Bronchiectasis as a cause of unexplained breathlessness in HIV infected patient

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Citation

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Abstract

Human immunodeficiency virus (HIV) infection has been associated with a wide spectrum of pulmonary disease. Here we report a case of 32 year old, HIV seropositive male having cough and progressive breathlessness for one year. Progressive breathlessness was diagnosed to be due to unusually rapid airway obstruction associated with bronchiectasis in this patient.

INTRODUCTION

The lung is the site of a wide spectrum of disorders complicating the clinical course of the human immunodeficiency virus (HIV) infection. However, the occurrence of bronchiectasis has rarely been reported[1,2,3,4,5,6]. The incidence of bronchiectasis in the HIV infected population remains to be established, since it is frequently undiagnosed because of a low index of suspicion and because chest radiographs may be normal or nonspecific. Accelerated airway obstruction associated with bronchiectasis is one of the causes of unexplained dyspnea in HIV infected patients[1]. Here we are reporting this case because of clinical interest.

CASE SUMMARY

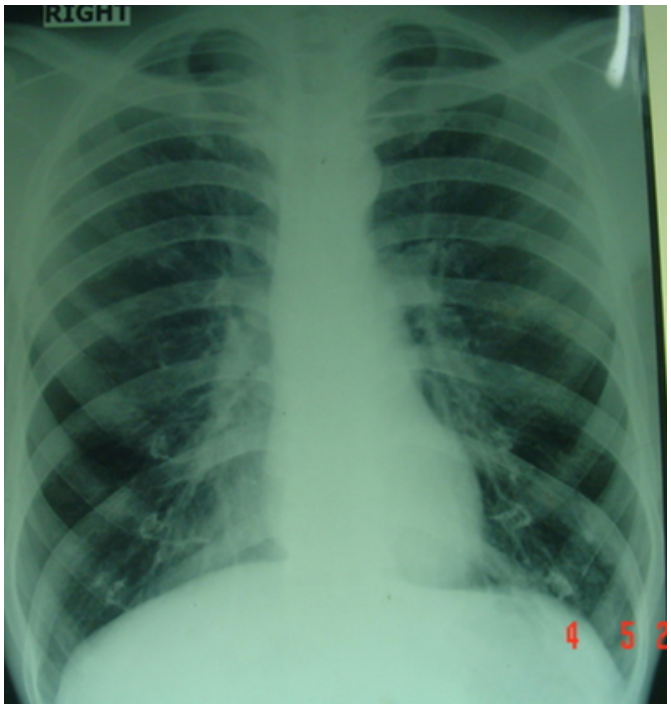
A 32 year old male, teacher by occupation was admitted to Department of Pulmonary Medicine, King George's Medical University, Lucknow with chief complaints of cough with minimal expectoration and progressive breathlessness for one year. There was no history of fever, loss of appetite and haemoptysis. There was no family history of pulmonary disease or a personal history of severe chest illness in childhood. Patient was a known case of HIV seropositive for last two year His CD4 count was 209/µL at that time. He was on antiretroviral therapy (lamivudine, stavudine, nevirapine) along with cotrimoxazole as prophylaxis for pneumocystis carinii infection. He also had a course of adequate antitubercular treatment three month back. Patient was a chronic smoker (10 pack years) and chronic alcoholic. He was also a known intravenous drug abuser and had stopped three years before when he was diagnosed to be a case of HIV seropositive.

On admission, he was ill looking, afebrile with a blood pressure of 110/68 mm Hg; pulse rate of 92 per minute and respiratory rate of 30 breaths per minute. The oxygen saturation by pulse oximetry was 86% on room air. There was clubbing of his fingers and toes of grade II. Examination of Respiratory system and other systems was unremarkable.

His hematological and bio-chemical investigations were with in normal limits. His PPD test showed 12 mm indurations after 72 hour. On admission his CD4 count was 497/µL. His chest radiograph was normal

Figure 1

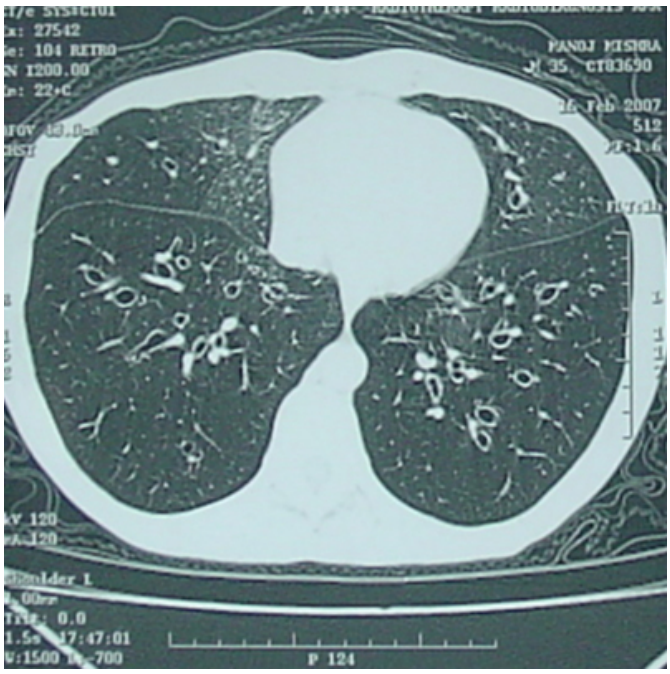
Figure 1: Normal looking Chest radiograph.



