissues from birds 1, 2, 3, 5 and 6 were confirmed as *Mycobacterium* spp. with acid-fast staining. The laboratory further identified these samples as MAC. Culture of frozen liver from bird 7 was performed approximately 2 years after collection and isolation was not successful.

Discussion

The submission of broilgas from a wildlife park that had died or been euthanased over a period of 10 years enabled monitoring of the mycobacterial prevalence within this bird population. In addition, avian mycobacteriosis had been diagnosed in an Australian bustard (*Ardeotis australis*) from the same park and in several pigeons/doves from a property in an approximately 30-km radius of the park (unpubl. data). Access to the broilga enclosures by flying birds and rodents may have allowed further dissemination of the organism to other bird and mammal species, with subsequent dispersal of mycobacteria within the greater environment. Unfortunately,

identification of the samples was simply MAC: subtyping was not undertaken. Preliminary analysis of two of the isolates (1161-11 and 176-14) using *M. avium* repetitive element IS1245¹⁷ did not detect any major difference between these isolates, suggesting that they shared the same origin (unpubl. data). However, a detailed molecular study using techniques such as whole-gene sequencing to investigate the link between the isolates reported here and also those that may be harboured by other species of birds or animals in the same region will be necessary to unravel the epidemiology of *M. avium* infection in the broilgas.

The presence of granulomas containing acid-fast organisms in birds is considered sufficient for a diagnosis of avian mycobacteriosis⁷ and therefore a diagnosis of mycobacteriosis was given in all of the present birds, although it was not isolated by bacteriology in two cases (birds 4 and 7). Antemortem intradermal injection of avian tuberculin and PCR and Ziehl-Neelsen staining of the faecal specimens from a number of the affected broilgas were attempted, but all these tests were found to be incapable of detecting the organism/infection (data not shown). That result was consistent with the very low number of organisms often found during histopathological examination of the lesions. In six of the seven broilgas, mycobacteria were thought to be the cause of death or reason for euthanasia. The death of bird 4 was thought to be from pulmonary oedema and possibly secondary to the space-occupying blood clot, with the granuloma being an incidental lesion. Given time, it is likely that this juvenile bird would have developed more widespread granulomas. Cloacal prolapse and poor body condition were the only externally visible abnormalities noted in the broilgas and were found in three and five of the birds, respectively. These findings are likely to have been only visible terminally, so are unlikely to represent useful antemortem clinical signs of possible mycobacteriosis. Reports in other bird species often indicate moderate to abundant mycobacteria within granulomas.^{3,14,18,19} Although the granulomas in the broilgas were often widespread, mycobacteria seen on Ziehl-Neelsen sections were rare in four cases; care should be taken to ensure these are not overlooked in sections from broilgas. The immunological basis for the low number of mycobacteria in granulomas can only be speculated, but may relate to the pathogenicity of the MAC strain/s involved or the broilgas' ability to suppress bacterial numbers and mount a strong granulomatous response. The latter is seen in sheep with *M. avium* subsp. *paratuberculosis* (Johne's disease) paucibacillary form with a combined Th1 and Th2 immune response; this is unlike the multibacillary form in sheep, where a Th2 response occurs.²⁰ It is possible a similar variation exists in the type of immune response mounted by broilgas, compared with that of many other birds.

Avian mycobacteriosis is generally considered to be an alimentary disease with dissemination to other organs in the body,¹⁸ unlike the pulmonary disease seen in humans.²¹ Although the predominant feature of the disease in these birds was alimentary, all birds in this series with disseminated acid-fast organisms had respiratory granulomas and all but two birds had pulmonary granulomas. The only

bird without respiratory involvement was bird 4, a juvenile bird that died of other causes.

The findings in this series indicate that broilgas with mycobacteriosis can be expected to have disseminated respiratory granulomas terminally. As none of these birds showed signs of respiratory illness, this finding may not be useful as an antemortem change to detect underlying mycobacteriosis. Avian mycobacteriosis has been considered primarily an alimentary disease, although in severe/advanced cases, the respiratory system may be affected.⁷ Consistent with this, the respiratory granulomas in these broilgas appeared less advanced than the alimentary and splenic granulomas, suggesting transmission occurred via the alimentary route. The presence of respiratory granulomas in other bird species reported varies from not mentioned^{2,3,14} to disseminated throughout the lungs,²²⁻²⁴ suggesting that the distribution varies in different bird species. Interestingly, pulmonary lesions were also seen in most birds from three series: captive white-winged ducks,⁵ captive mixed water birds¹ and another large study of a range of zoo birds.²⁵ It is possible that pulmonary lesions have previously been under-reported based on the paradigm of the alimentary nature of the disease. It may be important to conduct experimental studies in the future to determine if the low incidence of respiratory lesions is a predilection of the organism for the alimentary system or that the lesions are simply overlooked because of their obscured nature in the respiratory system.

Given the molecular differences between human and bird *M. avium* isolates, it is thought that most human MAC infections are spread by other humans or from the environment rather than from birds.⁷ Therefore, although the aerosol route of infection is important in both immunocompromised and immunocompetent people,⁹ it is unlikely that the presence of pulmonary granulomas in the broilgas will lead to zoonotic transmission. However, given the importance of environmental sources of mycobacteria in people,⁸ marked increases in environmental contamination of soil and water by the organism at the wildlife park could increase the slight risk of zoonotic infection via indirect transmission.

