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Pulmonary Pneumocystosis in a Patient with a CD4-Lymphocyte Count Greater than 200 cells/μL

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Abstract A 47-year-old man, who has no past medical history, presented to the hospital with shortness of breath. He was found to have HIV with a CD4 count of nearly 300 cells/μL, elevated LDH, and a chest x-ray depicting multifocal pneumonia with ground glass opacities. He was admitted to the intensive care unit and ultimately diagnosed with pneumocystis pneumonia status post bronchoscopy; making PCP his first AIDS-defining event. While PCP usually occurs when individual CD4 counts are <200 cells/μL, approximately 10% of cases occur in patients with higher counts.

Keywords: *acute dyspnea, human immunodeficiency, virus, pneumocystosis, pneumonia*

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1. Introduction

Opportunistic infections present more frequently in patients who are immunocompromised; especially those who are HIV positive. Most opportunistic infections are considered to be AIDS-defining illnesses; that is, they manifest in patients with CD4 counts <200 cells/μL. We report a case of a 47-year-old newly diagnosed HIV-infected man who developed pneumocystis jirovecii pneumonia despite a CD4 count of nearly 300 cells/μL; thus making PCP his first AIDS-defining event.

2. Case Presentation

A 47-year-old man from Nigeria with no past medical history presented to the emergency department with complaints of a non-productive cough and shortness of breath for 5 weeks. He had an emergency room visit 4 weeks ago at the onset of his symptoms, but was discharged with a diagnosis of community acquired pneumonia and prescribed treatment with amoxicillin and azithromycin. However, his symptoms, especially his shortness of breath, progressively worsened over time. An early chest radiograph taken 1 month prior with a posterior-anterior view is shown in Figure 1.

The patient describes his shortness of breath as occurring with exercise at first, but now he experiences breathlessness even at rest while watching television. Prior to his symptoms, he was able to run 1-2 miles per day. He mentions that his symptoms did not come on abruptly; rather began with a nagging dry cough that he first attributed to seasonal allergies. He has been intermittently taking an over the counter cough suppressant for the past month, but stated that it did not provide relief of his symptoms. He disclosed a fever of 38.3°C (101°F) earlier in the week which prompted him to take 2 acetaminophen tablets, but has not retaken his temperature since then. When asked about weight changes, he reported an unintentional weight loss of 9kg (20lbs) over the past 2 months.

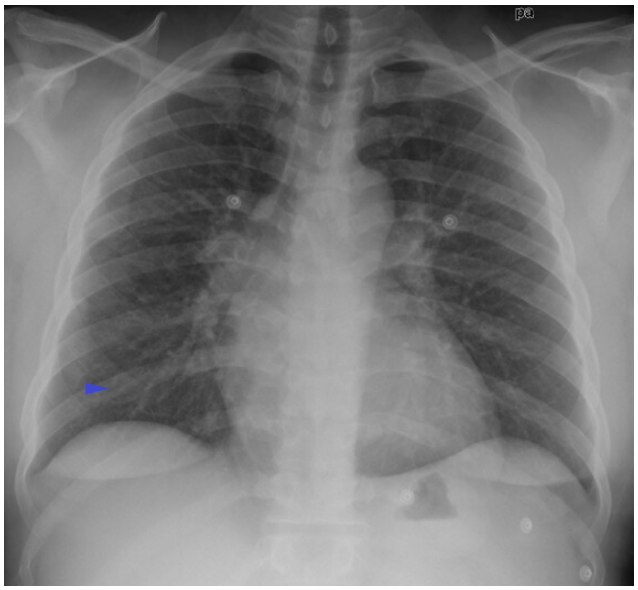

Figure 1. Chest radiograph with a posterior-anterior view. This image depicts very faint opacities in the right lower lung field (arrow), however, these changes are subtle

The patient follows with a primary care physician and did not have any past medical or surgical history. He takes a daily multivitamin and an over the counter cough suppressant as needed. He has seasonal allergies, but no allergies to medications. His parents are healthy, but his maternal uncle died at the age of 80 from lung cancer. He was born and raised in Nigeria and recently moved to the United States. He does not smoke cigarettes, drink alcohol, or use drugs. He works as a driver, is sexually active with a female partner, and uses occasional protection. He denies any night sweats, nausea, vomiting, dizziness, hemoptysis, chest pain, orthopnea, diarrhea, or dysuria.

Upon arrival to the emergency department, he was febrile to 100.4^{o}F, tachycardic to 106BPM, and hypoxic to 90% on room air. He was initiated on 2L nasal cannula, with an increase in oxygen saturation to 93%. On further examination, he appeared to be in mild distress with use of respiratory accessory muscles. Other significant exam findings included a tachycardic heart rate and right and left lower lobe crackles. A bedside point of care ultrasound was performed that approximated an ejection fraction at 55% without fluid overload. An ECG showed a tachycardic rate with a normal rhythm. A new chest x-ray was also performed in the emergency department and compared to his previous x-ray one month prior (Figure 2).

