Psittacosis is caused by *Chlamydia psittaci*, an obligate intracellular parasite found worldwide. Humans are infected with *C. psittaci* when the organism enters the bloodstream, usually through inhalation of dried excreta from diseased birds or through wound contamination with infected avian secretions. *C. psittaci* replicates in the liver and spleen and infects the lung and other organs hematogenously.

The clinical manifestations of human psittacosis range from a mild respiratory infection to a severe systemic illness. Symptoms are frequently described as flu-like with fever, headache, body aches, and dry or productive cough. Sore throat, chest pain, abdominal pain, vomiting, and diarrhea are variably present. Physical findings may include a pulse-temperature dissociation, localized lung crackles, hepatomegaly, splenomegaly, and a pale macular skin rash. Chest radiographs may demonstrate lesions that are atelectatic, patchy, miliary, nodular, or consolidated in one or both lungs. White cell counts, erythrocyte sedimentation rates, and liver function tests are usually normal. In severe illness, signs and symptoms of liver dysfunction, neurological impairment, and respiratory and renal failure may be present.

Since 1879 when psittacosis was recognized as a disease entity, cases have been reported in North and South America, Europe, Asia, and Australia. However, reports of psittacosis in Africa have been rare. An Ethiopian group, studying community-acquired pneumonia, published what they claimed to be the first report of psittacosis in Africa in 1994. The report published here is believed to be the first documented case of human psittacosis in Egypt.

**Case Presentation**

A 43-year-old American female, living in Egypt, presented at a company clinic in Cairo with a fever of two days' duration accompanied by dizziness, myalgia, fatigue, anorexia, occasional dry cough, and "heaviness" in her chest. She had been seen at the clinic a month earlier for a similar episode, associated with a severe headache, which resolved after taking doxycycline at 100 mg twice daily for 7 days.

The medical history revealed no underlying disease. Socially, the patient was a light smoker, a homemaker, and a bird-fancier. At the time of her illness, she was caring for three parrots, one of which was ill and later died.

On the initial exam, the patient was alert and appeared well, in spite of a fever of 38.8°C (101.8°F). Pulse was 108 per min, and respirations were 16 per min. Head, neck, and chest exams were normal. There was mild hepatomegaly with tenderness, but no detectable splenomegaly. No skin rash or icterus was present; however, trace amounts of urinary bilirubin and urobilinogen were detected by dipstick. She was given symptomatic treatment and sent home under observation.

Three days later, the patient developed a sharp pain in her left neck and shoulder, exacerbated by deep breathing and movement. Her temperature was 37.5°C (99.5°F) with pulse 96 per min and respirations 24 per min. Crackles were heard over the left, lower lung field. The liver was still enlarged, but no longer tender. Chest radiographs revealed partial pneumatic consolidation of the left lower lobe. A complete blood count disclosed mild anemia, not present a month earlier, and a normal white cell count of 7000 cells/mm³. The erythrocyte sedimentation rate was 25 mm/hr. Liver function tests and urinalysis were normal. The patient was unable to produce sputum for a Gram's stain. A TB skin test was non-reactive.

The patient was treated for atypical pneumonia, with doxycycline at 100 mg twice daily for 25 days. The differential diagnosis included mycoplasmal and chlamydial pneumonia, legionnaires’ disease, and Q-fever. She improved rapidly after the initiation of therapy. Within 3 days, she was afebrile and pain-free with a nonpalpable liver. After 1 week of therapy, the chest x-ray was normal.

Serological testing for psittacosis was arranged with difficulty. A local laboratory imported a complement-fixation test kit at a cost equivalent to the monthly salary of many Egyptian physicians. An acute serum complement-fixation titer was 1:40. Two convalescent serum samples drawn 2 weeks and 16 weeks after the initiation of therapy were sent to the Centers for Disease Control and Prevention (CDC) in the United States for immunofluorescence assay. CDC reported negative IgM titers on both samples and IgG titers of 1:256 and 1:64 at 2 and 16 weeks, respectively.

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J Travel Med 1997; 4:48-49.
Discussion

The patient’s laboratory tests, clinical presentation, and history of exposure to an ill psittacine bird all support the diagnosis of psittacosis. The diagnosis of psittacosis is based primarily on serological testing in corroboration with clinical and epidemiologic data. There are two methods by which serological testing is done: complement fixation (CF) and microimmunofluorescence (MIF) assay. Both tests are technically difficult, time consuming, expensive, and often unavailable.4

CF is the conventional diagnostic method. CF testing is considered diagnostic of psittacosis if there is a fourfold rise or fall of IgG titers in repeat serum samples over a period of up to 4 months is evidence of probable infection. Minimally significant MIF titers have not yet been defined, although a single IgG titer of 1:256 is considered high and indicative of infection in a patient with a compatible illness. IgM titers are probably insufficiently sensitive to be used alone for diagnostic purposes.4

While the patient’s serological results are indicative of psittacosis infection, they do not define the time of disease onset. It is possible that the initial attack of psittacosis in this case occurred one or more months earlier and that the illness reported represents either a reinfection or relapse of the disease.

The recommended treatment for psittacosis is tetracycline or its analogues daily for 14–21 days.13 With inadequate or delayed antibiotic therapy, psittacosis may become a chronic or recurrent disease.4 In a small number of cases, erythromycin, ofloxacin, and ceftriaxone have been reported to be as effective as tetracycline or its analogues daily for 14–21 days.5 With inadequate or delayed antibiotic therapy, psittacosis may become chronic or recurrent disease.4

Prevention

Human psittacosis may be prevented by avoiding exposure to diseased birds. Parrots, parakeets, cockatiels, and other psittacine birds are most often infected with C. psittaci, although other birds are also susceptible. The organism is excreted in the feces and nasal discharge of infected birds, which may be asymptomatic, and is resistant to drying, remaining viable for months.10

The United States Association of Public Health Veterinarians recommends that psittacine birds not acquired from disease-free breeding colonies receive feed containing at least 1% chlortetracycline (CTC) for a total of 45 days.10 The administration of antibiotics through drinking water is not effective. Currently, the United States government mandates 30 days of quarantine with CTC feed for all imported psittacine birds and advises the importers to continue the treatment for an additional 15 days. Treated birds should be isolated from untreated birds since reinfection may occur.

Conclusion

Psittacosis is probably under-reported in Africa and throughout the world because the disease lacks distinctive symptoms, and diagnostic tests are not readily available. As this case illustrates, psittacosis is present in Egypt and may pose a potential health threat to tourists and expatriates. Persons visiting or living in Egypt and other countries where the importation and sale of birds is not regulated or where regulations are not enforced should be advised to avoid visiting bird markets and to treat pet psittacine birds with CTC feed according to U.S. recommendations.

Acknowledgments

The author wishes to thank David A. Sack, M.D., and Kerry L. Taylor, D.V.M., M.Sc., for their review of this manuscript.

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