HIV AND BULLOUS LUNG DISEASE

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The mechanisms behind accelerated emphysema in adults with HIV infection and the HIV-infected smoking population are both multifactorial and unclear. However, the association of HIV and emphysematous lung disease is recognised. We describe a patient with HIV infection and accelerated emphysema, highlighting the facts that no other background disease predisposed him to these lung changes, and that smoking in conjunction with HIV infection acted synergistically to produce the changes.

CASE REPORT

A 38-year-old man with a 3-day history of shortness of breath, productive cough and pleuritic chest pain presented to the medical department at Helen Joseph Hospital, Johannesburg. There was no history of tuberculosis or other lung disease, but the patient had been a smoker for 10 years. He was on no medication. On examination he presented with a respiratory rate of 20/min, tachycardia (164 beats/min), and a blood pressure of 98/66 mmHg. He had decreased air entry on auscultation of the left chest. An initial chest radiograph revealed a differential transradiancy of the hemithoraces, and a diagnosis of spontaneous left pneumothorax was made. The patient was placed on oxygen and an intercostal drain (ICD) inserted. Blood tests revealed that he was HIV infected, with a CD4 count of 469 cells/µl and a white cell count of 469 x 10^9/l. He was not on antiretroviral therapy. Once his condition had stabilised, a high-resolution computed tomography scan of the chest was done (Figs 1 and 2). This revealed large bilateral, diffuse, predominantly apical, medial and lateral bullous lung disease associated with a small residual left pneumothorax. The underlying lung parenchyma deep to the paraseptal bullae was not affected. No radiological features of pre-existing consolidation, cavities or cysts were present, and there were no nodules, reticules or bronchiectasis. There was no lymphadenopathy or effusions. The diagnosis of spontaneous pneumothorax secondary to bullous lung disease in an HIV-positive male smoker was made.

Near-total re-expansion of the left lung occurred. The ICD was removed and the patient was discharged to an outpatient clinic.

DISCUSSION

Emphysematous lung disease has become a known pulmonary complication of HIV and AIDS. HIV-related bullous lung disease was first reported in the late 1980s. Subsequently numerous studies have indicated that the HI virus itself is a predisposing factor in the pathogenesis of bullous lung disease.[1-3]

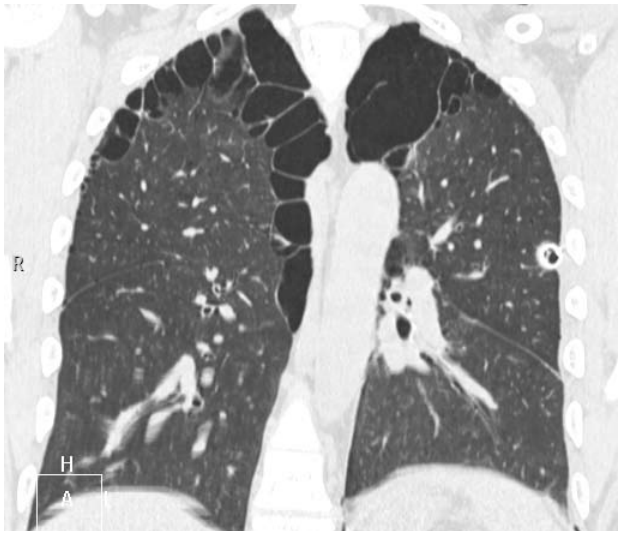

Fig. 1. Coronal reconstruction of the lung parenchyma on lung windows. Large bilateral apical and medial paraseptal bullae are present. Note the absence of any parenchymal pathology deep to the paraseptal bullae. An intercostal drain tip is seen in the left lateral pleural space.

