Case Report ISSN (O):2395-2822; ISSN (P):2395-2814

A case of Lymphocytic Interstitial Pneumonia in a defaulter HIV-positive patient.

Vishwas Gupta[1], Vasant Nagvekar[2], Pralhad Prabhudesai[3], Gajanan Rodge[4]

[1]DNB Resident, Pulmonary Medicine Department, Lilavati Hospital, Mumbai, Maharashtra, India.
[2]ID Consultant, Lilavati Hospital, Mumbai, Maharashtra, India.
[3]Consultant, Pulmonary Medicine Department, Lilavati Hospital, Mumbai, Maharashtra, India.
[4]Clinical Associate, Internal Medicine Department, Lilavati Hospital, Mumbai, Maharashtra, India.

Received: March 2018
Accepted: March 2018

ABSTRACT

After the introduction of the Antiretroviral Therapy (ART) very few case reports of Lymphocytic interstitial pneumonitis (LIP) with HIV are available till date, especially in adults. In children LIP is still considered as AIDS defining condition. LIP is a polyclonal proliferation of lymphocytes, which can be misdiagnosed as Pneumocystis carnii pneumonia (PCP) or Hypersensitivity pneumonitis (HP) if proper sampling is not done. The mainstay of treatment is ART. Here we are presenting a case of lymphocytic interstitial pneumonitis in a defaulter case of HIV-positive patient. The Video-Assisted Thoracoscopic lung biopsy was taken and diagnosis was made on the basis of histopathology examination. Hence, it is suggested to consider for LIP as a differential for cough and dyspnoea in all HIV positive cases, who are not on ART.

Keywords: Lymphocytic Interstitial Pneumonia (LIP), Antiretroviral Therapy (ART), lung biopsy

INTRODUCTION

Lymphocytic Interstitial Pneumonia (LIP) in HIV patient was an uncommon disease prior to Antiretroviral Therapy (ART) era, and is become even less evident after introduction of ART. LIP is characterised by interstitial and alveolar space infiltration of the lung by lymphocytes, plasma cells, and lymphoreticular elements.[1] Here we are presenting a case of LIP in a defaulter case of HIV

CASE REPORT

57 year female, presented with complains of dyspnoea, dry cough and weight loss around 12 kg since 3 months. She denied history of fever, known exposure with industrial dust, asthma, seasonal allergy, gastroesophageal reflux disorder, postnasal drip or smoking tobacco. She also denied any history of muscle weakness, joint pain, rash, and photosensitivity, colour changes in her digits, dysphagia, dry eyes or dry mouth. She was diagnosed as HIV positive since 6 year, taken ART for 3 years and defaulted. Three months prior to presentation her Chest X ray done was suggestive of reticular opacities in bilateral lower zones and a High Resolution CT scan chest done was suggestive of ground glass opacities with septal thickening in bilateral lower lobes. She was started on perfinadone by her physician. Her vitals on presentation were stable and SpO2 was 88% on ambient air, she had bibasal crepts, other systemic examination were normal. At this time differential of Pneumocystis carnii pneumonia (PCP) was made and she was treated with Cotrimoxazole and trimethoprim. Bronchoscopy was performed and bronchoalveolar lavage (BAL) sent for ziehl-neelsen (ZN) staining and culture for mycobacterium tuberculosis (MTB) were negative, also Pneumocystis carnii pneumonia (PCP) stain, Cytomegalovirus (CMV) inclusion bodies were not seen. After one month she again presented with similar complains. Her repeat High Resolution CT chest was showing worsening of prior ground glass opacities and intralobular septal thickening. Her CD4 was 183 cells/ microliter, Complete Blood Counts, Liver Function Test, Renal Function Test, 2D-echocradiography were normal. Cyclic citrullinated peptide antibodies (Anti CCP), antinuclear antibodies (ANA), ds DNA, RA factor were negative, EBV DNA in blood was positive. Patient was subjected for thoracoscopic lung biopsy; post procedure she was kept on noninvasive ventilation support, lung tissue showed no growth of mycobacterium and histopathology showed dense diffuse small lymphocytic infiltrates in interstitium suggestive of LIP. Few small cell epitheloid cell granulomas were also seen. Small lymphocytic infiltrate is predominantly T cell (CD3, CD5

Name & Address of Corresponding Author
Dr. Gajanan Rodge
Clinical Associate, Internal Medicine Department,
Lilavati Hospital,
Mumbai,
Maharashtra, India.

Positive) with B cell seen mainly in the small lymphocytic aggregates. Kappa and Lambda chain showed no light chain restriction. The lyphocytic infiltration was polyclonal and EBERish was negative. Patient was started on second line ART with tenofovir, lamivudine, atazanavir plus ritonavir, and prednisolone 40mg/ day. Patient showed dramatic improvement in her symptoms in 6 week after discharge. Her repeat High Resolution CT scan chest, done in December 2015, was suggestive of improvement in ground glass opacities and septal thickening.

DISCUSSION

LIP is a rare disorder likely occurs due to derangement in immune system. In adults who are HIV positive, onset of LIP does not correlate with CD4 count, although LIP is an AIDS defining condition in children.

