JOURNAL OF CLINICAL MICROBIOLOGY, Apr. 1998, p. 1101–1102 Vol. 36, No. 4
0095-1137/98/$04.00+0

NOTES

Mycobacterium tuberculosis Infection in a Green-Winged Macaw (*Ara chloroptera*): Report with Public Health Implications

RITA M. WASHKO,[1,2] HEIDI HOEFER,[3] TIMOTHY E. KIEHN,[4]* DONALD ARMSTRONG,[5] GUY DORSINVILLE,[2] AND THOMAS R. FRIEDEN[2,6]

Epidemic Intelligence Service, Division of Field Epidemiology,[1] and Division of Tuberculosis Elimination, National Center for Prevention Services,[6] Centers for Disease Control and Prevention, Atlanta, Georgia; Bureau of Tuberculosis Control, New York City Department of Health, New York, New York[2]; Animal Medical Center, New York, New York[3]; and Microbiology Service, Department of Clinical Laboratories,[4] and Infectious Disease Service, Department of Medicine,[5] Memorial Sloan-Kettering Cancer Center, New York, New York

Received 2 September 1997/Returned for modification 9 December 1997/Accepted 30 December 1997

***Mycobacterium tuberculosis* was isolated from the eyelid, skin, tongue, and lungs of a green-winged macaw (*Ara chloroptera*). Two persons living in the same household were culture positive for pulmonary tuberculosis 3 to 4 years before tuberculosis was diagnosed in the bird. Although humans have not been shown to acquire tuberculosis from birds, an infected bird may be a sentinel for human infection.**

Although *Mycobacterium tuberculosis* primarily causes disease in humans, many animals are reported to be susceptible to infection, including primates, swine, cattle, sheep, goats, dogs, cats, and elephants (3, 8). Infection with *M. tuberculosis* in birds is rare. Psittacine birds (principally the parrot family) are the only avian species known to become infected with *M. tuberculosis*, presumably due to close contact with tuberculous owners. There are four reports of infection in Amazon parrots: one each in a mealy parrot (1), a yellow-naped parrot (10), and a red-lored parrot (2) and one in an Amazon parrot which was not further identified (4). All of the parrots presented with cutaneous growths on the head and neck and had localized granulomas in the eye and retrobulbar tissues, the sinuses and nares, and the oral cavity. Two parrots were found to have systemic infections at necropsy (1, 4). The birds had no known contact with humans with tuberculosis.

This report describes disseminated *M. tuberculosis* disease in another psittacine bird, a green-winged macaw, and notes a possible human source of the infection. This report has implications for public health in that human-to-bird and, possibly, bird-to-human transmission of tuberculosis could create a perpetual reservoir of untreated tuberculosis.

Case report. An adult, imported green-winged macaw (*Ara chloroptera*) had been ill for several months with progressive eyelid swelling, intermittent diarrhea, occasional loud respiratory sounds, listlessness, and poor feeding. The bird had lived in a multiple-person household in New York City for at least 6 years. Several months prior to the presentation described here the bird had been evaluated for tachypnea by a veterinarian. At that time, specimens from facial lesions were positive for acid-fast bacilli on microscopic examination, but culturing was not performed. The owner was advised to euthanatize the bird but chose not to do so.

*Corresponding author. Mailing address: Microbiology Laboratory, Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY 10021. Phone: (212) 639-8181. Fax: (212) 772-9285. E-mail: kiehnt@mskcc.org.

The bird presented in April 1995 to the Animal Medical Center with multiple nodules on the eyelids and conjunctivae of both eyes as well as on the unfeathered areas of the face and head. The oral cavity contained numerous small white nodules on the tongue, choana, and glottis. The bird was moderately thin and had a cardiac murmur. Blood tests revealed a moderate leukocytosis, a mildly elevated aspartate aminotransferase level, and a marked elevation in the beta globulin level as determined by plasma protein electrophoresis. Radiographs revealed enlarged cardiac and hepatic silhouettes.

Microbiology. Acid-fast bacilli were seen in auramine acid-fast stains of biopsied tissue from the eyelid, tongue, and skin. Specimens were processed and inoculated into BACTEC 12B broth bottles (Becton Dickinson, Diagnostic Instrument Systems, Sparks, Md.) and onto Middlebrook 7H11 agar plates (Becton Dickinson Microbiology Systems, Cockeysville, Md.). In approximately 2 weeks, cultures were positive and the organism was identified as *M. tuberculosis* by conventional biochemical tests, including niacin production, nitrate reduction, and growth on thiophene-2-carboxylic acid hydrazide medium. A DNA probe for *M. tuberculosis* complex was positive (GenProbe, San Diego, Calif.). A standardized *IS*6110-based Southern blot hybridization protocol was used to determine the genotype of the isolate. Restriction fragment length polymorphism analysis revealed a three-banded pattern that is the most common pattern in New York City. The organism was susceptible when tested by the radiometric antimicrobial susceptibility test system (BACTEC) to ethambutol, isoniazid, pyrazinamide, rifampin, and streptomycin.

Epidemiology. Review of the New York City tuberculosis registry and hard copies of Department of Health records revealed that two people with a history of culture-confirmed pulmonary tuberculosis resided at the address listed for the bird's owner. They had extensive contact with the bird while they were potentially infectious, including placing food between their lips for the bird to grasp. The first person had drug-treatable tuberculosis diagnosed in 1991 and did not adhere to treatment but ultimately completed treatment while

incarcerated. The second person's disease was diagnosed in January 1992, but the isolate was not tested for antimicrobial susceptibility. The patient completed a full course of antituberculosis treatment.

