There are protean manifestations of human immunodeficiency virus (HIV) infection involving various organ systems. Rheumatologic manifestations of HIV infection include arthritis, myositis, vasculitis and the sicca complex-like syndrome. Previous reports found that among HIV-infected patients, 1.7% had Reiter’s syndrome, 1.7% had psoriatic arthritis, and 11.1% had various forms of oligoarticular/monoarticular or polyarticular arthritis [1-4]. We describe an HIV-infected patient with the initial presentation of interstitial lung disease (ILD) and lymphocytic infiltration in the minor salivary glands who developed *Pneumocystis carinii* pneumonia (PCP) after 3 courses of monthly methylprednisolone pulse therapy for ILD. Both enzyme immunoassay and Western blot analysis confirmed the HIV infection. After antibiotic treatment, the pneumonia gradually resolved. Autoimmune manifestations such as ILD and xerostomia may be initial presentations of HIV infection. Immunosuppressive drugs must be used cautiously in these patients. Screening for HIV is mandatory in the differential diagnosis of patients with ILD or xerostomia.

**Key words:** HIV, interstitial lung disease, lymphocytosis, xerostomia
Interstitial lung disease and xerostomia in HIV

IgA 354 mg/dL (normal, 82-453 mg/dL) and IgM 611 mg/dL (normal, 46-304 mg/dL). Because the ILD was suspected to have originated from autoimmune processes, monthly methylprednisolone (1000 mg once daily for 3 days) pulse therapy and daily azathioprine 50 mg were prescribed.

Dyspnea improved slightly during the first 2 courses of pulse therapy. However, on the 17th day after the third course of methylprednisolone pulse therapy, he developed fever, chills and dyspnea. Arterial blood gas revealed moderate hypoxemia (pH 7.595, partial pressure of O$_2$ 50.9 mmHg, partial pressure of CO$_2$ 23.0 mm Hg, HCO$_3^-$ 21.8 mmol/L, with fraction of inspired O$_2$ 40%). CRP was 15.7 mg/dL. Chest radiograph showed pulmonary infiltration over bilateral lungs, which was more severe on the right side (Fig. 3). Chest CT scan revealed pneumonic processes with multiple abscess formation in the right middle and lower lobes, and alveolitis in the left lung (Fig. 4). Fever and dyspnea persisted despite intravenous cefazolin plus gentamicin administration for 3 days followed by another 3 days of cefuroxime plus gentamicin treatment. Gram-positive cocci were found in sputum. In view of
the poor treatment effect, persistent hypoxemia and bilateral lung infiltration, staphylococcal, mycoplasma or PCP were suspected. Teicoplanin, ciprofloxacin and sulfamethoxazole-trimethoprim were given simultaneously. The ensuing laboratory investigations showed hemoglobin 9.5g/dL, white blood cell count 6100/µL (82% neutrophils, 9% lymphocytes, 10% monocytes), and platelet count 291,000/µL. Lymphocyte subpopulation analysis showed CD4 cells 4% and CD8 cells 85%. Both enzyme immunoassay and Western blot for HIV were positive. HIV viral loading test showed a viral copy number of 653,000 copies/mL. No pathogens were cultured from sputum and pleural effusion. Acid fast stain of the sputum smear and polymerase chain reaction for Mycobacteria tuberculosis from pleural effusion were negative. Serum IgG anti-cytomegalovirus antibody was positive but IgM anti-cytomegalovirus antibody was negative. Venereal disease research laboratory test, Treponema pallidum hemagglutination and serum cryptoccocal antigen were negative. Fever subsided and pneumonia resolved gradually after antibiotic therapy. Highly-active antiretroviral therapy was then started. Three months later, HIV viral load was undetectable and CD4 count was 248/µL.

Discussion

Diffuse infiltrative lymphocytosis syndrome (DILS) is characterized primarily by parotid gland enlargement, peripheral CD8 lymphocytosis, sicca syndrome and pulmonary involvement in HIV-infected subjects [5]. The prevalence of DILS is 3% in HIV-positive outpatients [6]. These patients have certain features that are unusual for the classic Sjögren’s syndrome. They are predominantly men and none have significant levels of autoantibodies. Salivary gland biopsy specimens show diffuse lymphocytic infiltration of the glandular parenchyma without epithelial lesions. On the contrary, in classic Sjögren’s syndrome, marked epithelial lesions are usually present [6,7].

Our patient was a homosexual male with progressive ILD and lymphocyte infiltration in salivary glands, which mimicked autoimmune disease. No autoantibodies were detected. He had dry mouth but no obvious eye dryness or parotid gland enlargement. In this clinical context, others etiologies of Sjögren’s-like syndrome should be considered. Malignant lymphoma, hepatitis C virus infection, and HIV infection have each been reported to be associated with Sjögren’s syndrome [4,8,9].

Initially, our patient was thought to have connective tissue disease and progressive ILD. Due to progressive air hunger with severe reduction in gas exchange capacity, and the findings of chronic inflammation and fibrosis on transbronchial biopsy, monthly pulse glucocorticoid therapy was scheduled to ameliorate the pulmonary destruction [10].

Consolidation and abscesses in the lower and middle lobes of the right lung and alveolitis in the left lung developed, but sputum cultures did not yield any pathogens. He did not undergo bronchoscopy examination due to dyspnea with moderate hypoxemia. In view of the manifestations on CT scan and plain chest films, as well as the clinical features, PCP was considered to be the main cause of pulmonary inflammation. Patients with malignancies, immunodeficiency, systemic glucocorticoid therapy with or without immunosuppressive drug treatment and connective tissue diseases are at risk of developing PCP [11-13]. The risk factors for PCP in our patient were systemic glucocorticoid therapy and HIV infection.

Pulmonary disorders, particularly respiratory infections, are an important cause of morbidity and mortality in HIV-infected individuals [14]. PCP remains the most common cause of life-threatening pulmonary infection in HIV-positive patients [15]. The classic radiographic presentation of PCP is a bilateral perihilar or diffuse symmetric interstitial pattern, which may be finely granular, reticular or ground glass in appearance [16,17]. The classic high-resolution CT finding for PCP is extensive ground glass attenuation, which corresponds to the presence of intra-alveolar exudates, consisting of fluid, organisms and debris [15].

In summary, ILD and xerostomia may be the first manifestations of HIV infection. Clinicians need to consider the possibility of concomitant HIV infection in patients from high-risk groups. Immunosuppressive drugs must be used cautiously in patients with autoimmune manifestations but without sufficient evidence to support the diagnosis. These patients may turn out to be HIV-infected individuals.

References


