

Case Report

Pneumocystis Jirovecii Pneumonia and Pulmonary Thromboembolism as Primary Manifestations of Human Immunodeficiency Virus Infection: A Case Report and Clinical Course

Monroy-Meneses Carlos Eduardo^{1*}, Pérez-Millán Karla Janeth², Flores-Hernandez Daniela³, Ruiz-Gonzalez Samantha Lizeth³, Carmona-Tapia Daniela Alejandra³

¹Department of Internal Medicine, General Hospital Toluca, Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado, México

²Department of Diagnostic and Therapeutic Imaging, Centro Médico Lic. Arturo Montiel Rojas, Instituto de Seguridad Social del Estado de México y Municipios, México

³Department of Internal Medicine, General Hospital Toluca, Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado, México

***Corresponding Author:** Monroy-Meneses Carlos Eduardo

Department of Internal Medicine, General Hospital Toluca, Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado, México

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Abstract: Opportunistic infections such as *Pneumocystis jirovecii* pneumonia (PJP) and thromboembolic complications are frequent and well-known manifestations in patients with advanced human immunodeficiency virus (HIV) infection. Their simultaneous presentation as the initial HIV diagnosis in a previously healthy patient is rare and represents a diagnostic and therapeutic challenge due to its potentially fatal nature.

Keywords: HIV, *Pneumocystis Jirovecii*, Pulmonary Thromboembolism, Opportunistic Pneumonia, Nosocomial Pneumonia, Septic Shock.

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INTRODUCTION

Human immunodeficiency virus (HIV) infection continues to be one of the main causes of acquired immunosuppression worldwide. When not diagnosed in time, the natural progression of the virus can lead to acquired immunodeficiency syndrome (AIDS), characterized by opportunistic infections, neoplasms, and metabolic or cardiovascular complications [1]. Among the most common opportunistic infections is *Pneumocystis jirovecii* pneumonia (PJP), which is a major cause of acute respiratory failure in patients with CD4 counts <200 cells/mm³ [2]. New diagnostic tools such as PCR on sputum or BAL and BDG have improved early detection [3]. Likewise, HIV-infected patients are at higher risk of thromboembolic events, due to chronic inflammation, endothelial dysfunction, and co-infection with other pathogens [4]. Mortality in PJP with

complications (e.g., thromboembolism, superinfection) can reach 30–50% [5]. The coexistence of PJP and pulmonary thromboembolism is rare and may delay diagnosis and complicate treatment [6].

This report describes a case of simultaneous presentation of PJP and pulmonary thromboembolism as primary manifestations of a previously undiagnosed HIV infection.

CASE PRESENTATION

A 36-year-old man presented to the emergency department. He was a teacher with a 2-month history of unintentional weight loss of 8 kg, generalized anxiety disorder, and insomnia without medical management.

He first presented to emergency room (ER) with insomnia and anxiety and was started on clonazepam and sertraline with outpatient follow-up. He returned to the

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ER due to added asthenia, adynamia, and fever of 38°C. He was started on amoxicillin/clavulanic acid and discharged home. Fever persisted, reaching 39°C, with progressive dyspnea on moderate exertion, prompting another ER visit. On admission with blood pressure: 135/75 mmHg, heart rate: 130 bpm, respiratory rate: 30 bpm, O₂ saturation: 65%. On physical exam: signs of respiratory distress, decreased chest expansion, bilateral basal coarse crackles on auscultation, requiring supplemental oxygen via simple face mask at 7 L/min. Chest X-ray (Image 1): bilateral reticular thickening.

CBC: thrombocytopenia (67,000), lymphopenia (400), D-dimer 74,829. ABG: pH 7.51, PCO₂ 20, PO₂ 60, FiO₂ 28%, Pa/Fi 214.

Internal medicine admitted the patient. Due to suspected pulmonary embolism, contrast-enhanced chest CT revealed a filling defect in the right lower lobar artery and pulmonary infarction in the superior and posterior basal segments (Image 2).

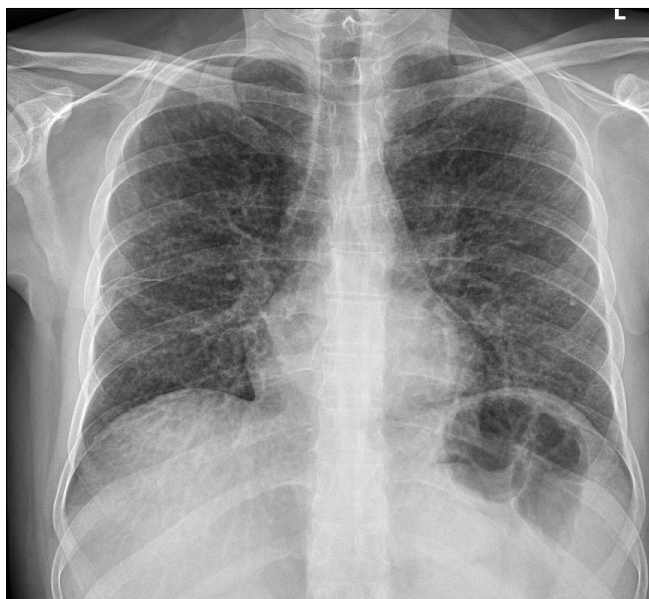


Image 1: Posteroanterior chest radiograph: bilateral perihilar predominant reticular thickening of the pulmonary interstitium



Image 2: Pulmonary CT angiography, axial cut and mediastinal window: after contrast injection, a filling defect is observed in the right lower lobar artery; triangular juxtaplural hyperattenuation in the ipsilateral superior and posterior basal segments is consistent with pulmonary infarction

Based on CT findings, an HIV serologic test was ordered and returned reactive. Treatment was started with trimethoprim-sulfamethoxazole, methylprednisolone, and enoxaparin. A western blot test confirmed HIV positivity.

