Case Report

Pneumocystis jirovecii Pneumonia: A Life-threatening Infection of the Immunocompromised

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Abstract

Pneumocystis pneumonia (PCP) is a fatal pulmonary infection caused by an ascomycetous fungus, *Pneumocystis jirovecii*. The infection is mostly seen in patients with underlying disease conditions that alter the host immune status. However, immunocompetent hosts may also be transiently infected with self-limiting mild lower respiratory tract infection. Diagnosis of PCP requires demonstration of cysts of *Pneumocystis* in the lungs of the patient. Although an open lung biopsy is the most reliable method, a bronchoalveolar lavage is always more practical and nearly as sensitive. We report a case of a 32-year-old human immunodeficiency virus (HIV)-infected woman who presented with a 2-month history of nonproductive cough, respiratory distress, and fever. Her sputum examination showed growth of *Klebsiella pneumonia* and was sensitive only to levofloxacin. No acid-fast organisms were demonstrated. High-resolution computerized tomography scan of the thorax showed consolidated airspaces bilaterally with ground-glass opacities. Her CD4 count was 110 cells/mm^3. PCP was diagnosed by performing a Giemsa stain of bronchoalveolar lavage, which revealed cysts of *P. jirovecii*. Diagnosis was further confirmed by the elevated serum (1-3)-β-D glucan levels. She was started on co-trimoxazole and prednisolone, following which her symptoms gradually improved. PCP is one of the most common causes of HIV-associated mortality and a rapid diagnosis with an early initiation of treatment can significantly improve patient outcomes.

Keywords: Co-trimoxazole, immunocompromised host, *Pneumocystis jirovecii*, *Pneumocystis pneumonia*

Introduction

Pneumocystis pneumonia (PCP), a life-threatening pulmonary infection is caused by a yeast-like fungus, *Pneumocystis jirovecii*.[1] *P. jirovecii* pneumonia is a lung infection occurring mainly in human immunodeficiency virus (HIV)-infected patients with a helper T cell count <200 mm^3, recipients of solid organ transplantation, and people on immunosuppressive drugs.[2] A significant risk of PCP infection is seen in HIV-infected patients when glucocorticoids are coadministered with immunosuppressive medications.[1,3] HIV patients being treated with *Pneumocystis* chemoprophylaxis, highly active antiretroviral therapy (ART), and preventive treatment with trimethoprim/sulfamethoxazole (TMP/SMX) has decreased the incidence and mortality in developed countries.[1,2] *P. jirovecii* is associated with high mortality; a minimum of 10%–20% of patients with PCP have a fatal prognosis.[1]

A high mortality is noted when there is no treatment with prescription medications, patients with lung disorders, or those requiring ventilatory support. [1-3] PCP spreads through the inhalation route. *Pneumocystis* is species-specific, with humans being the sole reservoir.[2] After attaching to the Type I alveolar epithelium, the fungus transits from its trophic form to a cystic state.

Lung damage is mainly caused by the host's exuberant inflammatory response, leading to impaired gas exchange, resulting in hypoxia which eventually leads to respiratory failure. *P. jirovecii* is carried within the respiratory tracts of both symptomatic and asymptomatic individuals. The latter may unknowingly become reservoirs and spread the fungus to immunocompromised individuals.[1]

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Case Report

This is a case report of a 32-year-old woman who came to our hospital with complaints of nonproductive cough for 2 months, respiratory distress, and low-grade fever. For the past 4 months, she has been on antiretroviral therapy of tenofovir, lamivudine, and efavirenz for her HIV-positive diagnosis. Her vitals were: blood pressure 100/60 mm Hg, pulse rate 60 beats/minute, respiratory rate 18 breaths/min, oxygen saturation (SpO_2) 90% in room air, and body temperature 38°C. There was no rash, lymphadenopathy, or any bleeding manifestations. On further examination, there were chest rales with normal heart sounds. No additional sounds or murmurs were heard. Neurological examination revealed no motor, sensory, or autonomic deficits.

A chest X-ray showed features of bilateral, diffuse pulmonary infiltrates [Figure 1]. High-resolution computerized tomography thorax showed consolidated airspaces bilaterally with ground-glass opacities. The results of complete blood count, urinalysis, and biochemical analysis were normal, except for an elevated white cell count: 13000/mm^3. A raised serum lactate dehydrogenase (LDH): 629 (normal range: 119–229 U/L), a raised serum C-reactive protein: 12.8 mg/dl (normal range: <0.2 mg/dl), and a CD4 + lymphocyte level of 110 cells/μl were other significant findings. Her sputum examination showed no acid-fast organisms but the growth of *Klebsiella pneumonia* which was sensitive only to levofloxacin. Blood culture did not yield any microorganisms. The patient was started on once-daily dosage of intravenous levofloxacin (500 mg), along with nebulization (salbutamol) treatment three times daily. Her condition worsened on the 3rd day and she had to be put on noninvasive positive pressure ventilation. A fiberoptic bronchoscopy revealed a typical endobronchial system and bronchoalveolar lavage was obtained from the left lobe which was then stained by Giemsa stain, and it revealed cysts of *P. jirovecii* [Figure 2]. Diagnosis of *P. jirovecii* pneumonia was further confirmed by elevated serum (1-3)-β-D glucan level (40.2 pg/ml; normal level: <6.0 pg/ml).