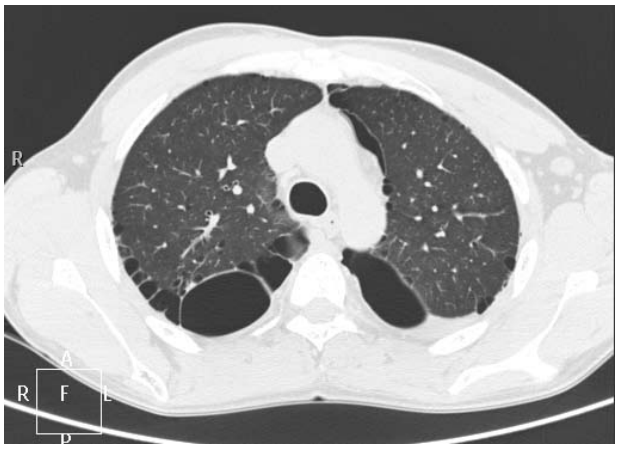

Fig. 2. Axial computed tomography scan on lung windows. Large bilateral paraseptal bullae are demonstrated with residual antero-medial pneumothorax.

In 1989 a publication by Kuhlman *et al.* compared the incidence of bullous lung damage in a group of HIV-positive patients with that in a similar group of immunocompromised patients with acute leukaemia.[4] Bullous lung damage was found in 42% of the HIV-positive group, as opposed to 16% of the acute leukaemia group. The average age of the HIV-positive group was also significantly lower than that of the leukaemia group. The study documented the distribution of bullous

changes to be predominantly apical and peripheral. Of the patients 70% had a history of previous documented pulmonary infections (in particular *Pneumocystis jirovecii* pneumonia) and 13% did not, emphasising the direct effect of HIV itself on the lung parenchyma. Spontaneous pneumothoraces were a common complication in patients with bullous lung disease.

Other studies also emphasise the role of HIV in premature emphysema and bullous lung disease. HIV-infected subjects have significantly more emphysematous lung damage than HIV-negative smokers.[1,5] HIV-associated emphysema also occurs over a much shorter period of time than smoking-related emphysema in HIV-negative patients. This is thought to reflect an increase in and a susceptibility to damage caused by cigarette smoking in HIV-positive patients.[1]

In 2000 Diaz and co-workers compared the incidence of emphysema in an HIV-positive group and an HIV-negative group matched for age and smoking history.[5] The incidence of emphysema was 15% for the HIV-positive and 2% for the HIV-negative group. Smoking was the single most important risk factor in the HIV-positive group, contributing to 37% of the patients with emphysema in this group, compared with 0% in the HIV-negative group.

The differential diagnosis of bullous lung disease includes tobacco smoking, intravenous drug use (methyphenidate, heroin, cocaine), marijuana and cocaine smoking, and a long list of diseases including α_1-antitrypsin deficiency, HIV infection, auto-immune and connective tissue disorders, bullous sarcoidosis, idiopathic giant bullous emphysema and neurofibromatosis.[2]

A higher percentage of cytotoxic lymphocytes has been demonstrated in broncho-alveolar lavage specimens of HIV-infected patients than in uninfected subjects.[1,5] In the 1990s reports of accumulation of CD8 cytotoxic T lymphocytes in the lungs of patients with severe chronic obstructive pulmonary disease (COPD) appeared. This may explain the accelerated emphysema in HIV-infected patients, whose lung response to the HIV infection is characterised by the accumulation of CD8 lymphocytes in alveolar spaces. Lymphocytic alveolitis, defined as more than 15% lymphocytes in broncho-alveolar lavage, is a common finding in HIV-infected subjects.[1]

More recently, highly active retroviral therapy (HAART) has been associated with a significant decrease in the number of CD8 cells in broncho-alveolar lavage. This raises the interesting possibility that the incidence of COPD in HIV-infected subjects may decrease significantly in the HAART era.[1]

CONCLUSION

The spectrum of HIV-associated accelerated emphysematous lung changes carries significant morbidity. It is therefore important to recognise early lung changes as well as understand the predisposing factors associated with accelerated bullous lung disease in the hope that prevention, early detection and treatment will decrease morbidity and mortality in these patients.

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