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CASE STUDY

PSITTACOSIS

Dhanalakshmi B

Department of Community Health Nursing, Sree Balaji College of Nursing, Bharath University, Chrompet, Chennai, Tamil Nadu, India

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ABSTRACT

The word "psittacosis" is used when the disease is carried by any species of bird belonging to the family Psittacidae, whereas "ornithosis" is used when other birds carry the disease. In humans, after an incubation period of 5–19 days, the symptoms of the disease range from inapparent illness to systemic illness with severe pneumonia. It presents chiefly as an atypical pneumonia. Complications in the form of endocarditis, liver inflammation, inflammation of the heart's muscle, joint inflammation, keratoconjunctivitis (occasionally extranodal marginal zone lymphoma of the lacrimal gland/orbit), and neurologic complications (brain inflammation) may occasionally occur. .[5] Most persons respond to oral therapy doxycycline, tetracycline hydrochloride, or chloramphenicol palmitate.

INTRODUCTION

Psittacosis — also known as parrot fever, and ornithosis — is a zoonotic infectious disease caused by a bacterium called *Chlamydophila psittaci* and contracted from infected parrots, such as macaws, cockatiels and budgerigars, and pigeons, sparrows, ducks, hens, gulls and many other species of bird. The incidence of infection in canaries and finches is believed to be lower than in psittacine birds. In certain contexts, the word "psittacosis" is used when the disease is carried by any species of bird belonging to the family Psittacidae, whereas "ornithosis" is used when other birds carry the disease.[1]

Signs and Symptoms

In humans, after an incubation period of 5–19 days, the symptoms of the disease range from in apparent illness to systemic illness with severe pneumonia. It presents chiefly as an atypical pneumonia. In the first week of psittacosis the symptoms mimic typhoid fever: prostrating high fevers, joint pains, diarrhea, conjunctivitis, nose bleeds and low level of white blood cells in the blood.[2] Rose spots can appear and these are called Horder's spots.[3] Spleen enlargement is common towards the end of the first week. Diagnosis can be suspected in case of respiratory infection associated with splenomegaly and/or epistaxis. Headache can be so severe that it suggests meningitis and some nuchal rigidity is not unusual. Towards the end of the first week stupor or even coma can result in severe cases.

The second week is more akin to acute bacteremic pneumococcal pneumonia with continuous high fevers, cough and dyspnea. X-rays show patchy infiltrates or a diffuse whiteout of lung fields.

Complications in the form of endocarditis, liver inflammation, inflammation of the heart's muscle, joint inflammation, keratoconjunctivitis (occasionally extranodal marginal zone lymphoma of the lacrimal gland/orbit), and neurologic complications (brain inflammation) may occasionally occur. Severe pneumonia requiring intensive-care support may also occur. Fatal cases have been reported (less than 1% of cases).

Diagnosis

Blood analysis shows leukopenia, thrombocytopenia and moderately elevated liver enzymes. Differential diagnosis must be made with typhus, typhoid and atypical pneumonia by Mycoplasma, Legionella or Q fever. Exposure history is paramount to diagnosis. Diagnosis involves microbiological cultures from respiratory secretions of patients or serologically with a fourfold or greater increase in antibody titers against *C. psittaci* in blood samples combined with the probable course of the disease. Typical inclusions called "Leventhal-Cole-Lillie bodies"[4] can be seen within macrophages in BAL (Bronchial Alveolar Lavage) fluid. Culture of *C. psittaci* is hazardous and should only be carried out in biosafety laboratories.

Treatment

The infection is treated with antibiotics. Tetracyclines and chloramphenicol are the drugs of choice for treating patients

with psittacosis.[5] Most persons respond to oral therapy doxycycline, tetracycline hydrochloride, or chloramphenicol palmitate. For initial treatment of severely ill patients, doxycycline hyclate may be administered intravenously. Remission of symptoms usually is evident within 48–72 hours. However, relapse can occur, and treatment must continue for at least 10–14 days after fever abates.

Epidemiology

Psittacosis was first reported in Europe in 1879.[6]
In 1929, a highly publicized outbreak of psittacosis hit the United States. Although not the first report of psittacosis in the United States, it was the largest up to that time. It led to greater controls on the import of pet parrots.[6] The aftermath of the outbreak and how it was handled led to the establishment of the National Institutes of Health.[7]

From 2002 through 2009, 66 human cases of psittacosis were reported to the Centers for Disease Control and Prevention and most resulted from exposure to infected pet birds, usually cockatiels, parakeets, and macaws. Many more cases may occur that are not correctly diagnosed or reported. Bird owners, pet shop employees, zookeepers and veterinarians are at risk of the infection. Some outbreaks of psittacosis in poultry processing plants have been reported.

In Birds,

Chlamydia psittaci infection is referred to as avian chlamydiosis (AC). Infected birds shed the bacteria through feces and nasal discharges, which can remain infectious for several months. Many strains remain quiescent in birds until activated under stress. Birds are excellent, highly mobile vectors for the distribution of chlamydial infection because they feed on, and have access to, the detritus of infected animals of all sorts.

Signs

C. psittaci in birds is often systemic and infections can be inapparent, severe, acute or chronic with intermittent shedding. Signs in birds include "inflamed eyes, difficulty in breathing, watery droppings and green urates."[8]

Diagnosis

Initial diagnosis may be via symptoms, but is usually confirmed via an antigen and antibody test. A PCR-based test is also available. Although any of these tests can confirm psittacosis, false negatives are possible and so a combination of clinical and lab tests is recommended before giving the bird a clean bill of health.[8] It may die within three weeks.

Epidemiology

Infection is usually via the droppings of another infected bird, though it can also be transmitted via feathers and eggs,[9] and is typically either inhaled or ingested.[8]

C. psittaci strains in birds infect mucosal epithelial cells and macrophages of the respiratory tract. Septicaemia eventually develops and the bacteria become localized in epithelial cells and macrophages of most organs, conjunctiva, and gastrointestinal tract. It can also be passed in the eggs. Stress will commonly trigger onset of severe symptoms, resulting in rapid deterioration and death. *C. psittaci* strains are similar in virulence, grow readily in cell culture, have 16S-rRNA genes that differ by <0.8%, and belong to eight known serovars. All should be considered to be readily transmissible to humans.

C. psittaci serovar A is endemic among psittacine birds and has caused sporadic zoonotic disease in humans, other mammals, and tortoises. Serovar B is endemic among pigeons, has been isolated from turkeys, and has also been identified as the cause of abortion in a dairy herd. Serovars C and D are occupational hazards for slaughterhouse workers and for people in contact with birds. Serovar E isolates (known as Cal-10, MP or MN) have been obtained from a variety of avian hosts worldwide and, although they were associated with the 1920s–1930s out break in humans, a specific reservoir for serovar E has not been identified. The M56 and WC serovars were isolated during outbreaks in mammals.

Treatment

Treatment is usually via antibiotics, such as doxycycline or tetracycline, and can be administered via drops in the water, or injections.[9] Many strains of *C. psittaci* are susceptible to bacteriophage.

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