Management of mycobacteriosis is a difficult undertaking, given the ability of mycobacteria to persist in the environment and the difficulties in antemortem diagnosis to identify asymptomatic birds. Painter discussed the measures taken to prevent avian mycobacteriosis within a wildfowl collection in South Wales, which were ineffective, revealing how difficult it is to successfully establish and maintain a mycobacteriosis-free population of birds.⁴ The most successful approach to managing poultry flocks affected by the disease is depopulation and repopulation, along with replacement of soil and litter.^{18,26} This is less viable with non-domestic species because of their high individual value and possibly difficulties in decontaminating their environment, when compared with poultry in sheds. Some researchers have also suggested this mode of mycobacterial control is outdated and not appropriate for collections of wildlife, as most birds in their study exposed to mycobacteria did not become infected.²⁷ However, the latter statement is not true in all situations, with the same study indicating this approach best applies to

populations where the incidence of disease is low.²⁷ The same research group found that living in a small enclosure and extended exposure to infected birds significantly increases the risk of infection²⁵, indicating that although exposure to infected birds does not equal infection, it can still be important. Factors influencing the transmission of avian mycobacteriosis are poorly understood, but may relate to exposure to contaminated environments and infected birds, age at exposure, length of duration of exposure to infected birds, stresses such as small enclosures, movement and handling of birds, pinioning and poor environmental conditions^{25–28}, and genetic susceptibility.⁵ Birds affected by mycobacteriosis have significant lesions and bacterial load before exhibiting clinical signs, leading to extensive environmental contamination from shedding¹⁸ prior to diagnosis or death. Although this environmental contamination with *Mycobacteria* is likely partially responsible for the ongoing infections of brolgas at this site, other bird populations exposed to avian mycobacteriosis do not necessarily develop disease.²⁷ Therefore, the number of mycobacterial infections in this group of brolgas may indicate increased susceptibility to mycobacteriosis, possibly because of genetic susceptibility, extended duration of exposure to infected birds or other unknown factors. It has been suggested previously that captive white-winged ducks' (*Cairina scutulata*) limited genetic diversity contributes to their high rate of infection.⁵ This is also possibly true of these captive brolgas, given their presence in a breeding program with unknown genetic diversity. The general susceptibility of wild brolgas to avian mycobacteriosis is unknown, so future studies to investigate brolgas' susceptibility to mycobacteriosis are required. Furthermore, it would be interesting to know whether the minor genetic differences between southern and northern brolga populations engender a different susceptibility to avian mycobacteriosis. Given that decontamination of the environment is impractical, and repopulation of a breeding program requires the sourcing of uninfected stock, continuation of this breeding program may be futile. Moreover, it is essential that the introduction of birds to the wild with potential mycobacterial infections does not occur.

Conclusion

Avian mycobacteriosis is an important disease that has been reported to affect a large variety of bird species. The disease in brolgas appears to be predominantly alimentary, similar to previous reports in other bird species, but also with an important respiratory component. Avian mycobacteriosis is a particular problem in captive bird populations and can hamper attempts for captive-breeding programs and maintenance of threatened species such as the brolga.

The continued presence of avian mycobacteriosis in brolgas at this wildlife park over 10 years indicates that major changes in management of the disease need to occur or the breeding program be abandoned. Continuation of the breeding program as it has been over the course of these submissions endangers the health of the other species of birds at the park and nearby bird and wildlife populations.

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Figure 1. Photograph of bird 3, showing the typical gross appearance of the broilgas at display stage with enlargement of the liver because of miliary distribution of nodules throughout.

Figure 2. Photograph of bird 2 showing cloacal granulomas and prolapse of the cloaca.

Figure 3. Histological specimen of spleen from bird 6 showing coalescing granulomas effacing the splenic parenchyma (H& E).

Figure 4. Histology section from bird 6 showing a hepatic granuloma with a central necrotic core (H&E).

Figure 5. Hepatic granuloma in bird 6, showing scattered pink filamentous bacteria within macrophages. The arrows point to a cluster and an individual organism (Ziehl-Neelsen).

Table 1. Tissue distribution of necrotic nodules and other gross pathological changes within seven broilgas affected by avian mycobacteriosis

Case accession no.	Bird 1 (547/05)	Bird 2 (425/06)	Bird 3 (1073/06)	Bird 4 (353/08)	Bird 5 (1161/11)	Bird 6 (176/14)	Bird 7 (218/14)
Signalment	4-year-old male	5-year-old female	Juvenile, female	Juvenile	1-year-old male	Adult female	Adult
Liver	+	+	+	+	+	+	+
Spleen	+	+	+		+	+	+
Lung		+	+		+	+	+
Mouth	+		+				
Air sacs	+					+	
Caecal tonsils	+	+					+
Proventriculus	+	+			+		+
Cloaca	+	+				+	
Bone marrow		+	+				
Heart						+	
Subcutis			+			+	
Oviduct			+				
Small intestine	+	+	+		+	+	+
Kidneys						+	+
Other findings	Poor body condition; prolapse of the cloaca	Lice; atherosclerosis (aorta); prolapse of the cloaca	Poor body condition; mycotic pneumonia	Poor body condition; pulmonary oedema; peritoneal fluid; reorganising haematoma in peritone	Decomposing	Poor body condition; pericardial effusion; serous atrophy of fat; protozoa in endothelium of brain,	Poor body condition; lice

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