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An Atypical Case of PCP Pneumonia in a Patient on Chronic Immunosuppression

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Colonization or Infection? An Atypical Case of Pneumocystis Pneumonia in a Patient on Chronic Immunosuppression

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Introduction

Pneumocystis pneumonia (PCP) is an opportunistic infection caused by *Pneumocystis jirovecii* (previously *Pneumocystis carinii*), a yeast-like fungus colonizing up to 20% of immunocompetent adults. Progression from colonization to infection often occurs in the immunocompromised host. However, given this potential for non-infectious colonization, and in the absence of a clear etiology of immune-compromise such as HIV, diagnosis of PCP can be difficult. Diagnosis traditionally involves three core measures: chest x-ray (CXR) showing diffuse bilateral infiltrates, respiratory compromise, and identification of *Pneumocystis* in sputum or bronchoalveolar lavage (BAL). Novel diagnostic modalities including *Pneumocystis* qualitative PCR and serum B-D-glucan assay (Fungitell) have emerged in clinical practice to assist in diagnosis, however alone cannot truly differentiate colonization from infection. We present an atypical case of PCP, in which the above-mentioned diagnostic tests were used in both diagnosis and management of an HIV un-infected patient with history of prolonged immunosuppressant use for rheumatologic disease.

Case Report

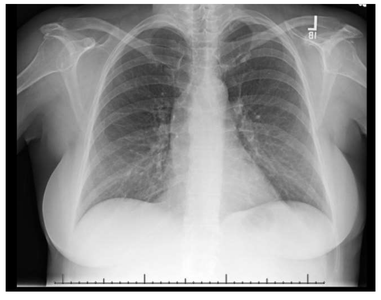
A 50 year-old woman presented to the emergency department with non-productive cough of 10 day duration, associated with subjective fevers and headaches. Past medical history was significant for mixed connective tissue disorder and seronegative SLE, on methotrexate 25 mg weekly, Plaquenil 400 mg daily, Mycophenolate mofetil 1500 mg twice daily, and prednisone 10 mg daily.

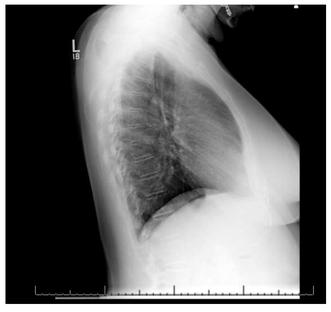
On admission, she endorsed fevers, fatigue, joint pains, dry cough, headaches, and shortness of breath with minimal exertion. On objective assessment she was febrile to 103 F (39.4 C), tachycardic to 125, and tachypneic to 22 with SpO2 86-88% on room air, improved with 3.0 liters oxygen by nasal cannula. Physical examination revealed acutely distressed woman, with oral thrush, a malar rash, photophobia, bilateral crackles with mild inspiratory rhonchi at lung bases, and regular rhythm tachycardia without murmurs.

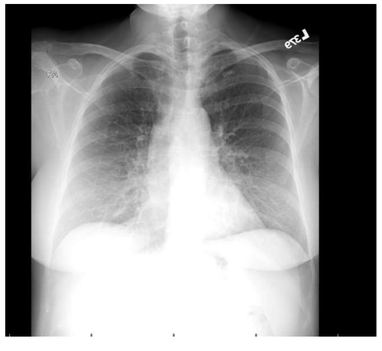
Case Report

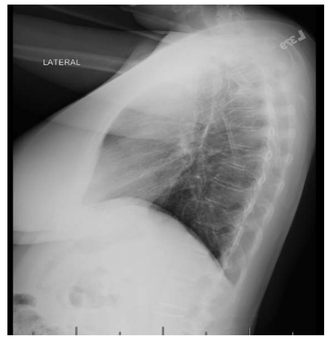
Laboratory evaluation demonstrated no leukocytosis, and no electrolyte abnormalities. Erythrocyte sedimentation rate (ESR) was elevated to 23 mm/Hr, and C-reactive protein level was elevated to 5.3 mg/dL. Arterial blood gas testing on room air revealed PO2 61.4, PCO2 41.9, pH 7.45, and SpO2 90.9%, with an A-a gradient 36.

Blood cultures collected on admission remained negative. Sputum culture and gram stain was unremarkable, and respiratory virus PCR was negative. Qualitative *Pneumocystis jirovecii* PCR on sputum sampling was positive, with a normal Fungitell (serum B-D-glucan) level of 31 pg/mL. HIV-testing was negative. Additional infectious workup, including lumbar puncture and 2-D echocardiogram, was unremarkable. Radiographic evaluation included chest x-ray which revealed mild non-specific perihilar and bibasilar opacifications questionable for atelectasis, and follow-up non-contrast chest CT again demonstrated bibasilar band-like opacities concerning for atelectasis.









Given her history of immunosuppressive therapy, most notably prolonged use of prednisone, with development of acute exertional dyspnea and hypoxemia and positive *Pneumocystis* qualitative PCR, and otherwise negative infectious work-up, she was diagnosed with PCP.

Case Report

She was started on a regimen of clindamycin 600 mg every 8 hours and primaquine 30 mg daily for 21 days of therapy, along with prednisone 40 mg daily adjuvant therapy given the extent of hypoxemia and A-a gradient on arterial blood gas testing, along with suspicion for underlying rheumatologic flare. She demonstrated dramatic improvement and was discharged on hospital day 8, without recurrence of symptoms at one week follow-up.

Discussion

Diagnosis of PCP in HIV-uninfected patients is based on a combination of clinical, laboratory, and radiographic findings. However, assessing progression of *Pneumocystis* colonization to infection in HIV-uninfected patients is difficult, especially in cases where typical radiographic findings are absent and adjuvant diagnostic modalities including serum B-D-glucan assay are normal. History of immunosuppressant use along with evidence of unremitting respiratory distress raises clinical suspicion for PCP in this case, along with positive *Pneumocystis* PCR testing. This case highlights the importance of high clinical suspicion and use of multiple diagnostic modalities in proper diagnosis and management of PCP.

Conclusion

PCP in HIV-uninfected patients is based on a combination of clinical, laboratory, and radiographic findings. When typical radiographic findings are absent and diagnostic modalities including B-D glucan assay are normal, patient history and high clinical suspicion become an important part of establishing the diagnosis of PCP.

References

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