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CASE REPORT

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Empyema due to *Rhodococcus hoagii*, an unusual presentation

Empiema por Rhodococcus hoagii, una presentación poco habitual

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ABSTRACT

Rhodococcus hoagii is an unusual infectious agent in humans. It typically presents as an opportunistic infection in immunosuppressed patients, although it has also been reported as an occupational disease following exposure to farm animals (such as horses, goats, sheep and cattle). The most common clinical presentation is slowly resolving cavitary pneumonia, so it should be included in the differential diagnosis of tuberculosis. Less common manifestations include pleural effusion, empyema, and lung abscess. We report the case of a 57-year-old man, HIV-positive, who was employed as a racetrack stable worker, diagnosed with empyema caused by *Rhodococcus hoagii* (presently *R. equi*).

Key words: empyema | infection | *Rhodococcus hoagii* | HIV | occupational exposure

RESUMEN

En humanos, *Rhodococcus hoagii* es un agente infeccioso inusual. Suele producir infecciones oportunistas en pacientes inmunosuprimidos aunque también está descrita como una enfermedad relacionada con la exposición ocupacional (contacto con animales de granja como caballos, cabras, ovejas, vacas en ámbito laboral). La forma de presentación más común es como neumonía cavitada de lenta resolución, por lo que debe considerarse como diagnóstico diferencial de la tuberculosis; y con menor frecuencia como derrame pleural, empiema o absceso pulmonar. Se presenta el caso de un paciente masculino de 57 años, retrovirus positivo, trabajador en establos del hipódromo, con diagnóstico de empiema por *Rhodococcus hoagii* (actualmente *R. equi*).

Palabras clave: empiema | infeccion | *Rhodococcus hoagii* | HIV | exposición ocupacional

INTRODUCTION

Rhodococcus hoagii (formerly *Rhodococcus equi*) is a Gram-positive intracellular coccobacillus commonly found in the soil and is best known in veterinary medicine as a pathogen causing infection in horses. In humans, it is an opportunistic pathogen that primarily causes pulmonary infection, typically presenting as chronic cavitary or necrotizing pneumonia in immunocompro-

mised hosts, particularly individuals with AIDS or solid organ transplantation. Less commonly, pulmonary abscess and empyema have also been reported as clinical manifestations.

CASE REPORT

A 57-year-old man, former smoker (40 pack-years), with HIV (undetectable viral load, CD4 count 142 cells/mm^3; excellent antiretroviral therapy adherence) was recently diagnosed with chronic obstructive pulmonary disease (COPD), Global Initiative for Chronic Obstructive Pulmonary Disease (GOLD) group B, stage 2. His COPD is well controlled with salmeterol/fluticasone and has no recent exacerbations. His occupational history included maintenance work in horse stables at a racetrack for 3 years, with regular exposure to horses. He presented with a 45-day history of dyspnea, New York Heart Association (NYHA) class II, left-sided chest pain, productive cough with hemoptoic sputum, and febrile episodes. During this period, he sought care at multiple emergency departments and received several antibiotic regimens without clinical improvement. Physical examination: oxygen saturation 96% on room air, afebrile, abolition of vesicular breath sounds in the left lower-middle lung field with local dullness to percussion and dullness over the spine. Laboratory testing revealed leukocytosis of 25,100 µL with neutrophilia; hematocrit 33%, hemoglobin 11 g/dL, urea 9.3 mmol/L; the remainder was unremarkable. Sputum acid-fast bacilli smear repeated twice was negative. Contrast-enhanced CT of the chest demonstrates centrilobular emphysema and a tree-in-bud pattern in the left upper lobe. There is left pleural thickening and a loculated pleural effusion, associated with an area of increased attenuation within the lingula, described as a mass with spiculated margins that exhibits enhancement following intravenous contrast administration. Fiberoptic bronchoscopy revealed abundant mucopurulent secretions but no evidence of endoluminal lesions. Bronchoalveolar lavage (BAL) and bronchoscopic transbronchial biopsy (BTB) samples were submitted for microbiological analysis, which yielded negative results. Following evaluation by thoracic surgery, the patient underwent left video-assisted thoracoscopic surgery (VATS) for pulmonary decortication and pleural biopsy. Pathological anatomy of the pleural biopsy revealed an acute-on-chronic, nonspecific, abscessed inflammatory process. Pleural fluid samples were also collected for microbiological culture; both the tissue and fluid samples were positive for *Rhodococcus hoagii* (sensitive to vancomycin, erythromycin, and levofloxacin). Consequently, a 28-day course of dual intravenous antibiotic therapy with vancomycin and levofloxacin was initiated, resulting in a favorable clinical response. A follow-up chest CT was performed (Figure 1). The patient was subsequently transitioned to oral antibiotic therapy. To date, the patient has completed a 6-month course of antibiotic therapy and is scheduled for follow-up radiological and pulmonary function testing.

