A Silent Progression: Unmasking HIV with Life-Threatening Pneumonia in a Previously Healthy Woman

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Background: Pneumocystis jirovecii pneumonia (PCP) remains a leading opportunistic infection in untreated HIV/AIDS. Delayed HIV diagnosis, especially in older adults and non-traditional risk groups, increases morbidity and mortality.

Case Presentation: We present a case of a 66-year-old woman with hypertension, diabetes, and hyperlipidemia who developed cough, dyspnea, and weight loss after failed outpatient pneumonia therapy. CT chest showed bilateral ground-glass opacities, and she improved with Bactrim and steroids. Months later, she re-presented with worsening respiratory failure and was diagnosed with PCP pneumonia, CMV viremia, and AIDS. She denied recent sexual activity; further history revealed a single unprotected encounter with her ex-husband years after divorce. Despite maximal antimicrobial and antiretroviral therapy, her course was complicated by immune reconstitution inflammatory syndrome (IRIS) and sepsis, and she ultimately died.

Conclusion: This case underscores the importance of maintaining HIV suspicion in atypical pneumonia, even in older or perceived low-risk patients. Early recognition, comprehensive infectious workup, and timely initiation of HAART and corticosteroids are critical to improving outcomes.

- Pneumocystis jirovecii pneumonia (PCP) is one of the most common and life-threatening opportunistic infections in patients with untreated HIV/AIDS, particularly when CD4 counts fall below 200 cells/μL. It typically presents with subacute fever, dyspnea, nonproductive cough, and hypoxemia out of proportion to physical exam findings. Imaging classically shows bilateral ground-glass opacities, and diagnosis requires microbiologic confirmation by PCR or staining of induced sputum or BAL. Adjunctive corticosteroids improve survival in moderate to severe disease, and early initiation of antiretroviral therapy (ART) reduces AIDS-related morbidity and mortality.
- Cytomegalovirus (CMV) reactivation becomes clinically significant in patients with profound immunosuppression (CD4 <50 cells/μL). While CMV viremia is common, invasive disease such as pneumonitis requires compatible clinical and radiologic features plus virologic or histologic confirmation. Coinfection with PCP and CMV can complicate management, worsen prognosis, and increase mortality.
- This case highlights the importance of maintaining high suspicion for HIV in patients with atypical pneumonia, timely recognition of PCP, and the impact of CMV viremia in advanced immunosuppression.

Case

A 66-year-old woman with hypertension, hyperlipidemia, and diabetes presented with chest pain, cough, and shortness of breath. She was admitted with acute hypoxic respiratory failure due to pneumonia. Initial sputum cultures grew *Streptococcus anginosus*. Despite broad-spectrum antibiotics, her condition worsened, requiring high-flow oxygen, then BiPAP. Workup revealed newly diagnosed HIV/AIDS, complicated by Pneumocystis jirovecii pneumonia (PCP) and superimposed CMV viremia/pneumonia.

Hospital Course

Day 1: Admitted with pneumonia and acute respiratory failure. Started on ceftriaxone and azithromycin → escalated to vancomycin and zosyn due to worsening hypoxemia.

Day 5–7: Oxygen needs increased (HFNC → BiPAP). Imaging showed bilateral infiltrates consistent with organizing pneumonia. High-dose IV steroids started.

Day 10: HIV test returned positive → antiretroviral therapy (Biktarvy) initiated. Developed fever → antibiotics changed to meropenem.

Day 12–14: Remained clinically stable; transferred to step-down unit. Fungitell assay positive.

Day 20: Bronchoscopy performed → PCP DFA and PCR positive. Caspofungin, avycaz, and vancomycin added.

Day 21: Developed severe hyponatremia (<120) unresponsive to fluids; treated with hypertonic saline per endocrinology.

Day 26: CMV PCR positive → started on ganciclovir.

CMV DNA, QN PCR	>6.30 ▲
CMV, DNA, QN Real Time PCR	>2,000,000 ▲
CMV DNA, Quantitative PCR	!

Day 31: Respiratory failure worsened; patient intubated, transferred to ICU, and started on vasopressors for septic shock.

Day 33: Ganciclovir changed to foscarnet due to clinical deterioration. Continued high-dose solumedrol.

Following days: Course complicated by acute kidney injury, thrombocytopenia requiring transfusions, disseminated intravascular coagulation (DIC), and refractory shock on multiple vasopressors.

Day 36: Despite maximal antimicrobial therapy, steroids, dialysis, and ventilatory support, the patient continued to decline and developed multiorgan failure.

Results

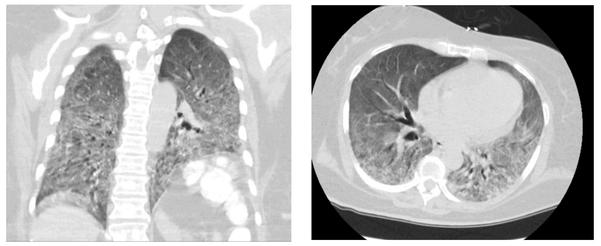

Figure 1 and 2. CT Chest AP and transverse view: Revealing diffuse ground glass opacities

Discussion

- Pneumocystis jirovecii pneumonia (PJP) is a serious infection in advanced HIV/AIDS, usually treated with trimethoprim-sulfamethoxazole and steroids. Most patients improve, but some fail therapy due to profound immunosuppression or co-infections.
- In this case, the patient's respiratory failure did not improve with standard PJP treatment. Further testing revealed superimposed CMV viremia, a complication linked to high mortality. CMV can worsen lung injury, suppress bone marrow, and interact with other infections. Starting antiretroviral therapy can also trigger immune reconstitution inflammatory syndrome (IRIS), adding to inflammation.
- This case shows the challenge of managing multiple opportunistic infections in AIDS. Clinicians should suspect CMV when PJP does not respond to standard therapy, as early antiviral treatment may help but prognosis remains poor in critically ill patients.

- This case demonstrates that HIV and its complications remain possible in all patients.
- Opportunistic infections in HIV/AIDS may mimic bacterial pneumonia (fever, cough, and dyspnea)
- Lack of improvement with standard therapy should raise suspicion for HIV/AIDS
- Unchecked infection can progress to sepsis with DIC
- Anchoring bias on a "typical" diagnosis (e.g., bacterial pneumonia) can obscure recognition of HIV/AIDS.
- Clinicians must maintain high suspicion for HIV in patients with atypical pneumonia, even in low-risk or older populations. Broad infectious workups, including fungal and viral pathogens, are critical in severe, non-resolving pneumonia. Early initiation of HAART and corticosteroids is vital in PCP.

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