Wild-Pigeon-Related Psittacosis in a Family

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Three members of a family acquired psittacosis after exposure to a wild pigeon. Each of the patients had pulmonary infiltrates, prominent headache, abdominal complaints, and serologic evidence for infection with Chlamydia psittaci. Of 759 cases of psittacosis reported to the Centers for Disease Control for the period of 1974 to 1981, some 75 (10 percent) were associated with pigeons. Fifty-two of the cases were associated with domestic pigeons and 23 with wild pigeons. Pigeons represent a largely unrecognized reservoir of psittacosis in the United States.

Psittacosis is an infrequent cause of pneumonia. While the association with psittacine birds (parrots and parakeets) and turkeys is widely recognized, the risk of acquiring the disease from pigeons is less well known. We recently saw three members of a Cambodian family admitted with fever and pneumonia to medical services at this hospital during one weekend. Persistent questioning through an interpreter revealed exposure to a sick wild pigeon and allowed us to make the diagnosis of psittacosis.

Case Reports

Case 1

The first patient was a 22-year-old Cambodian man admitted because of headache, fatigue, night sweats, mild cough, and abdominal pain. The symptoms had been present for two weeks. Except for an episode of lead intoxication related to his employment at a battery factory, he had previously been well.

On admission, his temperature was 39.5°C (103.1°F), respiratory rate was 16/min, heart rate was 80 beats per minute, and blood pressure was 115/80 mm Hg. The findings from physical examination were otherwise normal, without evidence for hepatospleno megaly or pulmonary consolidation. An examination of sputum was nondiagnostic, with no predominant bacteria seen. The chest x-ray film demonstrated a right middle lobe infiltrate. The leukocyte count was 10,700/cu mm, with 5,400 polymorphonuclear leukocytes and 1,070 band cells per cubic millimeter. Serum electrolyte levels were normal. The level of serum glutamic-oxaloacetic transaminase (SGOT) was increased (128 IU/L, normal, 5 to 28 IU/L), as was the level of lactic dehydrogenase (LDH) (510 IU/L, normal, 98 to 194 IU/L). The concentrations of alkaline phosphatase and bilirubin were normal. The patient was assumed to have a pneumococcal pneumonia, and intravenous therapy with penicillin was begun. On the third day of hospitalization, he became more acutely ill, and the infiltrate was larger.

Because two other members of his family had been admitted with similar complaints, the patient was questioned through an interpreter about common exposures. The patient's wife reported that a sick bird had been found outside their apartment four weeks previously, had been brought indoors for three weeks, and was released the week prior to admission. The bird, which began to fly around the living room after two weeks, did not have nasal discharge or watery feces. The family identified the bird (using photographs and Peterson's Field Guide to the Birds2 as a rock dove (pigeon). Because of this information, therapy with penicillin was discontinued, and treatment with tetracycline was begun; the patient became afebrile within one day. The immediate complement fixation titer against Chlamydia group antigen (performed at the Minnesota Department of Health) was 1:8, and the titer rose to 1:32 four weeks later. Serologic studies for Mycoplasma and Histoplasma were negative. The patient received two weeks of therapy with tetracycline and recovered fully.

Case 2

The second patient was the 62-year-old father of the first patient, who lived in the same apartment. He was admitted because of fever, chills, cough, nausea, and headache for a period of one week. His medical history was unremarkable.

On admission, the patient appeared acutely ill, with an oral temperature of 40.2°C (104.4°F). There were decreased breath sounds over the right upper lobe and no hepatosplenomegaly. A single analysis of sputum showed more than ten epithelial cells per low-power field, more than 25 neutrophils per low-power field, and multiple bacteria morphologic forms. A chest x-ray film demonstrated a right upper lobe infiltrate. The white blood cell count was 10,900/cu mm, with 5,177 polymorphonuclear leukocytes and 2,070 band cells per cubic millimeter. The serum electrolyte levels were normal. The LDH level was 259 IU/L, and the SGOT level was 115 IU/L.

