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Research Article

HIV or AIDS Infection is Related to an Elevated Danger of Lung Cancer Regardless of Smoking

Nabila Younas[1*], Farwa Sikandar[2], Muhammad Usman[3]

1Department of Medicine, Services Hospital, Lahore, Pakistan
[2]Department of Medicine, Akhter Saeed Trust Hospital, Lahore, Pakistan
[3]Department of Medicine, Mayo Hospital, Lahore, Pakistan

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ABSTRACT

Aim: HIV-contaminated people are increasingly at risk for cellular lung breakdown, though the increase reflects their heavy use of tobacco remains an open question.

Methods: Since 1988, a member of infusion drug clients has been tentatively identified in Baltimore, Maryland, using semi-annual clinics, research center and social information, as a result of the Acquired Immunodeficiency Syndrome (AIDS) link to the intravenous experimental study. Our current research was conducted at Mayo Hospital, Lahore from October 2019 to September 2020. The cellular disintegration of the lungs through the connection with the National Demise Index was recognized. Cox relapse of risks was used to examine the cellular lung hazard effects of HIV disease, smoking status, medicines and clinical factors.

Results: Among the 2087 members of the "AIDS Link to the Intravenous Experience" study, which was carried out over 19,830 person-years, 54 cell failures in the pulmonary passages were recognized; 15 of these passages involved people infected with HIV. All but one patient (95%) were smokers, who smoked an average of 1.2 packs per day. Mortality due to cell decay in the lungs increased during the period of highly dynamic antiretroviral treatment, in contrast to the exceptionally dynamic antiretroviral treatment period (proportion of mortality rate, 4.9; 99% certainty interval, 1.7-18). After adjusting for age, gender, smoking status, and treatment period, HIV infection was associated with extensive cellular degradation in the lungs (hazard ratio, 3.6; 95% certainty range, 1.6-9.7). Previous lung infections, particularly non-infectious diseases and asthma, have shown patterns of extensive cellular degradation in lung risk. The use of illegal drugs was not associated with extensive cellular degradation in lung risk. In HIV-infected people, smoking remains the most important hazard factor; CD4 cell counts and HIV load are not closely related to extensive cellular degradation in the lungs, and extensive hazard patterns with the use of exceptionally aggressive antiretroviral therapy are not critical.

Conclusion: Contamination with HIV is associated with an increased danger of autonomous cellular lung breakdown.

Key words: HIV, AIDS Infection, Elevated Danger of Lung Cancer Regardless of Smoking, Cellular lung breakdown

***Correspondence:**

Nabila Younas, Department of Medicine, Services Hospital, Lahore, Pakistan, E-mail: phdali786@gmail.com

INTRODUCTION

Cellular degradation in the lungs is the third most important damage in people infected with HIV, after AIDS, which defines malignant tumors, Kaposi's sarcoma and non-Hodgkin's lymphoma. Epidemiological tests have shown an increased danger of cellular decomposition in the lungs in people infected with HIV (Thompson SC, *et al.*, 1996). With delayed endurance due to the availability of HAART, moroseness and mortality due to cellular breakdown in the lungs may increase for a very long time thereafter in HIV-infected individuals. One question that remains is whether the observed relationship between HIV disease and cellular degradation in the lungs simply reflects a high rate of smoking in HIV-infected individuals or whether it is a gratuitous consequence of HIV disease henceforth (Phelps RM, *et al.*, 2001). Previous studies of lung cell degradation in HIV-infected individuals have encountered some obstacles, including the lack of appropriate non-HIV-infected reference groups, dependence on lung cell degradation in all subjects to be examined with those from HIV-infected populations, limited subsequent experience during the HAART period, and the modest number of cases of lung cell degradation (Wistuba II, *et al.*, 1998). Previous findings may be influenced, in particular, by the uncontrolled frustration associated with smoking, which is the predominant hazard factor for cell decay in the lungs. Since HIV-infected people generally smoke more heavily than anyone else, insufficient control of this novel hazard factor could cause a clear relationship between HIV disease and cell degradation in the lungs (El-Solh A, *et al.*, 1997). We evaluated the cellular degradation in the lungs of the mortality of members of the AIDS Link to the Intravenous Experience (ALIVE) study, a huge and long-standing accomplice of drug infusion clients. Using a longitudinal assessment of smoking among HIV-infected and uninfected limbs for a full subsequent period, we were able to analyze the relationship between HIV disease and cellular degradation in the lungs, while directly representing openness to smoking (Bonnet F, *et al.*, 2004).