LIP is one of the variant of the lyphoproliferative disorder of lung ranging from benign, small, airway-centred cellular aggregates to malignant lymphomas. In an nonsmoker, normal adult human lung ,the constellation of unencapsulated lymphoid follicles, cellular collections, and loosely distributed mucosal lymphocytes constitute the Bronchus Associated Lymphoid Tissue (BALT) The extent of proliferation of Bronchus Associated Lymphoid Tissue (BALT) is responsible for various morphologic patterns and HIV can induce such types of proliferation.[2,3] Animal data convincingly display a relationship between HIV and LIP. Bronchio alveolar lavage specimen shows a significantly increased number of cells expressing human T-lymphotropic virus (HTLV) type III RNA in HIV-positive patients with LIP compared to HIV positive patients with lung pathology other than LIP.[4] An acquired benign diffuse proliferation of BALT includes follicular bronchitis/bronchiolitis (FBB), diffuse lymphoid hyperplasia, and LIP. LIP represents a benign polyclonal proliferation usually of mature B or T cells. In HIV-positive adults, a nonspecific interstitial pneumonitis is much more common than LIP. LIP is a non-specific response to multiple known and unknown stimuli. In 1966, Liebow and Carrington suggested many of these stimuli, including viral agents, either by their direct action or by their inducing failure in lung immune surveillance mechanisms.[5] Simultaneous infection with EBV and HIV may amplify the risk of development of LIP. Mutations in the noncoding regulatory (promoter) regions of B cell lymphoma 6 (BCL6) gene have been identified in several pulmonary lymphoproliferative disorders, including LIP. Definitive diagnosis of LIP requires a surgical lung biopsy.[6,7] The histopathologic findings of this case were consistent with the ATS guidelines.[8] Infectious aetiologies of diffuse pulmonary opacities (e.g. PCP, cytomegalovirus and mycobacteria) are an important differential diagnosis. The pathologic appearance of LIP and hypersensitivity pneumonitis is also similar. So far no randomized controlled trial has been done but on the basis of several observation it has been suggested to initiate ART in all symptomatic LIP patients along with glucocorticoids.[9,10]

CONCLUSION

Because of the rarity of LIP in adult population especially in patient who are not on ART, the diagnosis often missed if lung biopsy is not performed. The cause of lung infiltrate often overlooked because of high incidence of opportunistic infection like PCP in HIV positive individual. Hence, it is suggested that in HIV positive adult patients who are not on ART, should be evaluated for LIP as a cause of chronic cough and dyspnoea and the appropriate ART should be initiated along with steroids.

REFERENCES

1. Swigris JJ, Berry GJ, Raffin TA, Kuschner WG. Lymphoid interstitial pneumonia: a narrative review. Chest 2002;122(6):2150-64.
2. Walker BD, Chakrabarti S, Moss B, Paradis TJ, Flynn T, Durno AG, et al. HIV-specific cytotoxic T lymphocytes in seropositive individuals. Nature 1987 Jul 23-29;328(6128):345-8.
3. Ho DD, Pomerantz RJ, Kaplan JC. Pathogenesis of infection with human immunodeficiency virus. N Engl J Med. 1987 Jul 30;317(5):278-86.
4. Chayt KJ, Harper ME, Marselle LM, Lewin EB, Rose RM, Oleske JM, et al. Detection of HTLV-III RNA in lungs of patients with AIDS and pulmonary involvement. JAMA 1986;256:2356–9.
5. Liebow A, Carrington C. Diffuse pulmonary lymphoreticular infiltrations associated with dysproteinemia. Med Clin North Am 1973; 57:809–43
6. Travis WD, Costabel U, Hansell DM, King TE Jr, Lynch DA, Nicholson AG, et al. An official American Thoracic Society/European Respiratory Society statement: Update of the international multidisciplinary classification of the idiopathic interstitial pneumonias. Am J Respir Crit Care Med. 2013 Sep 15;188(6):733-48.
7. Cosgrove GP, Schwartz MI. Lymphocytoplasmic infiltrations of the lung. In: Schwarz MI, King TE, Editors. Interstitial Lung Disease, 5th ed. Shelton, CT, USA: People's Medical Publishing House; 2011. p. 1045.
8. American Thoracic Society, European Respiratory Society. American Thoracic Society/European Respiratory Society International Multidisciplinary Consensus Classification of the Idiopathic Interstitial Pneumonias. This joint statement of the American Thoracic Society (ATS), and the European Respiratory Society (ERS) was adopted by the ATS board of directors, June 2001 and by the ERS Executive Committee, June 2001. Am J Respir Crit Care Med 2002; jan15;165(2):277-304.
9. Scarborough M, Lishman S, Shaw P, Fakoya A, Miller RF. Lymphocytic interstitial pneumonitis in an HIV-infected adult: response to antiretroviral therapy. Int J STD AIDS 2000 Feb;11(2):119-22.

10. Teirstein AS, Rosen MJ. Lymphocytic interstitial pneumonia. Clin Chest Med 1988 Sep;9(3):467-71.

How to cite this article: Gupta V, Nagvekar V, Prabhudesai P, Rodge G. A case of Lymphocytic Interstitial Pneumonia in a defaulter HIV-positive patient. Ann. Int. Med. Den. Res. 2018; 4(3): ME40-ME42.

Source of Support: Nil, **Conflict of Interest:** None declared