Chlamydia psittaci

An Uncommon Cause of Community-Acquired Pneumonia

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hlamydia psittaci is an uncommon cause of community-acquired pneumonia. Fewer than 200 cases of psittacosis are reported annually to the Centers for Disease Control and Prevention; however, many cases are believed to be unreported. Unrecognized cases not properly treated may result in significant morbidity and possibly have a fatal outcome. We describe a 42-year-old man with multilobar pneumonia and respiratory compromise secondary to infection with *C psittaci*. The patient also exhibited hepatic involvement and mild anemia as a result of systemic infection. Appropriate antibiotic therapy and respiratory support resulted in a good clinical outcome. The diagnosis of psittacosis requires serologic verification. All confirmed cases should be reported to local and state health departments and may require further investigation by the Centers for Disease Control and Prevention. (Arch Fam Med. 1993;2:997-1001)

Community-acquired pneumonias are commonly seen by family physicians and are usually of a viral or bacterial origin. Pneumonias that have extrapulmonary manifestations and fail to respond to traditional outpatient therapy are usually referred to as "atypical pneumonias." Included in this group of respiratory pathogens are members of the Mycoplasma, Legionella, and Chlamydia genera. Respiratory infections caused by Chlamydia, especially Chlamydia pneumoniae, are becoming more commonly recognized in adults. The following case discusses a less commonly seen type of atypical pneumonia—that caused by Chlamydia psittaci.

REPORT OF A CASE

A 42-year-old man presented to his physician's office with a 1-week history of productive cough, myalgia, and low-grade fever. He was diagnosed as having bronchitis and was treated with a twice daily regi-

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men of trimethoprim in combination with sulfamethoxazole.

Three days later, he presented to the emergency department complaining of shortness of breath, fever, and a worsening cough productive of blood-tinged sputum. Additional complaints included headache and diarrhea. A chest roentgenogram revealed bilateral basilar infiltrates (**Figure 1**). An arterial blood gas on room air revealed a PaO₂ of 52 mm Hg with an 88% saturation. His white blood cell count was 6.5×10°/L, with 55% neutrophils, 40% band forms, and 4% lymphocytes. He was admitted to the hospital with a diagnosis of community-acquired pneumonia.

The patient's medical history was unremarkable for any previous hospitalizations or chronic medical illnesses, including asthma. He had no history of pneumonia or tuberculosis exposure. He took no medications regularly. He was a lifelong nonsmoker and he denied any alcohol use. He was employed as a maintenance worker and had no significant environmental exposures. His wife operated a pet bird shop adjacent to their house where she sold and bred domestic and imported birds. There

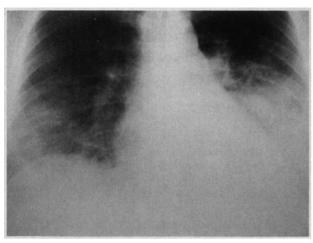


Figure 1. Patient's chest roentgenogram at admission showing bibasilar pulmonary infiltrates.

was no recent travel history; however, the patient had been on vacation 2 weeks before admission during which time he spent working at his wife's pet shop.

On admission, the patient was moderately ill, appearing with a temperature of 39°C, pulse rate of 80 beats per minute, blood pressure of 130/89, and respirations of 20 breaths per minute.

Sclera were anicteric and there were no oropharyngeal lesions. The lung examination revealed left-sided rales with decreased basilar breath sounds. The results of the cardiac examination were normal, with no murmur or rub. The abdominal examination was negative for hepatomegaly or splenic enlargement. The results of the extremity and neurological examinations were normal.

Laboratory data obtained the day of admission revealed a hemoglobin level of 118 g/L, a mean corpuscular volume of 76 fL, and a platelet count of 243×10^9 /L. The electrolytes were normal except for a potassium level of 3.3 mmol/L. An electrocardiogram showed sinus tachycardia with nonspecific ST-segment and T-wave abnormalities.

Blood and sputum cultures were obtained in the emergency department in addition to *Legionella pneumophila* and *Mycoplasma pneumoniae* antibody titers. The patient was started on therapy with a 50% ventilation mask and given aerosolized treatments with albuterol sulfate every 4 hours. Empiric therapy with intravenous cefuroxime sodium and oral erythromycin base was instituted.

Approximately 24 hours after admission, the patient complained of worsening shortness of breath and chest discomfort. An arterial blood gas revealed a PaO₂ of 46 mm Hg. He was transferred to the intensive care unit and therapy with a 100% oxygen nonrebreather mask was started. A pulmonary consultant discontinued the cefuroxime and added gentamicin sulfate and mezlocillin sodium to the antibiotic regimen. The erythromycin was also changed to an intravenous form. The patient was oxygenated via nasal continuous positive airway pressure as it was believed that mechanical ventilation could result in barotrauma if the patient had a necrotizing pneumonia.

