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Findings in Asymptomatic HIV Infected Patients Undergoing Chest Computed Tomography Testing: Implications for Lung Cancer Screening

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Abstract

Background—HIV infected persons have a two to five-fold increased unadjusted risk of lung cancer. In the National Lung Screening Trial (NLST), computed tomography (CT) screening was associated with a reduction in lung cancer mortality among high-risk smokers. These results may not generalize to HIV infected persons, particularly if they are more likely to have false-positive chest CT findings.

Methods—We utilized data including standardized chest CT scans from 160 HIV infected and 138 uninfected Veterans enrolled between 2009–2012 in the multicenter Examinations of HIV Associated Lung Emphysema (EXHALE) Study. Abnormal CT findings were abstracted from clinical interpretations of the scans and classified as positive by NLST criteria vs. other findings. Clinical evaluations and diagnoses that ensued were abstracted from the medical record.

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Contributorship:

KS and KC participated in the design, data collection, analysis and manuscript writing. JW and SS participated in the analysis and manuscript writing. SB, AJ, JK, MRB, KA, DR and GSH all participated in the design, data collection, and manuscript writing portions of the study.

Competing Interests:

No authors report any relevant financial conflicts of interest to this study.

Results—There was no significant difference by HIV in the proportion of CT scans classified as positive by NLST criteria (29% of HIV infected and 24% of HIV uninfected, $p=0.3$). However, HIV infected participants with CD4 counts <200 cells/mm³ had significantly higher odds of positive scans, a finding that persisted in multivariable analysis. Evaluations triggered by abnormal CT scans were also similar in HIV infected and uninfected participants (all $p>0.05$).

Conclusion—HIV status was not associated with an increased risk of abnormal findings on CT or increased rates of follow-up testing in clinically stable outpatients with CD4 cell count >200 . These data reflect favorably on the balance of benefits and harms associated with lung cancer screening for HIV infected smokers with less severe immunodeficiency.

Keywords

HIV; lung cancer; non-AIDS malignancies; lung cancer screening; lung nodules

Background

Similar to the general population, lung cancer is now the leading cause of cancer death in HIV infected persons.[1, 2] Compared to HIV uninfected persons, there is a two- to five-fold unadjusted increase in the risk of lung cancer in persons infected with HIV.[3-7] Although some of this excess risk is attributed to higher smoking rates,[6, 8, 9] elevated lung cancer risks in HIV infected persons persist even after controlling for smoking and is increased among those with low CD4 cell count.[3, 4, 6] These data suggest that HIV infection is an independent risk factor for lung cancer.[3-6]

The National Lung Screening Trial (NLST) recently demonstrated a reduction in lung cancer mortality associated with computed tomography (CT) lung cancer screening in heavy smokers from the general population.[10] As a result, the National Comprehensive Cancer Network and other national organizations have published guidelines recommending low-dose CT (LDCT) screening in patients at high risk for lung cancer.[11-13] Additionally, lung cancer screening with LDCT has been adopted by some private health insurers and the Veterans Affairs Health System.[14, 15] As HIV infected heavy smokers may have more than twice the risk of lung cancer of HIV uninfected smokers,[3, 4] they may be a unique high-risk group that can be targeted for lung cancer screening interventions.

A potential concern in implementing widespread lung cancer screening is that approximately 20% of CTs have positive findings that require additional work-up, while only 1% of scans will reveal lung cancer.[16] Follow-up tests frequently include additional diagnostic CTs, but may include more invasive procedures such as fine needle aspiration or surgical biopsy that may lead to potentially severe complications.[17] As HIV infected patients are more likely to have a history of lung infections or other pulmonary diseases that may lead to structural lung changes, positive screening tests may be more common in HIV infected smokers.[18-21] The increased risk of lung cancer in HIV infected persons is less likely to affect the positivity rate, given the relatively low number of cancers expected to be detected by screening. Despite this, clinicians caring for HIV infected patients may be aware of the higher risk of lung cancer and other malignant and non-malignant lung diseases, and as a consequence be more likely to aggressively evaluate abnormal imaging findings. Therefore,

determination of the rate of positive findings on chest CT scanning in HIV infected persons and the subsequent follow-up evaluations of these findings would provide important information on the applicability of NLST data to HIV infected smokers.

In this study, we used data from a prospective cohort of asymptomatic HIV infected and uninfected Veterans, most with a significant smoking history, to compare the frequency of incidental chest CT findings, particularly pulmonary nodules, observed on chest CT scans obtained for research purposes. We then estimated the proportion of CT scans in HIV infected and HIV uninfected participants that would have been considered positive by NLST criteria. We compared the clinical evaluations triggered by positive CT scans in order to determine whether HIV infected patients were more likely to undergo additional diagnostic procedures.