Symptom resolution and decreased oxygen requirement were achieved after 7 days of treatment. Discharge was considered, but clinical deterioration

occurred with increased oxygen demand. Follow-up CT showed posterior basal consolidation (Image 3).



Image 3: Axial chest CT, lung window: generalized ground-glass opacities with superimposed interlobular septal thickening (“crazy paving” pattern) and peribronchial interstitial thickening. Right posterior basal consolidation and bilateral basal linear atelectasis observed

Nosocomial bacterial superinfection was diagnosed. Treatment was started with carbapenem for hospital-acquired pneumonia. After 6 days of antibiotics, the patient improved and oxygen requirements decreased. He was discharged. Thirteen days later, CD4 count returned: 12 cells/mm³: Viral load: 3 million copies.

After completing 21 days of TMP-SMX, ART with bictegravir/emtricitabine/tenofovir (50/200/25 mg) was initiated. Three days later, the patient presented with

fever of 39.5°C, dyspnea at rest, blood pressure: 94/56 mmHg, heart rate: 126 bpm, respiratory rate: 26 bpm, O₂ saturation: 60%. Chest CT revealed generalized ground-glass opacities with interlobular septal thickening consistent with a “crazy paving” pattern (Image 4).

The patient deteriorated, developed worsening pneumonia, and died of septic shock of pulmonary origin.

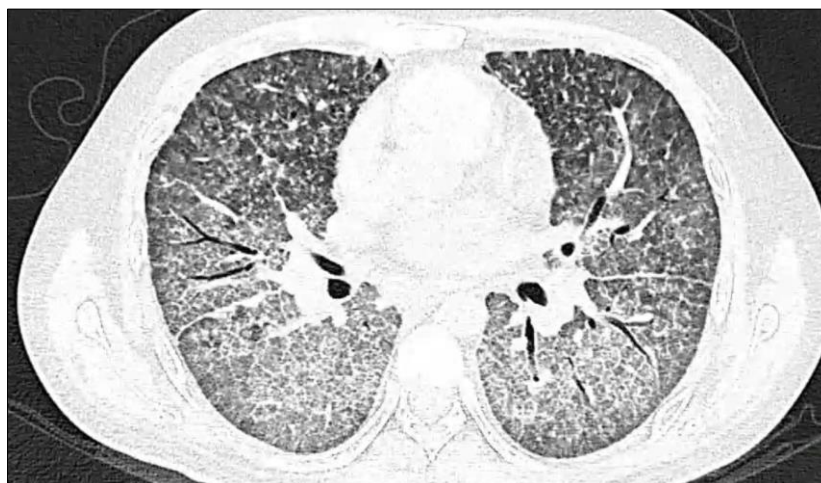


Image 4: Axial chest CT, lung window: bilateral ground-glass opacities with superimposed interlobular septal thickening (“crazy paving”) and peribronchial thickening

DISCUSSION

Pneumocystis jirovecii pneumonia is a leading cause of hospital admission in patients with advanced HIV. Clinically, it presents with progressive dyspnea, fever, and dry cough. Radiologically, a diffuse interstitial pattern or ground-glass opacities may be seen [7]. This patient’s clinical picture (hypoxemia, fever,

lymphopenia, elevated D-dimer) was characteristic of PJP and pulmonary embolism. Confirmation was achieved by chest CT and HIV serology, as recommended in current guidelines [3]. Early initiation of TMP-SMX and steroids is the treatment of choice [8]. A ≥ 21 -day course of TMP-SMX with corticosteroids in severe PJP is recommended by current guidelines [9, 10].

Enoxaparin is appropriate for coexisting thromboembolism [5].

Pulmonary thromboembolism (PTE) is more prevalent in HIV-positive individuals than in the general population. Its incidence increases as CD4 count decreases and viral load increases [11]. In this case, the elevated D-dimer (>70,000), tachycardia, severe hypoxemia, and CT findings were decisive for diagnosis.

Initiation of antiretroviral therapy (ART) in patients with severe opportunistic infections must be carefully timed to avoid immune reconstitution inflammatory syndrome (IRIS). In PJP, however, early ART initiation—usually within the first 14 days—is recommended [12].

This patient developed complications including nosocomial bacterial superinfection, likely due to profound immunosuppression and prolonged hospitalization. Despite multidisciplinary treatment, the patient progressed to septic shock and died. The reappearance of symptoms after ART initiation suggests possible IRIS or nosocomial pneumonia, both associated with poor prognosis [6]. Mortality remains high in advanced PJP, especially in cases with CD4 <50 cells/mm³ and acute complications [5].

CONCLUSIONS

Pneumocystis jirovecii pneumonia and pulmonary thromboembolism may be initial manifestations of HIV infection. Their simultaneous presentation is rare and requires a high index of suspicion.

Early recognition and prompt initiation of ART and antimicrobial therapy are essential to improve prognosis. The coexistence of PJP and pulmonary embolism should raise concern for underlying HIV infection.

Severe immunosuppression (CD4 <50 cells/mm³) is associated with poor prognosis despite adequate treatment. HIV testing should be considered in young patients with atypical respiratory infections, severe hypoxemia, and hematologic abnormalities without apparent cause.

ART initiation must be carefully timed alongside opportunistic infection management to reduce IRIS risk.

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