An Atypical Case of PCP 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**

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.

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**