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Case Report

Interstitial lung disease – A rare diagnosis in HIV

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ARTICLE INFO

Article history:
Received 15 August 2015
Accepted 7 October 2015
Available online 6 November 2015

Keywords:
HIV
Interstitial lung disease
Pulmonary fibrosis
Idiopathic lung disease
Antiretroviral therapy

ABSTRACT

A case report of a 30-year-old HIV patient presented with features of interstitial lung disease usually misdiagnosed as tuberculosis or PCP and wrongly treated. HRCT of the lung suggestive of interstitial lung disease with no evidence of tumor or *Pneumocystis jiroveci*. The patient responded to steroids and was continued on antiretroviral treatment.

1. Introduction

Interstitial lung diseases have been reported in HIV-infected patients,[1–4] including lymphocytic interstitial pneumonitis (LIP), nonspecific interstitial pneumonitis (NSIP), organizing pneumonia, pulmonary alveolar proteinosis, and hypersensitivity pneumonitis.

Interstitial lung disease is still a poorly understood condition in patients with Human Immunodeficiency Virus (HIV) infection. The presentation may be like *Pneumocystis jiroveci* pneumonia or Koch's chest. The diagnosis is by exclusion.

2. Case report

A 30-year-old male, with known case of HIV on antiretroviral therapy (ART) since July 2013, was admitted with history of anorexia and dry cough with dyspnea and low-grade fever. X-ray chest showed reticulonodular opacities in bilateral lung fields with apicobasilar gradient and small cystic lucencies (honey combing) (Fig. 1), and therefore, empirically, ATT was started. However, patient showed no improvement with ATT. He was a nonsmoker, non-alcoholic, and he denied any past history of blood transfusion or sexual promiscuity.

On examination, the patient had low-grade fever, anicteric, normotensive, and no lymphadenopathy. Auscultation revealed bilateral occasional rhonchi with fine basal crepts in both lung fields, and he had mild hepatosplenomegaly.

Investigations revealed Hb 11.6 gm%, peripheral blood film was suggestive of normocytic, normochromic anemia with mild anisocytosis, TLC 8700/mm^3, polymorphs 71%, lymphocytes 17%, eosinophils 1.4%, and monocytes 10.7%; platelet count was 3.34 lacs/mm^3 and ESR was 71 mm/1st hour. Blood sugar, renal profile, lipid profile, and hepatic functions were normal except for serum proteins of 7.1 gm% and serum

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http://dx.doi.org/10.1016/j.injms.2015.10.006
0976-2884/

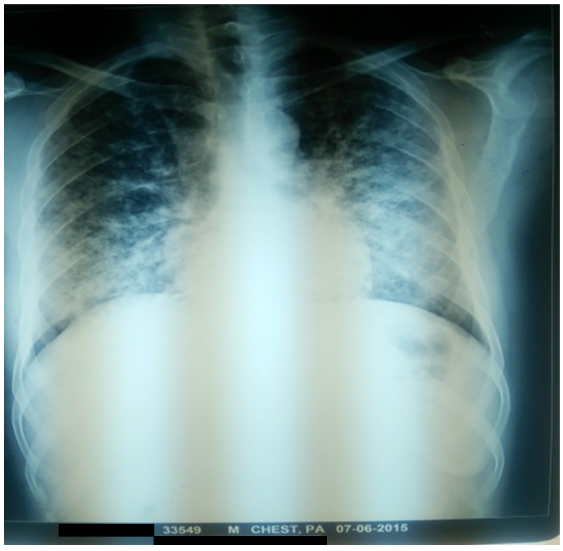

Fig. 1 – Original X-ray PA chest reticulonodular opacities in bilateral lung fields with apicobasilar gradient and small cystic lucencies (honey combing).

albumin of 2.9 gm%. HIV I was positive (by three different antigens) and absolute CD4 count was 268 cells/mm^3. Sputum for AFB was negative.

CT chest revealed marked intralobular, interlobular, and peribronchovascular interstitial thickening predominantly in posterior basal segments and in subpleural location suggestive of ILD (Fig. 2). Pulmonary function tests (PFT) showed a restrictive ventilatory defect. Arterial blood gas analysis showed mild hypoxemia (O_2 saturation of 95.9%).

Bronchoscopic examination was normal, bronchoscopic alveolar lavage was negative for *Pneumocystis jiroveci* and AFB, and TB-PCR was negative.

A diagnosis of HIV stage (WHO clinical stage II) with nonspecific interstitial pneumonia was made. The patient was started on prednisolone and bronchodilators, and antiretroviral treatment was continued. ATT was withdrawn.

The patient improved over the next few weeks. A chest X-ray and PFT performed after 1 month showed improvement. Steroids were tapered off and maintained at low dose. A repeat CD4 count after 2 months was 318 cells/mm^3.