Ethical considerations

Patient permission was obtained for the publication of this case, supported by a signed informed consent.

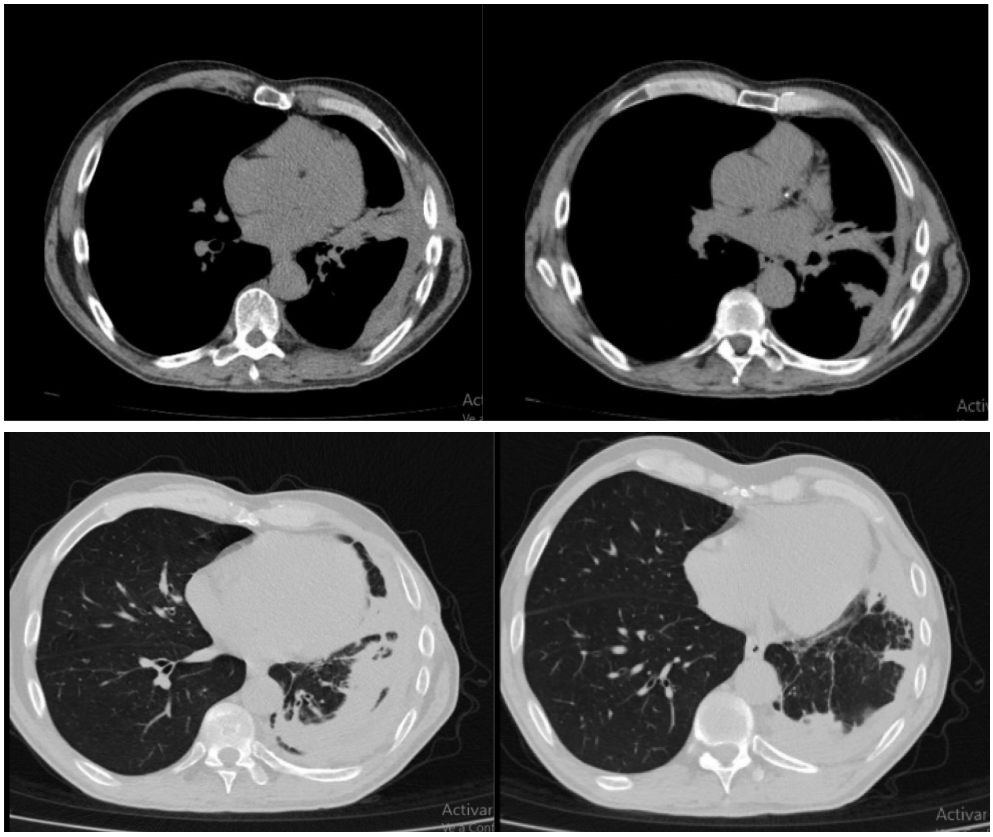
DISCUSSION

Rhodococcus hoagii is a soil-dwelling organism that colonizes the gastrointestinal tract of numerous herbivores and is commonly found in their feces, pasture soils, and other agricultural environments. Not only horses but also pigs, goats, and cattle should be considered potential sources of *R. hoagii* infection in humans.[1] More than 300 cases of R. hoagii infection have been reported since the first description of this disease in humans, in 1967.1 In that initial report, Golub et al identified the organism as the causative agent of a cavitary right upper lobe pneumonia, isolated from bronchoalveolar lavage fluid in a 29-year-old man with autoimmune hepatitis who had been receiving long-term corticosteroid therapy.[2]

Most reported cases occur in immunocompromised individuals, including patients with human immunodeficiency virus (HIV) infection, solid organ transplant recipients, and those undergoing cancer treatment.[1]

The primary route of infection acquisition is believed to be inhalation of the microorganism from contaminated soil. However, alternative routes have been described, including direct inoculation through skin or mucosal injuries and ingestion of contaminated food.[4] In several cases, exposure to farm and domestic animals, particularly dogs, has been considered a potential source of infection

Figure 1. Chest CT with mediastinal (1A) and lung parenchyma (1B) windows showing left lung volume loss associated with fibrotic bands extending from the pulmonary hilum to the periphery. A small left pleural effusion with underlying pleural thickening is also visualized.