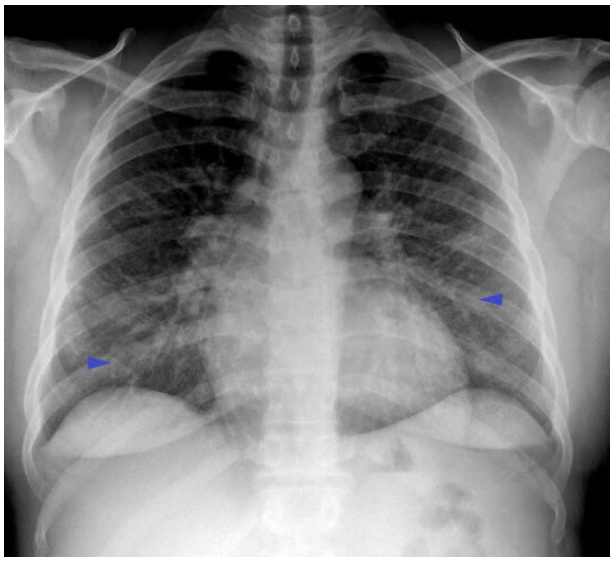

Figure 2. Chest radiograph with a posterior-anterior view. Similar to the previous chest x-ray taken 1 month prior, ill-defined hazy faint opacities can be visualized in the right lower lung (arrow). However, newly developed faint hazy opacities can now be seen in the left midlung (arrow), concerning for a possible multifocal pneumonia

Initial laboratory testing returned which was significant for WBC 10.08 K/μL with 62.3% neutrophils and an elevated LDH of 555 U/L. Arterial blood gas showed a pH of 7.43, PCO2 30mmHg, PO2 of 59mmHg, and calculated A-a gradient of 53.2mmHg. Other tests included a negative troponin, pro BNP of 5pg/mL, and negative rapid COVID-19 testing. A urine culture, HIV ag/ab screen, AFB sputum culture, and an interferon gamma release assay were pending.

The patient became hypoxic to the high 80s and he was placed on high-flow nasal cannula and admitted to the medical intensive care unit. He met 3/4 SIRS criteria and sepsis protocol was initiated with 30cc/kg/hr IV fluids and 2 sets of blood cultures were drawn before starting broad spectrum antibiotics. He was placed on airborne and contact precautions. Computed tomography angiography was performed to evaluate for a pulmonary embolism (Figure 3).

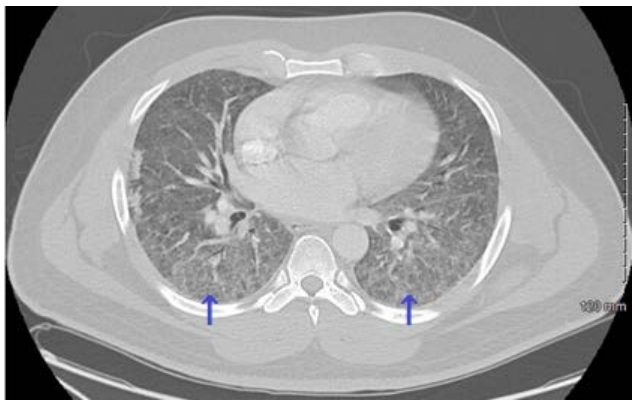

Figure 3. Contrast-enhanced computed tomography scan of the chest with coronal view. This image depicts diffuse groundglass parenchymal opacities which can be visualized peripherally (arrows)

At this time, the patient's interferon gamma release assay was negative, but he was found to be HIV positive. Infectious Disease was consulted and the patient was immediately started on highly active antiretroviral therapy (HAART). A viral load was 122,342 copies/mL and CD4 count was 293 cells/μL. Additional laboratory testing was obtained including fungal culture, aspergillus galactomannan and cryptococcal serum antigen testing, legionella and histoplasma urine antigen testing, toxoplasmosis serology, (1-3)-beta-D-Glucan (BDG) testing, and a full STD panel; all of which were negative except for serum BDG.

A bronchoalveolar lavage (BAL) was performed to evaluate for PCP. The BAL specimen was sent for PCP PCR, PCP smear, AFB culture, MTB PCR, cell count, cytology, and viral, fungal, and bacterial culture. The patient was found to be PCP positive and was started on trimethoprim/sulfamethoxazole (TMP/SMX) and prednisone. A post-bronchoscopic portable chest x-ray was taken (Figure 4).

