Case Report

Non-specific Interstitial Pneumonia in an HIV Positive Patient

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Abstract

A 56 year old male presented with features of interstitial lung disease and was detected HIV positive. Histopathology of the lung suggested non-specific interstitial pneumonia with no evidence of tumour or *Pneumocystis carinii*. The patient responded to steroids and was started on anti-retroviral treatment. ©

Introduction

Non-specific interstitial pneumonia (NSIP) is still a poorly understood condition in patients with Human Immuno-deficiency Virus (HIV). The presentation may be like *Pneumocystis carinii* pneumonia (PCP). The diagnosis is by exclusion and by histopathological features that are unlike the classical interstitial lung disease (ILD). Its importance lies in the fact that the prognosis is better compared to other forms of ILD.

Case Report

A 56 year old male was admitted with a 1 ½ years history of anorexia and about 18-20 kg weight loss. Five months prior to hospitalization he started having dry cough with dyspnoea and low grade fever. He was then started empirically on antitubercular treatment (ATT). The patient showed no improvement with ATT. He was not a known diabetic or hypertensive. He was a non smoker and a reformed alcoholic for the last one year and he denied any past history of blood transfusion or sexual promiscuity.

On examination the patient was afebrile, pale, anicteric, normotensive and had small discrete lymphnodes in the right supraclavicular, bilateral axillary and inguinal regions. Chest showed bilateral occasional rhonchi with fine basal crepts and he had hepatosplenomegaly.

Investigations revealed Hb of 8.6gm%, peripheral smear was normocytic, normochromic, TLC 6500 / mm^3, polymorphs 56%, lymphocytes 33%, eosinophils 6% and monocytes 5%, platelet count was 1.56 lacs / mm^3 and ESR was 134 mm / 1st hour. Blood sugar, renal and hepatic functions were normal except for serum proteins of 9.8gm% and serum albumin of 2.9gm%. HIV I was positive (by three different antigens) and absolute CD4 count was 239 cells / mm^3. X-ray chest was normal. Sputum for AFB was negative. CT scan of chest revealed diffuse ground glass attenuation with relative sparing of the right middle lobe with subtle peripheral reticulonodular infiltrates (Fig. 1). Pulmonary function tests (PFT) showed a restrictive ventilatory defect. Arterial blood gas analysis showed mild hypoxemia (O_2 Saturation of 88.7%).

Bronchoscopic examination was normal, lavage showed predominant lymphocytes (55%), 30% neutrophils and 15% macrophages. It was negative for Pneumocystic carinii and AFB. A transbronchial biopsy showed reduced alveolar spaces with thickened septae and dense uniform chronic inflammatory cells comprising chiefly of lymphocytes and plasma cells in the interstitium with areas of fibrosis and alveolar pneumocytic hyperplasia. There was no evidence of granulomas, Pneumocystis carinii, inclusion bodies or tumour (Fig. 2). Right inguinal lymphnode biopsy showed reactive changes on histopathology with no granulomas or AFB. A diagnosis of HIV state (WHO clinical stage III) with non specific interstitial pneumonia was made. The patient was started on prednisolone and bronchodilators, 3 drug antiretroviral treatment was added and ATT was withdrawn. The patient improved over the next few weeks. A chest x-ray and PFT after 1 month were normal. Steroids were tapered off and stopped after 3 months. A repeat CD4 count after 4 months was 368 cells / mm^3.

Discussion

The concept of Non Specific Interstitial Pneumonia (NSIP) was suggested by Katzenstein and Fiorelli (1994) to identify a group of interstitial lung diseases that have

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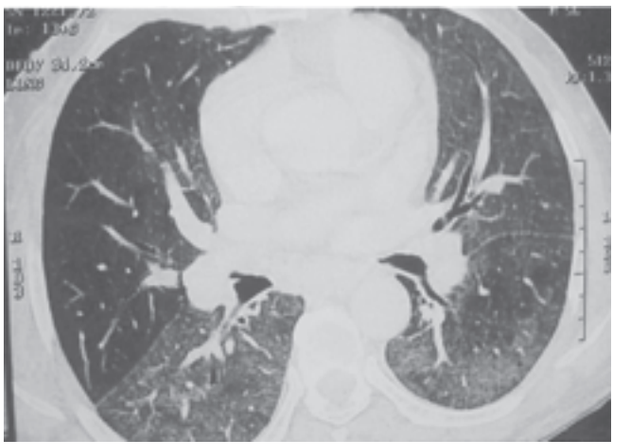

Fig.1 : HRCT sections showing diffuse ground glass attenuation with relative sparing of right middle lobe.

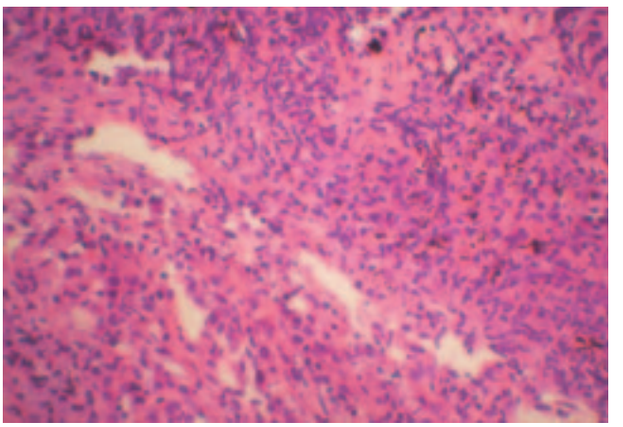

Fig.2: H and E stained section showing thickened interalveolar septae with areas of fibrosis and non-specific inflammation.

a more favourable outcome compared to idiopathic pulmonary fibrosis.[1] It is still a poorly defined condition, in immunocompromised hosts where it is diagnosed after exclusion of opportunistic infections and neoplasms.[2] 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. In adult AIDS patient with pulmonary symptoms, the reported incidence varies from 4.6% to 38%.[2] It is relatively more common in Africa where the reported incidence of PCP is low in patients of pulmonary diseases of undetermined aetiology.[2] The presentation is similar to PCP with breathlessness, cough and fatigue. About 50% patients may present with weight loss. Constitutional symptoms are less common.[1] Crackles are initially basal but may be widespread and inspiratory rhonchi may be present.[3] Radiological features are non-specific and like any interstitial lung disease. The presence of increase in percentage of lymphocytes in bronchoalveolar lavage along with other findings such as an HRCT and pulmonary function test strengthen the suspicion of NSIP.[1,3] Lung biopsy may show a mainly interstitial inflammation or fibrosis or a combination of the two and is negative for infection or tumour.[2] The histological features do not fit into other forms of ILD. specifically usual interstitial pneumonia (UIP) and desquamative interstitial pneumonia (DIP). 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.[1] 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. NSIP resolves spontaneously or responds to steroids and is not directly responsible for the patient's death. It has a better prognosis than DIP or UIP but it does mark a downward trend in the course of HIV infection.[5] Our patient showed a dramatic response to steroids clinically, radiologically and physiologically and he is symptom-free at six months of follow up.

In situations where transbronchial or open lung biopsy is not possible, after a failure of response to treatment of infectious causes and with stable viral load and CD4 counts, the diagnosis of NSIP should be entertained.[2]

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