After the bird's diagnosis, the two household members previously treated for tuberculosis underwent repeat evaluation, including chest radiographs and sputum cultures. All results were negative. Two other household members, who were known to have positive tuberculin skin tests, had normal chest radiographs. Three people who came in contact with the bird at the veterinarian's office were later noted to have no response to tuberculin skin testing.

Necropsy. The bird was seized and euthanatized under the authority of the New York City Health Code after the owner refused multiple requests to have the bird euthanatized. Necropsy revealed miliary granulomatous pneumonia, hepatitis, stomatitis, glossitis, myocarditis, endocarditis, and conjunctivitis. Acid-fast bacilli were identified in the lung, liver, and skin lesions. Histopathology revealed classic tuberculous nodules with a central necrotic core surrounded by epithelioid macrophages, multinucleated giant cells, and a fibrous capsule. There was no mineralization. Specimens from the lungs and skin were culture positive for *M. tuberculosis*, and the antimicrobial susceptibility and restriction fragment length polymorphism patterns of the postmortem isolates were identical to those of the antemortem isolates.

Discussion. Birds can have avian, bovine, and human tuberculosis. More recently, disseminated disease with the emerging human pathogen *Mycobacterium genavense* has been reported for a variety of birds (5–7, 9). Mycobacterial infections in nonpsittacine birds are usually caused by the ubiquitous soil and water organism *Mycobacterium avium* (3, 8), and the route of infection is usually the alimentary tract. Most lesions develop in the intestinal tract, liver, and spleen. Birds only occasionally suffer from pulmonary *M. avium* disease.

Parrots suffering from disease caused by *M. tuberculosis* usually present with cutaneous lesions. This report describes a macaw with active tuberculosis caused by *M. tuberculosis*. Epidemiologic investigation revealed that two people who lived in the same house with the pet bird and who had close respiratory contact with it had active tuberculosis 3 to 4 years before diagnosis of tuberculosis in the bird. One patient did not adhere to antituberculosis treatment for more than 1 year. The bird probably contracted the disease from its human housemates. This type of situation presents public health implications in that a bird could acquire multiply drug-resistant strains, possibly infect other humans, and even act as a perpetual reservoir if the bird survives as long as the one discussed in this report.

Any parrot with ocular, sinus, oral, or cutaneous nodular lesions should be considered suspect for *M. tuberculosis* infection. It is imperative from a public health perspective that tissue be sent for culture and not only for acid-fast staining. Birds probably acquire the infection from humans, but it is not known if humans can acquire the infection from birds. However, birds may be sentinels for human infection.

REFERENCES

1. **Ackerman, L. J., S. C. Benbrook, and B. C. Walton.** 1974. *Mycobacterium tuberculosis* infection in a parrot (*Amasona farinosa*). Am. Rev. Respir. Dis. **109:**388–390.
2. **Brown, R.** 1990. Sinus, articular and subcutaneous *Mycobacterium tuberculosis* infection in a juvenile red-lored Amazon parrot, p. 10–15. *In* Proceedings of the 1990 Annual Conference of the Association of Avian Veterinarians.
3. **Hines, M. E., II, H. M. Kreeger, and A. J. Herron.** 1995. Mycobacterial infections of animals: pathology and pathogenesis. Lab. Anim. Sci. **45:**334–351.
4. **Hinshaw, W. R.** 1933. Tuberculosis of human origin in an Amazon parrot. Am. Rev. Tuberc. **28:**273–278.
5. **Hoop, R. K., E. C. Böttger, P. Ossent, and M. Salfinger.** 1993. Mycobacteriosis due to *Mycobacterium genavense* in six pet birds. J. Clin. Microbiol. **31:**990–993.
6. **Kiehn, T. E., H. Hoefer, E. C. Bottger, R. Ross, M. Wong, F. Edwards, N. Antinoff, and D. Armstrong.** 1996. *Mycobacterium genavense* infections in pet animals. J. Clin. Microbiol. **34:**1840–1842.
7. **Portaels, F., L. Realini, L. Bauwens, B. Hirschel, W. M. Meyers, and W. De Meurichy.** 1996. Mycobacteriosis caused by *Mycobacterium genavense* in birds kept in a zoo: 11-year survey. J. Clin. Microbiol. **34:**319–323.
8. **Quinn, P. J., M. E. Carter, B. Markey, and G. R. Carter.** 1994. *Mycobacterium* species, p. 156–169. *In* P. J. Quinn, M. E. Carter, B. Markey, and G. R. Carter (ed.), Clinical veterinary microbiology. Mosby-Wolfe, London, England.
9. **Ramis, A., L. Ferrer, A. Aranaz, E. Liebana, A. Mateos, L. Dominguez, C. Pascual, J. Fdez-Garayazabal, and M. D. Collins.** 1996. *Mycobacterium genavense* infection in canaries. Avian Dis. **40:**246–251.
10. **Woerper, M. S., and W. J. Rosskoph.** 1983. Retro-orbital *Mycobacterium tuberculosis* infection in a yellow-naped Amazon parrot (*Amazona ochrocephala auropalliata*), p. 71–76. *In* Proceedings of the 1983 Annual Meeting of the Association of Avian Veterinarians.