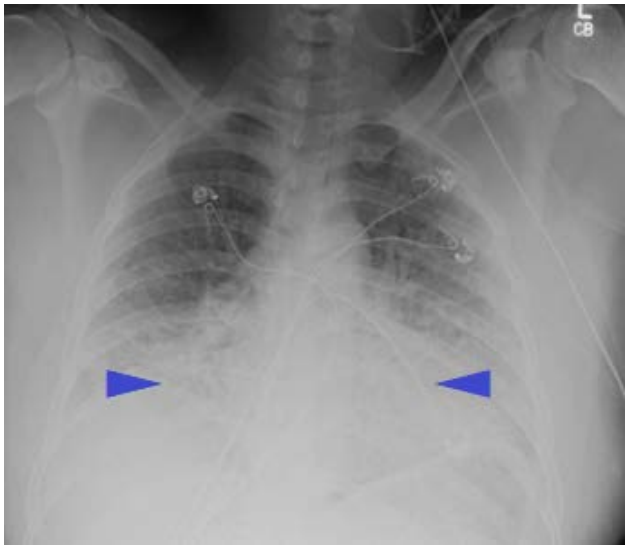

Figure 4. A portable chest radiograph with anterior-posterior view taken post-bronchoscopy revealed diffuse bilateral airspace opacities and a worsening pneumonia (arrows)

Fortunately, over the next week, the patient's respiratory symptoms and oxygen requirements gradually improved with treatment. He remained hemodynamically stable and was downgraded to the medical floor. He was ultimately discharged on TMP/SMX, Prednisone, and HAART therapy with appropriate outpatient follow-up. Upon follow-up visit months later, his viral load had significantly decreased and CD4 count had increased to 500 cells/μL.

3. Discussion

Pneumocystis Jirovecii Pneumonia (PCP) is an atypical opportunistic infection that causes life-threatening pulmonary complications in HIV-positive patients; especially those with AIDS. While it usually occurs when individual CD4 counts are <200 cells/μL, it can rarely occur in those with higher counts. The use of highly active antiretroviral therapy (HAART) has led to a significant decrease in the number of PCP infected individuals in the United States; however, PCP is still a prevalent AIDS-defining illness in many developing countries [1].

The pathophysiology of PCP, likely transmitted through aerosolized particles, involves interaction with extracellular proteins which help facilitate attachment to type I alveolar pneumocytes [2]. This attachment gives the infectious agent the ability to transition from its small non-rigid trophic form with a single haploid nucleus to a more rigid larger cystic form with up to eight separate intracystic bodies [3]. Ultimately, cyst rupture and proliferation of cells lead to a foamy, eosinophilic exudate that fills alveolar spaces.

Fever is often the first symptom of PCP, followed by a nonproductive cough and dyspnea over the course of weeks. Physical examination findings may consist of hypoxemia, tachypnea, respiratory accessory muscle use, and scattered rales or rhonchi; but sometimes a lung examination is normal. Helpful laboratory testing includes obtaining serum LDH and BDG. Serum LDH levels may correspond to the degree of inflammation and lung injury, while a negative BDG has been shown to be helpful in ruling out PCP cases in HIV-infected individuals [4]. Classic radiological features involve extensive ground glass attenuation visualized on high-resolution computed tomography [5].

A severe PCP infection can progress to acute respiratory distress syndrome; which usually requires mechanical ventilation. The diagnosis of ARDS can be made based on the following criteria: acute onset, bilateral lung infiltrates on radiography of noncardiac origin, and a PaO2/FiO2 ratio < 300mmHg [6]. According to the Berlin definition, ARDS disease severity can be classified into three subtypes by using an arterial pressure of oxygen (PaO2) to fraction of inspired oxygen (FiO2) ratio (Figure 5).

ARDS Subtype	PaO2/FiO2 Ratio
Mild	200-300 mmHg on PEEP ≥ 5cmH2O
Moderate	100-200 mmHg on PEEP ≥ 5cmH2O
Severe	≤100 mmHg on PEEP ≥ 5cmH2O

Figure 5. Table displaying ARDS disease severity based on PaO2/FiO2 ratio [7]. While high-flow nasal cannula and noninvasive ventilation may be used, a large number of patients will require intubation [8]. If the patient is mechanically ventilated, the goal of PaO2 is 55-80mmHg.

A bronchoalveolar lavage is the diagnostic modality preferred over an open lung biopsy, as sensitivity is generally estimated to be greater than 80% [9]. Common stains used for identification of PCP are GMS and toluidine blue, Giesma, and immunofluorescent techniques.

First-line treatment of PCP is trimethoprim-sulfamethoxazole (TMP-SMX) for a duration of 21 days in patients with HIV; no matter the CD4 count. In patients who experience significant adverse reactions, pentamidine and atovaquone may be considered. Adjunctive corticosteroid administration has been shown to decrease mortality in those with an arterial oxygen partial pressure < 70 mmHg or alveolar - arterial gradient > 35 mmHg on room air [10].

4. Conclusion

Acquired Immunodeficiency Syndrome (AIDS), which is the last stage of HIV infection, occurs when the number of CD4 cells falls below 200 cells/μL or if a diagnosis of an opportunistic infection such as PCP is made. While it is rare for pneumocystosis to develop in those with CD4-lymphocyte counts greater than 200 cells/μL, it is still a possibility. Clinical suspicion for the diagnosis of PCP should be raised when interacting with patients presenting with pneumonia-like symptoms who are from an exceptionally large HIV-infected population.

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Statement of Competing Interests

The authors of this case report have no competing interests.

Compliance with Ethical Standards and Informed Consent

Informed consent was obtained for the publication of this case report.

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