CASE REPORT

Pulmonary hyalinizing granuloma in HIV/AIDS

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A 55-year-old man who was recently diagnosed with HIV/AIDS developed multiple bilateral pulmonary nodules after starting highly active antiretroviral therapy. Workup confirmed the diagnosis of pulmonary hyalinizing granuloma. This is the first described case of pulmonary hyalinizing granuloma in HIV/AIDS, and may represent a rare form of immune reconstitution inflammatory syndrome.

Key Words: HIV/AIDS; Immune reconstitution inflammatory syndrome; Multiple pulmonary nodules; Pulmonary hyalinizing granuloma

Pulmonary hyalinizing granuloma (PHG) is a rare condition first described in 1977, which usually manifests as multiple bilateral pulmonary nodules of lamellar hyaline collagen deposits (1). It often presents with mild pleuritic chest pain, cough and dyspnea, or can be asymptomatic, detected incidentally on imaging. PHG presents as single or multiple nodules, and radiographically can resemble primary or secondary malignant lesions. Fewer than 100 cases have been reported in the literature (1-3); there is no effective treatment for secondary malignant lesions. Fewer than 100 cases have been reported in the literature (1-3); there is no effective treatment for secondary malignant lesions.

A 55-year-old man who was recently diagnosed with HIV/AIDS was seen in July 2000, complaining of three months of fatigue and mild exertional dyspnea. He had been diagnosed with HIV/AIDS following a febrile illness in April 2000. At diagnosis, his CD4 count was less than 100 cells/mm3 and his viral load was greater than 500,000 copies/mL. He had since finished treatment for both pneumocystis carinii pneumonia (PCP) and Helicobacter cinaedi infection, with no history of travel or occupational exposures. He was taking trimethoprim-sulfamethoxazole and azithromycin for prophylaxis of PCP and disseminated Mycobacterium avium complex infection, respectively.

Due to the patient’s ongoing mild shortness of breath and fatigue, a repeat chest x-ray was performed one month later, showing the persistence of the right upper lobe opacity. It appeared spiculated and measured approximately 1.5 cm in diameter. At this time, the patient’s viral load had decreased to 2681 copies/mL and his CD4 count was 396 cells/mm3.

In October 2000, the patient’s radiography (Figure 1) and computed tomography (Figure 2) of the chest revealed multiple, bilateral, smooth, well-defined pulmonary nodules affecting all lobes. The larger of the nodules had a perihilar distribution and no history of travel or occupational exposures. He was taking trimethoprim-sulfamethoxazole and azithromycin for prophylaxis of PCP and disseminated Mycobacterium avium complex infection, respectively.

His physical examination was normal; he was not hypoxic or febrile, and there was no clubbing or pulmonary findings. Pulmonary function tests showed mild obstructive lung disease. Chest radiography revealed a single, asymmetric opacity in the right upper lobe adjacent to the right hilum that was not present in April 2000, at the time of diagnosis of HIV/AIDS. Radiological interpretation suggested that it was either a composite shadow, a focal atelectasis or a focal inflammatory infiltrate. The following week, a repeat chest x-ray showed that the opacity was less prominent, and he received no treatment.

It was thought that his subjective feeling of dyspnea was related to his recent PCP infection. In July 2000, he was started on highly active antiretroviral therapy (HAART), consisting of zidovudine, lamivudine and ritonavir-boosted indinavir.

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In October 2000, the patient’s radiography (Figure 1) and computed tomography (Figure 2) of the chest revealed multiple, bilateral, smooth, well-defined pulmonary nodules affecting all lobes. The larger of the nodules had a perihilar distribution and there were also multiple subpleural nodules. There were no hilar adenopathy or pleural effusions. Clinically, the patient’s respiratory symptoms had improved; his viral load had dropped to 214 copies/mL and his CD4 count was 484 cells/mm3. He declined further investigations.

Repeat chest radiography in February and March 2001 showed marked progression in the size and number of the bilateral pulmonary nodules. The patient denied worsening of his
dyspnea; his viral load was less than 50 copies/mL and his CD4 count was 612 cells/mm$^3$ on HAART. Bronchial wash and transbronchial biopsy of a left-sided nodule performed in March 2001 were negative for infectious organisms, granuloma and malignancy. In May 2001, a fine needle aspirate biopsy from a lingular nodule showed an aspirate of scant cellularity consisting of red blood cells, benign bronchial cells, pulmonary macrophages and multinucleated histiocytes. A repeat fine needle aspirate biopsy of a right upper lobe nodule in August 2001 was similar to the first, but the material was too scant to perform special stains to rule out infection. In August 2001, a core biopsy of the right upper lobe lesion showed necrotic tissue partially surrounded by chronic inflammatory cells. Special stains were negative for acid-fast bacilli and fungi. Congo red stain was negative for amyloid.

Despite the negative fungal stains, on review, the pathologist thought that the nodules could represent a fungal infection, given the patient’s profound immunosuppression before starting HAART. However, an empirical three-month course of itraconazole did not improve the patient’s mild respiratory symptoms or alter the radiographic appearance of the nodules. It was thought that the original diagnosis of PHG was correct. A trial of corticosteroids was considered but not initiated given the patient’s minimal symptoms. Follow-up chest radiography five years later was unchanged.

**DISCUSSION**

PHG has no sex, race or age predilection, and usually presents with multiple bilateral pulmonary nodules (1). Patients can be asymptomatic, or complain of mild respiratory symptoms (cough, dyspnea, hemoptysis and pleuritic chest pain) or other nonspecific symptoms (fatigue, fever and weight loss). If present, symptoms are usually nonprogressive. The nodules are slowly progressive in size and number, and there is no specific treatment. Single large nodules are sometimes amenable to surgical resection. Radiographically, the differential diagnosis includes many causes of multiple bilateral pulmonary nodules, including malignancy, granulomatous diseases such as Wegener’s granulomatosis or sarcoidosis, amyloidosis, rheumatoid nodules and inflammatory pseudotumour, as well as infectious causes such as tuberculosis (TB) or histoplasmosis (1,2,4,5).

Pathology of the lesions in PHG shows concentric, whorled, lamellar hyaline deposits surrounded by plasma cells and lymphocytic infiltrate; early nodules show a predominance of the cellular components and older lesions show more lamellar collagenous bands (1,2). The presence of thick hyalinized collagen bands arranged in whorls, parallel arrays or in a vague storiform pattern is characteristic of PHG. Unlike...
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CONCLUSION

PHG is a rare, benign, immune-mediated lung disease with no specific treatment. To our knowledge, this is the first described case of PHG in a patient with HIV/AIDS. Because the diagnosis of PHG was temporally preceded by the initiation of HAART, it is possible that it could have been due to an immune reconstitution phenomenon. It is well known that patients with HIV/AIDS are at risk for usual common pulmonary infections, as well as TB, PCP and other fungal pneumonias that can present as multiple pulmonary nodules. However, PHG should also be considered in the differential diagnosis of multiple pulmonary nodules in the setting of treated HIV/AIDS.

REFERENCES

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