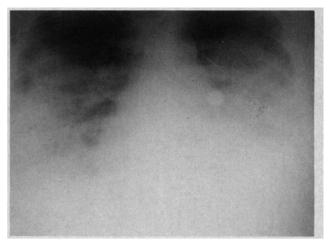


Figure 2. Patient's chest roentgenogram at day 5 of his hospitalization showing extension of the disease into the upper lobes.

Further history obtained from the patient's wife was that 4 months earlier, she had several parakeets at her shop that were ill. They were treated by a veterinarian with antibiotic-containing birdseed and recovered uneventfully. Infection with *C psittaci* was apparently suspected but not confirmed. No one else in the family had been ill since that time.

In light of this information, intravenous doxycycline hyclate was added to the patient's antibiotic regimen. Serum samples for antibodies to *C psittaci* and *C pneumoniae* were obtained.

The patient remained stable during the next 48 hours and did not require mechanical ventilation. A second chest roentgenogram revealed worsening of the initial infiltrates with extension into the upper lobes (Figure 2). The patient became afebrile on the fourth day after hospitalization and his oxygen requirements decreased. Sputum cultures during this time revealed 4+ normal flora, and antibodies for legionella and mycoplasma were negative. A subsequent white blood cell count was 5.8×10⁹/L. Laboratory studies disclosed other abnormalities as follows: γ -glutamyltransferase, 333 U/L; alkaline phosphatase, 387 U/L; lactate dehydrogenase, 4.29; aspartate aminotransferase, 70 U/L; and alanine aminotransferase, 149 U/L. The patient continued to clinically improve and on the ninth day after hospitalization, a report was received that confirmed evidence of chlamydial antibody by enzymelinked immunosorbent assay at 0.99 (0.71 or greater indicates a high level of detectable antibody). Two days later, IgG antibody specific for C psittaci antibody was positive at 1:128 (active infection indicated by a titer of 1:64 or greater). IgG antibody against C pneumoniae was positive at 1:64 (active infection indicated by a titer of 1:256 or greater). Intravenous antibiotic therapy was discontinued and the patient was placed on oral doxycycline. A third chest roentgenogram showed moderate clearing of the bilateral pulmonary infiltrates (Figure 3). The patient was discharged on the 13th day after hospitalization. At 1 week 1 month after discharge, the patient was well with no further pulmonary symptoms.

COMMENT

Psittacosis or "parrot fever" is a relatively uncommon infectious disease and thus infrequently seen by most practicing clinicians. From 1982 to 1991, there were 1344 cases reported to the Centers for Disease Control and Prevention (CDC). Only six deaths occurred in these patients. The actual incidence of the disease is likely much greater, but many less severe cases go undiagnosed or unreported.

The causative agent is an obligate intracellular bacterium *C psittaci*. It is closely related to the other chlamydial species *Chlamydia trachomatis* and *C pneumoniae* (TWAR strain [TWAR being the letter designates of the first two isolates of this species]). The DNA homology of these three species may result in cross-reactivity when serological testing for antibody is performed.

Chlamydia psittaci is transmitted to humans by numerous avian species, but most commonly by psittacine birds (from the Greek word psittakos for parrot), which include parrots and parakeets. The term ornithosis is sometimes used for this same disease but more specifically applies to disease spread by nonpsittacine birds, which include pigeons, chickens, finches, and turkeys. Chlamydia psittaci may be found in the tissues (especially liver and spleen), blood, feathers, and stools of infected birds. Transmission from birds to humans is via inhalation of infected aerosols from feces or fecal dust.² The organism then travels to the reticuloendothelial cells of the liver and spleen where it replicates. Hematogenous invasion of lung tissue and other organs then follows. An interesting feature applicable to this case is that birds who recover from infection may shed the agent for many months. Moreover, brief contact is all that is necessary to acquire the infection. Person-to-person transmission has been reported and may result in more severe disease.2,3

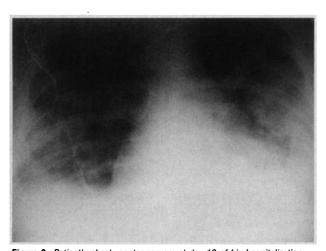


Figure 3. Patient's chest roentgenogram at day 13 of his hospitalization showing gradual clearing of the pulmonary infiltrates.

Table 1. Signs or Symptoms of Patients With Psittacosis—United States, 1975-1984*

Sign or Symptom	No. (%) of Patients
Fever	818 (72)
Cough	503 (44)
Headache	435 (38)
Weakness/fatigue	379 (33)
Chills	371 (33)
Myalgias	287 (25)
Nausea/vomiting	162 (14)
Anorexia	145 (13)
Chest pain	122 (11)
Diaphoresis	121 (11)

^{*}Percentages are based on 1136 patients (from the Centers for Disease Control and Prevention¹⁷).