3. Discussion

Interstitial lung diseases are poorly defined conditions in immunocompromised patients, where it is diagnosed after exclusion of opportunistic infections and neoplasms.[5] It occurs in HIV-positive patients when the CD4 and total lymphocyte counts are still preserved. The cause is unknown, but various agents have been suggested, including the HIV virus itself.[5] In adult AIDS patient with pulmonary symptoms, the presenting symptoms are nonspecific for most cases of interstitial lung disease and can include dyspnea, nonproductive cough, fatigue, and fever. Findings on lung examination may be normal or crackles may be noted. About 50% patients may present with weight loss. Constitutional

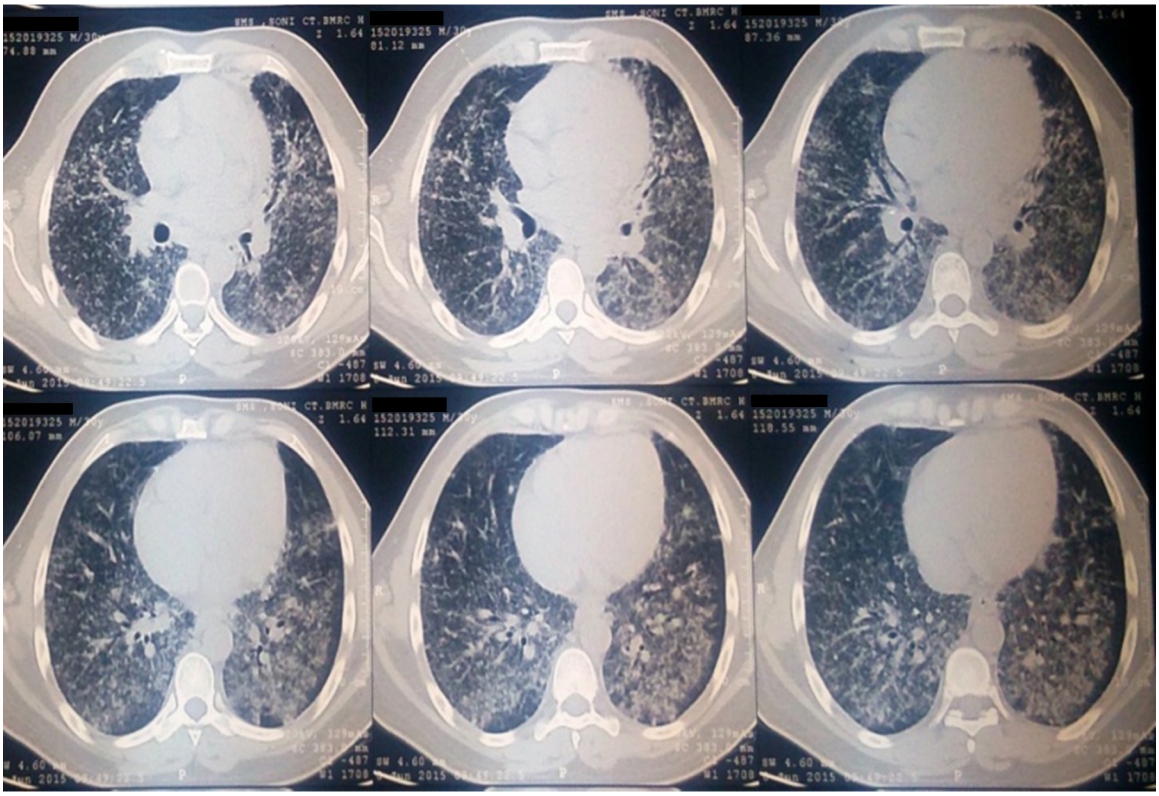

Fig. 2 – HRCT chest showing marked intralobular, interlobular, and peribronchovascular interstitial thickening.

symptoms are less common.[5] Crackles are initially basal but may be widespread and inspiratory rhonchi may be present.[6] Radiological features are nonspecific and like any interstitial lung disease.[3] HRCT and Pulmonary function tests often reveal a restrictive ventilatory defect and a decreased diffusing capacity strengthens the suspicion of ILD. Lung biopsy may show a mainly interstitial inflammation or fibrosis or a combination of the two and is negative for infection or tumor.[5] The histological features do not fit into other forms of ILD, specifically, usual interstitial pneumonia (UIP) and desquamative interstitial pneumonia (DIP).[7] In the fibrosing pattern of NSIP, there is a temporal uniformity, contrasting with the heterogeneity of UIP, in which dense collagen is associated with scattered fibroblastic foci.[8] In DIP, there is a uniform accumulation of macrophages within the alveolar spaces. The alveolar septa are mildly thickened by collagen deposition and by small number of lymphocytes, plasma cells, histiocytes, and eosinophils.[9] Our patient showed a dramatic response to steroids clinically, radiologically, and physiologically and he is symptom free at two months of follow-up.

Conflicts of interest

The author has none to declare.

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