The patient was then started on a regimen of SMX-TMP 1600 mg and 320 mg 8 hourly. Methylprednisolone [1000 mg] pulse therapy for 3 days was started followed by prednisolone 1 mg/kg/day. Her respiratory distress started subsiding 7th day onward. She was discharged after 9 days of hospitalization on SMX-TMP for pneumonia and a 21-day prednisone taper for inflammation.

Discussion

PCP was initially reported among the malnourished infants of Eastern and Central Europe after World War II. [1,4] The disease generally affects the immunocompromised. However, currently, immunocompetent individuals are no exception. [1,5] PCP is also reported in older populations without HIV infection due to the short-term use of high-dose steroids and low-dose methotrexate for rheumatoid arthritis treatment. [6,7]

Diagnosis of PCP can be challenging and multifactorial. Serum LDH level is usually elevated in HIV-infected patients and is therefore a sensitive finding, but high serum LDH levels may also be seen in immunocompromised individuals without HIV.[2] Since *P. jirovecii* cannot be grown on any culture media, its diagnosis requires microscopic detection and identification of the organism by staining using histochemical (Grocott-Gomori methenamine silver stain) or immunofluorescent stains.[1,2] Other respiratory pathogens, such as *Mycobacterium tuberculosis*, nontubercular mycobacterium, cytomegalovirus, Toxoplasma, disseminated Histoplasma, and bacterial pneumonia Cryptococcus should be also considered in its differential diagnosis.[2] The drug of choice for HIV-infected as well as uninfected patients is co-trimoxazole (TMP-SMX) orally or intravenously, 14 days for a mild case which may extend up to 3 weeks for severe cases. Intravenous pentamidine is usually administered in those who are intolerant of co-trimoxazole.[2] In case of sulfa allergy, alternative drugs are atovaquone, TMP, and dapsone, primaquine, and clindamycin, or pentamidine. Corticosteroids coadministered with anti-PCP therapy for 72 h aids in controlling lung inflammation

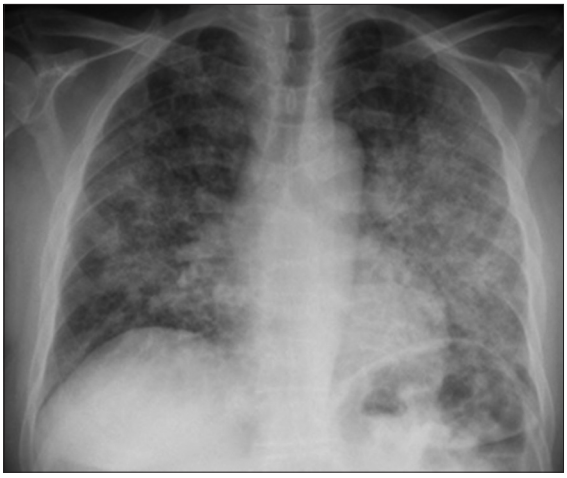

Figure 1: X-ray chest PA view showing bilateral diffuse consolidations

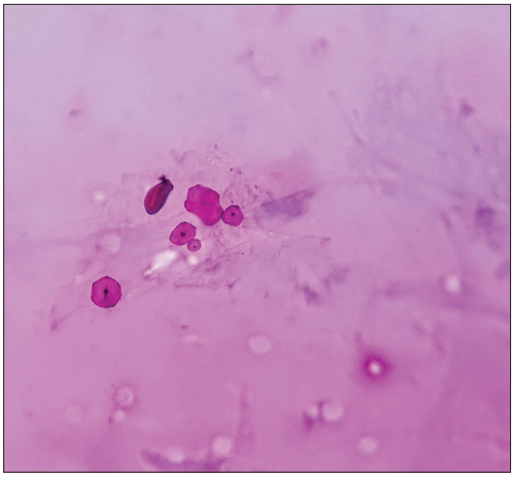

Figure 2: Giemsa stain smear showing cysts of *Pneumocystis jirovecii*

particularly in HIV-infected PCP patients with arterial oxygen partial pressure <70 mm Hg because corticosteroids could attenuate lung injury by blunting the inflammatory response initiated by the organisms' degradation and clearance.[2]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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