


CASE REPORTS

Rhodococcus equi infection: a cause of cavitary pulmonary disease in immuno-compromised patients

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SUMMARY

Rhodococcus equi is a facultatively-aerobic, gram-positive and intracellular bacteria that causes pulmonary and disseminated infections in immuno-compromised patients. It is a common agent that causes pneumonia in farm animals and only rarely is transmitted to man. I report a case of pneumonia caused by this agent in a patient with AIDS who had been misdiagnosed as tuberculosis and treated as such for five months before the correct diagnosis was made. The diagnosis should be suspected on clinical and radiologic grounds as treatment is considerably different from other opportunistic pathogens in AIDS.

INTRODUCTION

Pulmonary infection remains an important cause of morbidity and mortality in immuno-compromised patients. With the global epidemic of AIDS, an increasing incidence of unusual opportunistic pathogens has been observed. Rhodococcus equi is a weakly acid-fast, pleomorphic coccobacillus that is principally a pathogen of horses, cattle, swine, and sheep. In these animals, it causes pulmonary and sometimes disseminated infections.

Human cases have been reported, with one exception, in immuno-compromised patients. Ten other cases of Rhodococcus infection have been reported in AIDS patients, but as in the case presented here, this infection often is confused with tuberculosis. Though the clinical and radiologic features of this disease resemble those of tuberculosis, presence of weakly staining acid fast bacilli and poor response to antitubercular drugs, as well as failure to isolate mycobacteria, should suggest this diagnostic possibility.

CASE REPORT

A 35 year old man was admitted to the hospital in May 1993 because of chronic cough, nocturnal fevers, night sweats and shortness of breath. The patient had been in a stable state of health until four years earlier when he had tested positive for antibodies to the human immunodeficiency virus (HIV) type 1 during investigation for unexplained weight loss. The patient led a homosexual lifestyle but denied any intravenous drug abuse or history of blood transfusions. He had two cats and a dog but denied exposure to other animals. Several months before admission, the patient had been extensively investigated for unexplained watery diarrhoea which subsequently was treated with oral loperamide. Zidovudine was started at a dose of 200mg four times daily for CD4+ count of 485/mm³ (normal 537–571).

Physical examination revealed mild cachexia and an oral temperature of 38.1°C. There was no rash or lymphadenopathy. The blood pressure was 116/74mm Hg, and pulse was 102 with respirations at 22/minute. The heart was normal but crackles were heard in the right upper lobe. Abdominal examination was normal. The hematocrit was 46 pc; the white cell count was 7100 (7.1x 10⁹) with 78 pc neutrophils, six pc lymphocytes and 10 pc monocytes and four pc band forms. A tuberculin skin test (5TU) was negative, as was a skin test for Candida. A chest X-ray revealed an infiltrate in the right upper lobe. A sputum smear revealed weakly
acid-fast organisms but cultures in Lowenstein–Jensen (LJ) medium were negative after six weeks. Blood cultures were negative.

A diagnosis of pulmonary tuberculosis was suspected and the patient was started on isoniazid, ethambutol, and rifampicin at usual doses. The CD4+ lymphocyte count was 160/mm³. The patient was discharged from hospital after a week’s stay with resolution of fever. A month later the patient was seen at another hospital with fever, dyspnea and productive cough and was started on ciprofloxacin for presumed bronchitis without much improvement. Two weeks later the patient returned to the hospital with dizziness, severe dyspnea, fever and drenching sweats. The patient also was noted to have a 10 pound weight loss, as well as retrograde amnesia.

His physical examination revealed worsening cachexia, temperature of 38.9°C and supine blood pressure of 110/60. He had a pulse of 112 and respiratory rate of 24/minute. The heart was normal but the patient had wheezes and crackles in the right lung fields. The patient was disoriented in time but did not have any meningeal signs. The chest X-ray revealed worsening infiltrates with cavitary lesions in the right upper lobe. Magnetic Resonance Imaging of the brain was normal. The CD4+ lymphocyte count was 20/mm³ at his admission. Cerebrospinal fluid analysis revealed elevated protein of 80mg/100ml without pleocytosis. The rest of laboratory data revealed a haemoglobin of 8.4g/L, a platelet count of 119 x 10⁹ normal serum electrolytes and creatinine. Liver function tests were abnormal with lactate dehydrogenase of 2198 U/L, bilirubin of 2.3 and aspartate aminotransfere of 341 U/L. The arterial blood gas analysis revealed a pH of 7.45, a PaO₂ of 65mmHg with oxygen saturation of 93% on room air. Blood cultures as well as culture of bronchial washing grew Rhodococcus equi. Intravenous erythromycin 500mg four times daily was started based on sensitivity results. Rifampicin also was continued, while isoniazid and ethambutol were stopped.