His 2-D Echo was also normal. Sputum culture for pyogenic organisms was sterile after 48 hours of incubation. The sputum samples for acid-fast bacilli, fungal elements and pneumocystis carinii were negative and the culture of sputum by BACTEC also did not show any mycobacteria. Spirometry showed features of irreversible very severe airway obstruction (post bronchodilator FEV1 16% and FEV1/FVC ratio 53). Arterial blood gas analysis on room air showed hypoxemia (PaO_2 72.8 mmHg) without hypercapnia ($PaCO_2$ 34.2 mmHg). A high resolution computed tomography (HRCT) of the chest revealed dilated thick walled bronchi suggestive of bronchiectasis.

Figure 2

Figure 2:High resolution computed tomography of the chest showing bilateral bronchiectasis with predominant distribution in both lower lobe and right middle lobe.



Distribution of bronchiectasis was bilateral with predominance for both lower lobe and right middle lobe. The patient underwent fibreoptic bronchoscopy that did not reveal any endobronchial growth. Bronchoalveolar lavage smears revealed no bacteria, acid-fast bacilli, fungal element and pneumocystis carinii.

DISCUSSION

Pulmonary complications have been an important source of morbidity and mortality in the HIV infected patients. Both infectious and noninfectious complications have been well described[7,8,9]. However, evidence exists that HIV-infected individuals may acquire unexplained alterations in pulmonary function[10] and high resolution chest CT[11] independent of or prior to overt pulmonary complications (as in our case also).

No classical causes of genetic or acquired bronchiectasis, such as pertussis or measles pneumonia, foreign body inhalation, mycobacterial infection, cystic fibrosis hypogammaglobinaemia, rheumatoid arthritis, were identified in the present patient.

Although our patient was smoker, the clinical course was not compatible with smoke related bronchial disease alone because of short time over which the patient develops bronchiectasis.

Our patient was a known intravenous drug abuser. However, he had not used intravenous drug for more than 4 years when he developed dyspnea. Airway function analysis, chest radiography, and chest CT scan depicted differing pathologies from the panlobular emphysema or foreign particle embolization commonly associated with intravenous drug abusers[12]. Our patient did not have a past history of overdose or aspiration pneumonia, both of which are classical causes of bronchiectasis in intravenous drug abusers. Other lung diseases related to intravenous drug abuse, such as noncardiogenic pulmonary edema, bronchospasm, Eosinophilic pneumonia, clearly differ from the manifestations observed in the present patient[13].

Obstructive disorders due to opportunistic pneumonia or Kaposi's sarcoma have been reported previously in HIV-infected patients[14]. No evidence of mycobacterial, parasitic or mycotic agents was isolated from sputum and Bronchoalveolar sample in this patient. Radiological and endobronchial evidence of Kaposi's sarcoma was also absent. The obstructive disease observed in this patient was different from emphysema like syndrome, described in patients with prolonged HIV infection[15]. This emphysema like syndrome is characterized by hyperinflation with an increase in residual volume but only minimal airway obstruction. The radiological aspect differs from cases of bronchiolitis obliterans with organizing pneumonia previously reported in HIV infected patients and characterized by pulmonary consolidation[16]. Airway obstruction was associated with bronchiectasis in this patient. Diffuse bronchiectasis has been reported previously on chest CT scan studies in HIV-infected patients[1,2,3,4,5,6]. In a previous study, it was documented that AIDS patient have an accelerated form of bronchiectasis which can develop within 4 weeks[2]. This accelerated form of bronchiectasis can develop in the absence of mycobacterial infection or a history of prior recurrent pyogenic infection[2] as it was seen in our patient also.

In conclusion, accelerated airway obstruction associated with bronchiectasis should be added to the wide spectrum of respiratory complications of HIV infection which is one of the causes of unexplained dyspnea in these patients. The practice of pulmonary function tests in HIV infected patients complaining of exertional dyspnea permits a better characterization of this bronchial disease in future.

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