The majority of cases reported to the CDC occur in owners of pet birds. Zoo workers, pet-shop employees, parakeet breeders, pigeon fanciers, and veterinarians are also at risk of acquiring the infection. During the 1980s, there were two major outbreaks of psittacosis reported at turkey processing plants in Minnesota and North Carolina. More than 300 individuals were infected. Pigeons have also been identified as a major source of *C psittaci* infection, accounting for about 10% of all reported cases.

CLINICAL MANIFESTATIONS

The incubation period after exposure to *C psittaci* usually ranges from 7 to 21 days but may be longer. A variety of clinical presentations may occur in infected individuals (**Table 1**). The disease may be insidious with a gradual onset of headache, malaise, anorexia, and a dry cough. However, more often it will begin abruptly with high fever and shaking chills. The cough will often become more severe and productive of mucoid sputum with blood streaking. An interesting aspect of psittacosis is a relative bradycardia, which is typical for intracellular infections such as typhoid or brucellosis. Gastrointestinal symptoms, especially diarrhea and vomiting, are also seen. Headache is quite common and often severe enough to suggest meningeal involvement. Myalgias and arthralgias often accompany the illness.

Physical findings may include hepatomegaly and splenomegaly, the latter occurring in about 10% to 70% of infected individuals.² Dermatologic manifestations of psittacosis may include a macular rash known as "Horder's spots," splinter hemorrhages, erythema marginatum, urticaria, and erythema nodosum.^{8,9} Auscultation of the chest will often not correlate with the severity of the roentgenographic findings.

Infrequently seen but well-documented complica-

An illness characterized by fever, chills, headache, photophobia, lower or upper tract respiratory disease, and myalgias
Isolation of <i>Chlamydia psittaci</i> from a clinical specimen, or fourfold or greater increase in psittacosis complement-fixation antibody titer (≥32) between specimens obtained greater than 2 wk apart
Probable = a clinically compatible illness linked to a confirmed case or with supportive serologic test results (a complement-fixation antibody titer of 1:32 or greater from one or more specimens after onset of symptoms)

*These serologic findings may also occur as a result of infection with Chlamydia trachomatis or Chlamydia pneumoniae (from the Centers for Disease Control and Prevention¹⁴).

tions of *C psittaci* infection include endocarditis and myocarditis. ^{10,11}

Recently, Tsapas et al¹² reported a case of psittacosis in which the patient experienced a reactive arthritis. Only four similar cases have been reported in the medical literature.

LABORATORY FINDINGS

Most patients with psittacosis will have a normal or slightly elevated total white blood cell count. Leukopenia may occur in patients with more severe cases. Some patients will develop anemia during the acute phase of the illness. Additional abnormalities include elevations in hepatic transaminases (aspartate aminotransferase and alanine aminotransferase) along with an increased bilirubin and alkaline phosphatase concentration. The majority of these findings were seen in the patient described above; however, none are specific for psittacosis.

Chest roentgenographic manifestations may include segmental or lobar infiltrates, especially in the lower lung fields. However, miliary, atelectatic, and nodular patterns have also been described.²

DIAGNOSIS

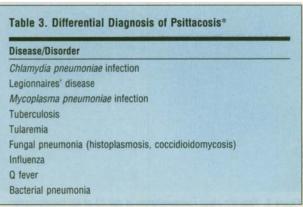
With an appropriate history and clinical presentation, a presumptive diagnosis of psittacosis can often be made. However, epidemiologic data suggest that at least 20% of infected individuals had no history of contact with birds. ^{2,13} The study by Crosse¹⁴ out of Great Britain included 219 patients, and only 62% reported contact with birds. Nonetheless, this remains an important historical point when questioning patients.

Confirmation should be done through the use of lab-

oratory studies. The organism can be cultured from the blood during the first 4 days of infection and from the sputum during the first 2 weeks. However, some authorities discourage routine culturing because of the risk it presents to laboratory workers. Isolation of C psittaci via culture from a throat swab is also possible. Serological testing is the usual method to confirm psittacosis. An IgG microimmunofluorescent antibody titer of 1:64 or greater is evidence of acute or recent infection (SmithKline Beecham Laboratories, Cypress, Calif). The detection of IgM antibody is also possible but is less specific as there may be cross-reactivity with the two other chlamydial species. An IgM immunofluorescent antibody (IFA) titer of 1:20 or greater is indicative of recent infection. An alternative serological test is the CDC requirement of a fourfold or greater increase in psittacosis complement-fixation antibody titer (≥32) obtained greater than 2 weeks apart and studied at the same laboratory (Table 2).15 The micro-IFA technique is the preferred method of testing as the complement-fixation test does not distinguish between the three species of Chlamydia. However, even with the micro-IFA technique, cross-reactivity remains a possibility and must be considered in the context of the entire clinical picture of the patient. This is especially true in light of the increased recognition of C pneumoniae becoming a more common respiratory pathogen, which should be considered in the differential diagnosis of psittacosis (Table 3). Early treatment with antibiotics may delay the immune response so a definitive serologic diagnosis may take several weeks.16 Therefore, the diagnosis is often made after full recovery or retrospectively.