METHODOLOGY

Members of the ALIVE study conducted biannual visits that included organized meetings, a computerized risk survey, clinical assessment, and an assortment of blood samples. Point-by-point data were discussed regarding lifestyle factors, including smoking status and illegal drug use. Because of the standardized structure of the biannual visits, the surveys focused on behavior during this period. Our current research was conducted at Mayo Hospital, Lahore from October 2019 to September 2020. The antiretroviral drug use reported by respondents was updated at each visit. HAART was characterized by the use of three antiretroviral drugs, including a protease inhibitor, a non-nucleoside reverse transcriptase inhibitor or a batcaver. The HAART period has been characterized as beginning May 1, 1999. Confirmations from emergency lung infection clinics were distinguished, and the analysis was confirmed by a standardized audit of clinical records. HIV immune response testing of uninfected subjects was performed using commercially available tests deciphered according to standard criteria. Deaths among ALIVE study members were

learned from relatives or accomplices through standard investigative tracking systems and, in addition, through the link to the National Death Index (updated to 2003). For example, death reports were extracted from state records and examined to affirm that malignant growth in the lungs was the cause of death. The impact of HIV status on cellular degradation in lung mortality rates in the pre-HAART and HAART periods was assessed using Poisson relapse methods. Relative Cox risk relapse models were used to examine the relationship between HIV disease and cellular degradation in transient lungs, controlling for other covariates.

RESULTS

At partner origin, the average age of ALIVE study members was 38 years old, 78% were male, 94% were African-American, and 25% were HIV-infected. At follow-up, an additional 334 members were identified as having seroconverted to HIV. At the chapter, 84% of members detailed their smoking, and of these, 46% smoked a total of 1 packet per day and 10% smoked a total of 2 packs per day. 68% of members detailed their smoking at each follow-up visit, and only 8% of them reported never having smoked cigarettes *(Table 1)*. Neither the ubiquity of actual smoking (93% for HIV-infected patients versus 94% for non-HIV-infected; Pp.17) nor the average measurement of cigarettes smoked (0.8 packs per day for both) contrasted with HIV status. All subjects had a history of infusion drug use; at follow-up, these subjects reported late infusion drug use at 54% of visits. Smoking of illegal drugs was finally accounted for at follow-up by 77% of participants. For 28 members, the primary reason for death was pulmonary malignancy *(Table 2)*; 15 of these patients were HIV-infected (4 patients seroconverted during follow-up). All of the HIV seroconversions on file occurred at least 4 years prior to the cell degradation in the lungs associated with death *(Figure 1)*. At death, HIV-infected persons with lung cell degradation were younger than non-HIV-infected persons (mean age, 53 years vs 57 years; Pp.07). Gender, race, smoking and drug use, as well as analysis of previous lung disease, were analyzed according to HIV status in persons with cellular degradation in the lungs (P 1.21, for all tests). All but one person with lung cell damage was found to be a cigarette smoker. HIV-positive and non-HIV-positive individuals reported smoking an average of 1.3 packs of cigarettes per day *(Table 3)*.

DISCUSSION

There were some limitations to our review. First, we were dependent on the finding of cellular degradation in the pulmonary passages, as opposed to episodes *(Table 4)*. Nevertheless, given the helpless endurance of cellular degradation in the lungs of patients, mortality can be a magnificent surrogate for occurrence; this is particularly valid for an unselected study population with peak disease phases (Alavanja MC, *et al.*, 1992). Moreover, our strategies for determining cell degradation in lung mortality were indistinguishable for HIV-infected and non-HIV-infected limbs (Brownson RC and Alavanja MC, 2000). Second, the amount of cellular degradation in lung passages was low, which limited some research (Littman AJ, *et al.*, 2004). Third, we had no data on the histological subtypes of cell degradation in the lungs, which could have given a biased assessment of the effect of smoking (Mayne ST, *et al.*, 1999). Finally, the study population is predominantly African-American and male, most are heavy smokers, and all have a history of infusion drug use. Subsequently, speculation about our findings to HIV-infected people of different races or to people who congregate for reasons of danger or who smoke less should be viewed with caution (Ramanakumar AV, *et al.*, 2006).

Table 1: The primary reason for death was pulmonary malignancy

Variable	Patients (n=28)
Age at the time of death, median years (range)	53 (40-68)
Male Sex	19 (70)
Race	Accuracy: 95%
Black	25 (93)
Other	2 (7)
HIV infected	14 (52)
Ever smoked cigarettes	26 (96)
Mean no of packs of cigarettes	1.2 (0.0-2.0)
Daily injection of drugs	7 (26)
Ever inhaled illicit drugs	18 (67)
Pre-existing lung disease	Accuracy: 95%
1 Hospital admission	4 (15)
>2 Hospital admissions	2 (7)
Prior non-infectious lung disease	Accuracy: 95%
1 Hospital admission	0 (0)
>2 Hospital admissions	1 (4)
Pulmonary infection	Accuracy: 95%
1 pulmonary infection	4 (15)
Recurrent pulmonary infection	2 (7)
Pneumonia	Accuracy: 95%
1 pneumonia diagnosis	4 (15)
Recurrent pneumonia diagnosis	2 (7)

Table 2: Multivariate model includes adjustment for all other variables

Variable	Multivariate model [HR (95% CI)]	HIV Risk estimate [HR (95% CI)]
Sex	2 (7)	2 (7)
Male	1	1
Female	2.0 (0.88-47.7)	1.9 (0.82-4.4)
Mean no of packs of cigarettes	2 (7)	2 (7)
Per 1 pack/day increase	1.8 (1.3-2.6)	1.8 (1.3-2.5)
HIV Status	2 (7)	2 (7)
Infected	3.4 (1.6-7.4)	3.6 (1.6-7.9)
Uninfected	1	1
Time period	2 (7)	2 (7)
Pre-HAART era	1	1
HAART era	1.9 (0.69-5.1)	1.7 (0.62-4.6)
Multivariate model includes adjustment for all other variables in the table. All models includes adjustment for age		

