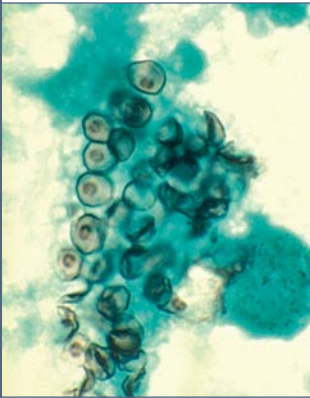


Pneumocystis jiroveci Overview

ABOUT THE ORGANISM

Pneumocystis organisms were first discovered in the early 20th century. The organisms have been identified in virtually every mammalian species, but have genetic characteristics that make them host-specific. The *pneumocystis* that infects humans was named *Pneumocystis carinii*; the resulting disease became known as *Pneumocystis carinii* pneumonia, or PCP. At the time, *P. carinii* was classified as a protozoa based on morphologic features, lack of growth on fungal media and susceptibility to antiprotozoal drugs. However, accumulating molecular genetic data showed that *P. carinii* should be reclassified as a fungus, which resulted in renaming the organism to *Pneumocystis jiroveci*. *P. jiroveci* is now the accepted name for the causative agent of PCP in humans. The acronym 'PCP' has been maintained and redefined to refer to Pneumocystis pneumonia.



Cysts of *Pneumocystis jiroveci* in smear from bronchoalveolar lavage.
Image courtesy of CDC.

CLINICAL MANIFESTATIONS

Primary infection with *P. jiroveci* is very common and largely asymptomatic. Serological studies have shown nearly universal seropositivity by two years of age. Until recently, *P. jiroveci* was thought to remain latent within a person following primary infection and reactivate when the immune system became compromised. Mounting evidence has shown that person-to-person transmission, including asymptomatic carriers, is the most likely means of acquiring new infections. Acquisition from environmental sources may also occur. Symptomatic disease, which manifests as PCP, is only seen in immunocompromised patients: those infected with HIV, cancer patients receiving chemotherapy, bone marrow and solid organ transplant recipients, as well as patients treated with corticosteroids and other immune suppressive agents. PCP is often the AIDS-defining illness in HIV patients, though the incidence has decreased due to the advent of HAART and prophylaxis. However, the incidence of PCP in non-HIV immunocompromised patients is increasing.

PCP most commonly presents as low-grade fever, dyspnea and cough. Physical examination often reveals tachypnea, tachycardia, and normal findings on lung auscultation. Hypoxemia is often found. The typical PCP x-ray shows diffuse, bilateral, fluffy infiltrates predominantly centrally located. Less common clinical presentations include extrapulmonary pneumocystis, such as hepatic pneumocystis, nodular granulomatous PCP and pleural effusions. Presentation of PCP in HIV patients is subtle, lasting from weeks to months, with a mortality rate of 10 to 20%. In non-HIV immunocompromised patients, PCP presents abruptly as respiratory failure, with a mortality rate of 30 to 60%.

LABORATORY DIAGNOSIS

P. jiroveci cannot be cultured in vitro. Therefore, routine diagnostics have relied upon microscopic examination of bronchoalveolar lavage (BAL) fluid or induced sputum utilizing either Giesma or direct immunofluorescence staining (DFA). However, these methods are technically difficult and dependent upon the quality of the sample. *P. jiroveci* infected cells disintegrate after a few hours; samples older than 3 hours must be rejected due to the increased risk of false negative results. Recently developed quantitative real-time PCR assays have the highest level of sensitivity and specificity and are gradually replacing microscopic techniques. In addition, quantitative real-time PCR offers the ability to monitor the patient's response to treatment over time. Lung biopsy tissue or BAL fluid are the specimens of choice for quantitative real-time PCR diagnosis.

TREATMENT

Prophylaxis against PCP in HIV-infected patients is recommended when the CD4+ count is less than 200 cells/mm³ or if there is a history of oropharyngeal candidiasis. Prophylaxis is also recommended for transplant patients. Trimethoprim-sulfamethoxazole (TMP-SMX) is considered first-line therapy in the prophylaxis of PCP. Intolerance and resistance to TMP-SMX can limit its use. Aerosolized pentamidine and atovaquone suspension have been used as alternatives. PCP prognosis depends on the amount of damage to the lungs prior to treatment.

Selected References

Bandt D, Monecke S. Development and evaluation of a real-time PCR assay for detection of *Pneumocystis jiroveci*. *Transpl Infect Dis*. 2007;(9):196-202.

Green H, Paul M, Vidal L, Leibovici L. Prophylaxis for pneumocystis pneumonia (PCP) in non-HIV immunocompromised patients. *Cochrane Database of Systematic Reviews* 2006, Issue 1. Art. No.: CD005590. DOI: 10.1002/14651858.CD005590.pub2.

Höcker B, Wendt C, Nahimana A, Tönshoff B, Hauser PM. Molecular evidence of *Pneumocystis* transmission in pediatric transplant unit. *Emerg Infect Dis*. 2005;11(2):330-332.

Stringer JR, Beard CB, Miller RF, Wakefield AE. Perspective: a new name (*Pneumocystis jiroveci*) for pneumocystis from humans. *Emerg Infect Dis*. 2002;(8):891-896.

Thomas CF, Limper AH. *Pneumocystis pneumonia*. *N Engl J Med*. 2004;(350):2487-2498