TREATMENT

Treatment of psittacosis is with tetracycline hydrochloride at a dose of 500 mg every 6 hours or doxycycline at a dose of 100 mg every 12 hours. ^{2,12,16} An oral or intravenous route may be used depending on the clinical circumstances. An alternative therapy is erythromycin for children or pregnant women. The new macrolide antibi-



^{*}From Schaffner² and Swartz and Schoolnik.¹⁵

otics clarithromycin and azithromycin may represent potential therapy based on their in vitro activity against *C* trachomatis. However, there are no clinical studies to date to support their efficacy. The offending organism has also been found to be sensitive in vitro to rifampin. The initial response to therapy may be slow and treatment should be for at least 10 days after defervescence as relapses have been reported.² In addition, initial infection does not confer lifelong immunity and reinfection has been documented.^{2,16}

PROGNOSIS

The prognosis for treated patients is quite good. Significant morbidity seems to be associated with reported cases and in some family outbreaks. The case fatality rate is only about 1% compared with 20% to 40% in the preantibiotic era.

CONCLUSION

Psittacosis is an infrequently seen but an underdiagnosed infectious disease. ¹⁷ A thorough history will often aid in making the diagnosis but should always be confirmed with serologic antibody testing. Whenever possible, the original source of infection should be identified to prevent further spread of *C psittaci*. Confirmed cases should be reported to local and state health departments or directly to the Respiratory Diseases Branch of the CDC. ¹⁷

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REFERENCES

- Centers for Disease Control. Summary of notifiable disease: United States, 1991. MMWR Morb Mortal Wkly Rep. 1991;40:57.
- Schaffner W. Chlamydia psittaci (psittacosis). In: Mandell GL, Douglas RG, Bennett JE, eds. *Principles and Practice of Infectious Disease*. 3rd ed. New York, NY: Churchill Livingstone Inc; 1990:1440-1443.
- Bourke SJ, Carrington D, Frew CE, Stevenson RD, Banham SW. Serological cross-reactivity among chlamydia strains in a family outbreak of psittacosis. J Infect. 1989;19:41-45.
- Hedberg K, White K, Forfang JC, et al. An outbreak of psittacosis in Minnesota turkey industry workers: implications for modes of transmission and control. Am J Epidemiol. 1989:130:569-577.
- Centers for Disease Control. Psittacosis at a turkey processing plant: North Carolina, 1989. MMWR Morb Mortal Wkly Rep. 1990;39:460-469.
- Henry K, Crossley K. Wild-pigeon-related psittacosis in a family. Chest. 1986; 90:708-710.
- Centers for Disease Control. Psittacosis surveillance, 1975-84. MMWR Morb Mortal Wkly Rep. 1987;36:33.
- Sernel JD. Cutaneous findings in a case of psittacosis. Arch Dermatol. 1984; 120:1227-1229
- Green ST, Hamlet NW, Willocks L, et al. Psittacosis presenting with erythemamarginatum-like lesions: a case report and a historical review. Clin Exp Dermatol. 1990;15:225-227.
- Shapiro DS, Kenney SC, Johnson M, Davis CH, Knight ST, Wyrick PB. Brief report: Chlamydia psittaci endocarditis diagnosed by blood culture. N Engl J Med. 1992;326:1192-1195.
- 11. Coll R, Horner I. Cardiac involvement in psittacosis. BMJ. 1967;4:35-36.
- Tsapas G, Klonizakis I, Casakos K, Koncouris L. Psittacosis and arthritis. Chemotherapy. 1991;37:143-145.
- Williams LP. Review of the epidemiology of chlamydiosis in the United States. J Am Vet Med Assoc. 1989;195:1518-1521.
- 14. Crosse B. Psittacosis: a clinical review. J Infect. 1990:21:251-259.
- Centers for Disease Control. Case definitions for public health surveillance. MMWR Morb Mortal Wkly Rep. 1990;39:28-29.
- Swartz MN, Schoolnik MD. Diseases due to chlamydia. In: Rubenstein E, Federman DD, eds. Scientific American Medicine. New York, NY: Scientific American: 1986:6-7.
- Lardner AD, Lett S, Harvey E, et al. Human psittacosis linked to a bird distributor in Mississippi—Massachusetts and Tennessee, 1992. MMWR Morb Mortal Wkly Rep. 1992;41:794-797.