The patient became afebrile after about five days of therapy and was discharged after a two week hospital stay. The patient was continued on oral erythromycin after discharge. Three months later the patient’s symptoms relapsed. He suffered a cardio–respiratory arrest and died soon after readmission to the hospital.

**DISCUSSION**

*Rhodococcus equi* is a well known pathogen of livestock, affecting cattle, horses and swine. It has been isolated from the soil in all continents except Antarctica. Like mycobacteria, it contains mycolic acid in its cell wall, so stains weakly positive on acid-fast staining. The organism is often seen in macrophages, suggesting that it is a facultative intracellular organism. The disease is thought to be acquired by inhalation. Gholub et al reported the first case of human infection by *Rhodococcus equi*. The organism can cause pulmonary as well as brain abscesses, osteomyelitis, and cutaneous abscesses, and can be isolated from blood, sputum, abscesses and bronchial washings, after culture in routine media such as chocolate agar or five percent sheep blood agar. There is usually heavy growth of non-hemolytic colonies which may be catalase positive, but urease and citrate negative. The pathogen is readily isolated but may be disregarded as a contaminant or, because it may be weakly acid-fast, be mistaken for mycobacteria.

Other epidemiologic features of this infection, such as the predilection for immunocompromised patients, only enhances the ease with which the infection is confused with tuberculosis. Furthermore, this organism often causes a cavitary lesion in the upper lobes of the lung which could also suggest tuberculosis. The lower lobes of the lungs also may be affected. It is interesting that in all cases of pulmonary infection so far reported, the chest reontgenograms were abnormal. Current antibiotic recommendations for this organism emphasize the need to initiate therapy with lipid soluble bactericidal antibiotics such as vancomycin, imipenem and erythromycin. The organism is sensitive to rifampin, and aminoglycosides, though resistance to β-lactam antibiotics has been reported. Maintenance therapy on antibiotics for three to six months after an initial course of antibiotic is recommended.

In conclusion, a case of Rhodococcus pneumonia in a young man with AIDS is presented. The diagnosis was initially missed. A high index of suspicion is needed to diagnose the infection. Definitive diagnosis is necessary as treatment usually leads to a good response. Use of lipid soluble bactericidal antibiotics to initiate therapy is recommended. In this era of AIDS pandemic, it cannot be emphasized too strongly that not all cavitary pneumonia in an immunodeficient patient is tuberculosis. This merely supports the updated aphorism... “That he who knows AIDS knows medicine.”

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INTRODUCTION

The prevalence of epilepsy in the developing countries of the tropics is much higher than in developed countries. The relatively high prevalence in developing countries is largely due to poor primary health care facilities, including a lack of effective immunisation programmes, and endemic parasitosis of the central nervous system (CNS).

Epileptic seizures may complicate CNS parasitosis acutely, or as a sequelae to chronic infection and brain scarring. Epilepsy is well recognised as a consequence of cysticercosis, toxoplasmosis, hydatid cysts and schistosomiasis. There are, however, no clinical reports associating epileptic seizures with filariasis, even though cerebral infestation by microfilaria is well known. We report a case of epileptic seizures occurring in association with filariasis.

CASE REPORT

A 70 year old farmer presented with a two year history of generalised, tonic–clonic epileptic seizures. Seizure frequency on presentation was once monthly. All seizures were preceded by an aura, and were associated with a post–ictal coma lasting several minutes, and amnesia for the event. Seizures were not associated with headaches, febrile illnesses, chronic drug use or features of hypoglycemia. The patient was not a known diabetic or hypertensive. There was no family history of epilepsy and no history of head trauma preceding the seizures.

General physical examination was normal. Blood pressure was 120/70 mmHg seated. Fundoscopy was normal. A detailed neurological and cardiovascular examination revealed no abnormality. Laboratory examination showed a PCV of 38 pc, a WBC total count of 8450 with eosinophilia of 24 pc. Microfilaria of Loa–loa were found in the blood. A skin–snip examination revealed microfilariae of Onchocerca volvulus. The patient did not consent to a lumbar puncture.

Electrolyte and urea were within normal limits. A skull X–ray was normal. A single interictal eight channel electroencephalography recorded over 30 minutes with hyperventilation did not reveal any abnormality. There are no facilities in our hospital for CAT scanning.

A clinical diagnosis of focal epileptic seizures with secondary generalisation, in association with multiple filariasis was made. The patient was treated with stan-