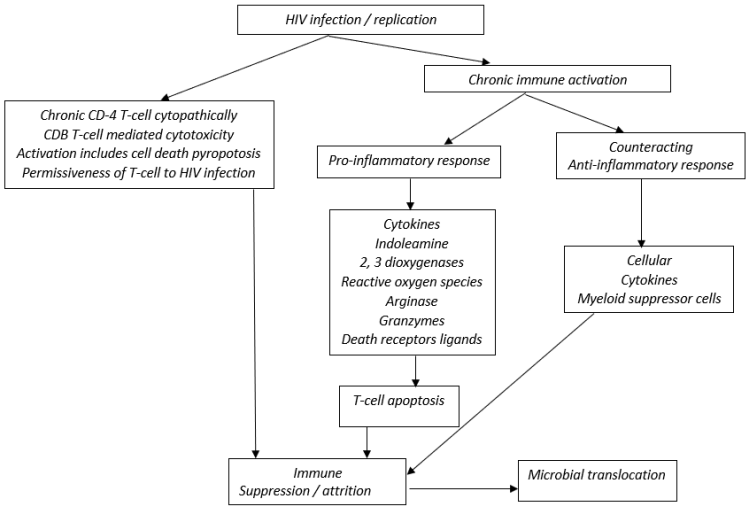

Figure 1: Flow chart of HIV infection

Table 3: HIV risk estimates by different model variables

Variable	Univariate model [HR (95% CI)]	Multivariate model [HR (95% CI)]	HIV Risk estimate [HR (95% CI)]
Daily injection of drugs			3.6 (1.6-8.0)
Yes	1	1	
No	0.61 (0.26-1.5)	0.55 (0.23-1.3)	
Inhaled illicit drugs			3.5 (1.6-7.7)
Never	1	1	
Ever	1.0 (0.44-2.3)	0.97 (0.42-2.3)	
Any Lung Disease			3.5 (1.5-7.9)
None	1	1	
>1 episode	2.1 (0.8-5.1)	1.2 (0.45-3.1)	
Non-infectious Lung Disease			3.6 (1.6-7.9)
None	1	1	
>1 episode	2.1 (0.28-16)	1.7 (0.22-13)	
Asthma			3.6 (1.6-7.9)
None	1	1	
>1 episode	4.8 (0.64-36)	4.8 (0.63-36)	
Any Pulmonary infection			3.4 (1.5-7.7)
None	1	1	
>1 episode	2.3 (0.91-5.6)	1.3 (0.49-3.4)	
Bacterial pneumonia			3.4 (1.5-8.1)
None	1	1	
>1 episode	1.4 (0.32-5.8)	0.94 (0.21-4.1)	
>2 episode	3.1 (0.70-13)	1.3 (0.26-6.1)	

Table 4: HAART era of HIV uninfected and HIV infected patients

Time period, HIV status	No of persons who died of lung cancer	Follow-up persons-years	Morality rate ratio (95% CI)	HIV Risk estimate [HR (95% CI)]
Pre-HAART era	1.3 (0.26-6.1)	1.3 (0.26-6.1)	1.3 (0.26-6.1)	1.3 (0.26-6.1)
HIV uninfected	1	5344	18.7	1
HIV infected	4	4858	82.3	4.4 (4.4-220)
All	5	10202	49	-
HAART era	1.3 (0.26-6.1)	1.3 (0.26-6.1)	1.3 (0.26-6.1)	1.3 (0.26-6.1)
HIV uninfected	12	6250	192	1
HIV infected	10	3383	296	1 (0.60-3.9)
All	22	9633	228	-
Both are combined	1.3 (0.26-6.1)	1.3 (0.26-6.1)	1.3 (0.26-6.1)	1.3 (0.26-6.1)
HIV uninfected	13	11594	112	1
HIV infected	14	8241	170	1.5 (0.56-3.5)
HAART era was defined as beginning of July, 96	1.3 (0.26-6.1)	1.3 (0.26-6.1)	1.3 (0.26-6.1)	1.3 (0.26-6.1)

CONCLUSION

Overall, our information supports the speculation that HIV disease causes cellular degradation in the lungs and proves that this impact is independent of smoking status. Further investigation should focus on understanding the organic components of how HIV contamination can accelerate the cycle of lung carcinogenesis. Given the large number of HIV-infected people who reported heavy smoking and who have smoked for longer periods of time because of their antiretroviral therapy, cellular breakdown in the lungs is likely to become an increasing problem for this population. A better understanding of the risk of smoking-related cell degradation in the lungs in HIV-infected individuals will help guide smoking cessation and improve interventions to decrease the effect of smoking. Close observation of cell degradation in the lungs of HIV-infected individuals and sharing of HIV-related information is warranted to study the risk of lung cell degradation related to markers of HIV infection and delayed use of HAART.

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