After cultures of blood, urine, and sputum were collected, intravenous therapy with ampicillin and erythromycin was begun. The patient's condition showed little change until it began to improve clinically on the third day of hospitalization. At that time, the history of exposure to the pigeon was known, and the patient's therapy was changed to oral treatment with tetracycline, which was continued for 14 days. The patient's recovery was uneventful, and he was discharged on the fourth day of hospitalization. His immediate complement fixation titer against Chlamydia group antigen was 1:16, and the titer was 1:32 four weeks later. Serologic studies for Mycoplasma and Histoplasma were negative. No serologic test for Legionella was performed.

Case 3

The third patient was the 54-year-old mother of the index case. She was hospitalized because of a one-week history of malaise, fever, nonproductive cough, midepigastic pain, and headache. Her symptoms began at the same time as those of her husband.

At the time of admission, her patient's temperature was 40°C (104°F), her pulse was 110 beats per minute, and the respiratory rate...
was 24/min. Findings on examination included a perforated right tympanic membrane and posterior bilateral rales on auscultation of the chest. Except for guaiac-positive stools, the findings from the patient's examination were unremarkable. The chest x-ray film demonstrated a left lower lobe infiltrate and a small left pleural effusion. The patient's white blood cell count was 7,900/cu mm, with 4,060 polymorphonuclear leukocytes and 2,172 band cells per cubic millimeter. The hemoglobin level was 10.9 g/dl. Abnormal results on tests of hepatic function included an LDH level of 273 I/U/L, SCOT level of 62 I/U/L, an alkaline phosphatase level of 240 units/L (normal, 30 to 115 units/L), and a y-glutamyl transferase of 126 units/L (normal, 0 to 65 units/L). Arterial blood gases with the patient breathing room air revealed a pH of 7.53, an arterial oxygen pressure of 62 mm Hg, and an arterial carbon dioxide tension of 31 mm Hg.

Intravenous therapy with penicillin was begun. The pleural space was tapped and revealed a transudative effusion with negative cultures. Evaluation for the gastrointestinal hemorrhage was unrevealing. When the history of exposure to the pigeon was known, the patient was treated with tetracycline for a period of two weeks. Her immediate complement fixation titer against Chlamydia group antigen was 1:256, with a repeat titer four weeks later of 1:512. Serologic studies for Mycoplasma and Histoplasma were again negative, and a cold agglutinin titer was less than 1:40. On follow-up examination, the patient had fully recovered.

**DISCUSSION**

This is apparently the first family outbreak in the United States of psittacosis associated with a wild pigeon. All three of these patients had clinical and serologic findings compatible with psittacosis. The illness was most likely acquired from a pigeon which they kept in their apartment for three weeks. Two other family members lived in the apartment but had no clinical complaints and declined to have blood drawn for serologic testing.

We believe that it is unlikely that the three cases of pneumonitis described herein were due to another infection. Besides the history of exposure to a pigeon, one of the patients met the criteria of the Center for Disease Control for a confirmed case, and the other two cases met criteria for presumptive cases. Two of the cases had nondiagnostic analysis and cultures of sputum, and all three had negative serologic studies for Mycoplasma and Histoplasma. The serologic diagnosis of psittacosis is most often made retrospectively, and it is rare (and possibly hazardous) to culture the organism during the acute episode. The institution of appropriate therapy with antibiotics may actually make it more difficult to confirm the diagnosis, since antibiotics can blunt the serologic response. The degree and frequency that such blunting occurs is not known and should not deter therapy.

Psittacosis was first described by Ritter in 1879, when he reported an illness which involved five members of his brother's household and two visitors. Three of the original seven patients died. The original association was with parrots and finches. The first cases in the United States were reported in 1904 by Vickery and Richardson, who described an illness affecting three of four family members after exposure to a sick green parrot. The etiologic agent of psittacosis (Chlamydia psittaci) was not isolated until 1930 by Bedson and Bland. 4

In 1941, K. F. Meyer reported that pigeons could serve as a reservoir for psittacosis and recommended that the disease be termed, "ornithosis," to indicate that birds other than psittacines could serve as vectors for the disease. A study of flocks of pigeons in Iowa and California found an incidence of complement-fixing antibody to Chlamydia group antigen which ranged from 30 to 90 percent and averaged 50 percent. 5 In 1965, Meyer isolated Chlamydia psittaci from 645 (19.9 percent) of 3,248 pigeons using techniques of tissue culture.