through inhalation of contaminated fecal material or direct contact with infected skin lesions in these animals.[4] Nosocomial acquisition has been reported rarely, and only one case of occupationally acquired infection has been described in a laboratory worker.[5]

The most common clinical manifestation of *R. hoagii* infection in humans is focal nodular or cavitary pneumonia, which may be complicated by empyema, pleural effusion, pneumothorax, or endobronchial granuloma formation.[4] Extrapulmonary manifestations have also been reported and include subcutaneous abscesses, lymphangitis, endophthalmitis, septic arthritis, osteitis, pericarditis, renal abscesses, sepsis, and central nervous system infections, including meningitis and brain abscesses (Table 1).

The most common radiographic findings are poorly defined areas of consolidation and irregular cavitary lesions in the lungs, which may be unilateral or bilateral and typically involve the upper lobes. Approximately 70% to 75% of *R. hoagii* infections present as slowly resolving cavitary pulmonary disease, with radiographic features resembling those of *Mycobacterium tuberculosis* infection. In patients with cavitary lung lesions, the microorganism may be readily identified in sputum specimens. However, when isolated from the sputum of patients with pneumonia, *R. hoagii* may be dismissed as normal respiratory flora because of its morphologic resemblance to oropharyngeal commensal diphtheroids.[3]

Management is often challenging because of the organism's intrinsic resistance to multiple antimicrobial classes.[6] Antimicrobial susceptibility testing should be performed for all *R. hoagii* isolates to guide therapy and avoid the use of agents to which the organism demonstrates *in vitro* resistance. *R. hoagii* is generally susceptible to erythromycin, rifampin, fluoroquinolones, aminoglycosides, glycopeptides, and imipenem.5 Combination antimicrobial therapy should be considered to minimize the risk of emerging resistance.[6]

Most patients require initial intravenous treatment with bactericidal agents, such as imipenem and vancomycin, for at least 2 weeks, with the duration guided by clinical response, followed

Table 1. Previously Reported Clinical Manifestations of Rhodococcus equi Infection. Extracted from Weinstock DM, et al[8]

Pulmonary nodules
Lung abscess
Pneumonia, with or without cavitation
Spontaneous pneumothorax
Wound infection
Traumatic keratitis and endophtalmitis
Bacteremia, isolated and catheter related
Fever of unknouwn origin and infected bone marrow
Peritonitis, spontaneous and peritoneal dialysis associated
Mesenteric adenitis, isolated or with peritonitis
Cervical adenitis
Osteomyelitis, by direct extension from pneumonia or due to disseminated infection
Septic arthritis
Brain abscess, spontaneous or postneurosurgical
Hepatyc abscess, by direct extension from pneumonia or due to disseminated infection
Renal abscess
Urinary tract infection
Subcutaneous or deep-tissue abscess
Sternal-bound infection following coronary bypass grafting
Ventricular shunt infection
Bronchobiliary fistula by direct extension from pneumonia
Pedunculated and sessile colonic polyps
Otomastoiditis
Thyroid abscess
Colitis mimicking Whippie´s disease
Spleen abscece
Prostate abscece

by oral combination therapy (eg, a macrolide plus a quinolone).[5] The duration of treatment should be individualized according to the extent of infection, the patient's immune status, *in vitro* susceptibility profile, and clinical response to therapy.[6] Some authors recommend prolonged treatment for at least 6 months to reduce the risk of relapse.[5,6] In patients with HIV infection, initiation or continuation of antiretroviral therapy is also recommended to improve prognosis and reduce mortality.[7] Finally, surgical intervention should be considered when medical therapy fails, particularly in patients with pulmonary abscesses or empyema, as occurred in the present case.

CONCLUSION

Increasing recognition of *Rhodococcus hoagii* as a human pathogen has improved laboratory identification of infection in both immunocompromised and immunocompetent individuals. Close communication with the microbiology laboratory is essential in order to reach a precise diagnosis when clinical specimens are submitted, because the organism may be overlooked or misclassified as a contaminant agent. *Rhodococcus* infections remain challenging to treat and often require prolonged dual antibiotic therapy and surgical drainage, as in the case we present. In patients with HIV infection, concomitant antiretroviral therapy has been associated with improved survival outcomes. Finally, this entity should be considered in the differential diagnosis of tuberculosis, which represents a significant challenge.

Conflict of interest

Authors have no conflicts of interest to declare.

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