Chronic infection of psittacine birds continues as a reservoir of psittacosis in the United States. Whether endogenous infection of wild pigeons constitutes a similar reservoir is not known. Pigeons usually live close to human habitation, and a large number of domestic pigeons are raised as poultry and for racing or show. Populations of pigeons in metropolitan areas are often very large; approximately 16,000 pigeons are destroyed annually for public health reasons in St. Paul, Minn (St. Paul Department of Health).

The clinical picture of avian chlamydiosis in the pigeon is variable. The acutely ill dove may become anorexic and develop conjunctivitis, diarrhea, ruffled feathers, and respiratory difficulty. 6,7 The most common course is probably inapparent infection or simply a mild diarrheal illness. Surviving birds become chronic carriers and may persistently contaminate their environment. In 1942, Meyer et al demonstrated that parent-to-nestling transmission may be responsible for sustaining avian chlamydiosis. The parents shed organisms in nasal and intestinal fluids which are ingested or inhaled by their nestlings.

Table 1 summarizes cases of psittacosis reported to the Center for Disease Control from 1975 to 1981. After a decline in total reported cases of psittacosis, there has been an upsurge in the years from 1977 to 1981. A portion of that increase can be attributed to several outbreaks in turkey-processing plants. 8 An increase of cases related to parakeets was recently observed in Minnesota during a period of active surveillance (M. T. Osterholm, Minnesota Department of Health, oral communication, 1982). The association with wild or domestic pigeons is also presented. During that six-year period, there were 23 cases of psittacosis acquired from wild pigeons and 52 cases associated with domestic pigeons.

Although there has been a family epidemic of psittacosis associated with a parakeet in the United States, 9 this family outbreak is apparently the first documented in the United States that was associated with wild pigeons. There are numerous isolated cases of pigeon-related psittacosis reported, such as five of
Table 1—Pigeon-Related Cases of Psittacosis Reported to the Center for Disease Control from 1975 to 1981*

<table>
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<th>Year</th>
<th>Total Cases</th>
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<th>Suspected</th>
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<tr>
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<td>Totals</td>
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</table>

*Total possible pigeon-related cases, 75; total wild pigeon-related cases, 23. Data were provided by Morris Potter, D.V.M., Division of Bacterial Diseases, Center for Disease Control, Atlanta.

†Confirmed case defined as four-fold increase in complement fixation antibody (CFA) to Chlamydia group antigen (CGA); presumptive case defined as compatible illness with CFA to CGA at least 1:16; and suspected case defined as compatible illness with exposure but no serologic studies obtained.

the nine cases detailed by Seibert et al14 in 1956. Schachter and Dawson13 described a small outbreak of psittacosis at the University of California, San Francisco, which they traced to pigeons that spent time on office windowsills. These investigations13 suggested that many cases of apparent nonavian psittacosis might have resulted from occult exposure to infected pigeon droppings.

There are no pathognomonic clinical features of psittacosis that aid in the diagnosis in the absence of a history of exposure to birds. All three of our patients had several interesting findings. These included headache, absence of splenomegaly, abdominal complaints, and abnormal levels of transaminases and LDH. The first case clearly demonstrated a pulse-temperature dissociation. The occurrence of headache and abdominal complaints are features of this disease that have previously been noted.14 Abnormal tests of hepatic function may reflect involvement of hepatic reticuloendothelial cells, which may also explain the hepatomegaly which is common in this disease.14 Occurrence of a pleural effusion (seen in patient 3) is also unusual but has been described.15

This report should remind physicians to ask about exposure to pigeons when taking a history from patients with atypical pneumonia. Pigeon-related cases constitute about 10 percent (75/759) of the cases of psittacosis reported to the Center for Disease Control between 1975 and 1981. Because the disease may be unrecognized and is probably underreported, this likely underestimates the number of cases occurring in the United States.

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