Methods

Study Cohort

We used data from the Examinations of HIV Associated Lung Emphysema (EXHALE) cohort, a multi-center sub-study of the Veterans Aging Cohort Study (VACS).[22] EXHALE is an ongoing observational, longitudinal study conducted at four Veteran Affairs (VA) Medical Centers (VAMC), namely the Atlanta, Bronx, Houston and Los Angeles VAMC. HIV infected outpatients in VACS are approached for enrollment in EXHALE, and are block-matched to HIV negative subjects by current smoking status to achieve a sample with similar prevalence of current smoking. Those with chronic lung diseases (as noted by history or clinical examination) other than chronic obstructive pulmonary disease (COPD) or asthma are excluded, as are patients with acute respiratory infections or illness in the four weeks prior to the baseline measurements. However, patients with respiratory infection more than 4 weeks prior to enrollment were not excluded. Enrollment began in 2009 and is ongoing. As a part of the study protocol, all participants received baseline chest CT scans. These CT scans were interpreted by radiologists at participating sites and results were reported in the participant's electronic medical record.

Study CT scans

Study CT scans were all obtained using a standardized acquisition protocol on multi-detector scanners that were calibrated across centers by scanning of a lung phantom. Radiation dose for EXHALE CT scans was ~3.5 millisievert (mSv) (as compared to 1.5 mSv for NLST screening scans and 8 mSv for standard CTs).[23] All CT scans were read by clinical radiologists practicing at each site at the time they were obtained, with results communicated to the participants' primary medical provider. As this study was not a formal screening protocol, all recommendations made by radiologists and any additional evaluation of findings by medical providers were based on their standard practice. Interpreting radiologists at some sites could access patients' electronic medical records and were not blinded to their HIV status. No subsequent CT scans were included in the study protocol, and the performance of any follow-up imaging was at the discretion of patients' medical providers.

Data Collection

Demographic data, current smoking status, and tobacco use history were collected as part of the baseline EXHALE survey. Data on comorbidities, including chronic obstructive pulmonary disease (COPD), asthma, hepatitis C infection, and prior infections and opportunistic illnesses were obtained from the VACS database and were based on International Classification of Disease, 9th Edition (ICD9) diagnostic codes using validated algorithms.[24] Laboratory values closest to the date of EXHALE baseline enrollment, including CD4 count, and HIV viral load, were collected from the VA electronic laboratory database. Combination anti-retroviral therapy (cART) use, as defined by the presence of a multidrug antiretroviral regimen filled by the pharmacy during a three-month baseline period, was assessed using pharmacy data.

CT scan reports were abstracted by a reviewer (KS) blinded to the HIV status of the study participants. Our primary outcome was the presence of a non-calcified nodule (NCN) 4mm in diameter or any findings suggestive of lung cancer (positivity as per NLST criteria) on baseline chest CT scan.[10] Data were collected on all pulmonary nodules described in the CT reports, including nodule size, number, and presence of calcification. Nodules that were reported without any size description were assumed to be less than 4mm in diameter. All nodules without reported size were present on CT scans with other NLST positive nodules, so this classification did not affect any study outcomes. Other outcomes of interest from the CT scan reports included other incidental findings such as emphysematous changes, pleural effusions, ground glass infiltrates, bronchiectasis, or granulomas. Adenopathy was noted if the radiologist described it as clinically significant or if pulmonary lymph nodes were described as greater than 10 mm. Recommendations for clinical or imaging follow-up made by the interpreting radiologist were also recorded.

We then reviewed the medical records of all participants to determine subsequent evaluations prompted by the scan results within 18 months following the study CT. In the clinical evaluations triggered by these CT scans, our outcomes of interest were follow-up examinations (including follow-up CT scans, Positron Emission Tomography (PET) scans, bronchoscopy, CT guided biopsies, and surgical biopsies) in the presence or absence of recommendations for follow-up in the CT scan reports, and eventual diagnoses made by these evaluations. In addition, we determined the number of primary care visits completed by the participants in the 6 months after their CT scan to determine if HIV infected participants were subject to more primary care contact.

Data Analysis

Demographics, smoking status and numbers of pack-year smoked were compared among HIV infected and uninfected participants using Chi squared or Fisher's exact for categorical variables, t-tests for normally distributed continuous variables and the rank-sum test for non-normal continuous variables. The proportion of CT studies meeting NLST positivity criteria, other clinically significant findings, follow-up recommendations, and types of subsequent clinical evaluations were compared by HIV status using chi-square tests. After determining the rates of suspicious nodules in different, clinically relevant CD4 strata among HIV infected participants (namely >350, 200-350, and <200), we found that a CD4 count of 200

cells/mm³ was an important threshold for comparison. We therefore stratified the HIV infected participants by baseline CD4 count (<200 cells/mm³ versus ≥200 cells/mm³) and compared baseline characteristics among these two groups using the same methods as our analysis for the full cohort. We then compared the proportion of CT studies meeting NLST positivity by CD4 cell count strata in HIV infected participants using a chi-square test. We then fit a logistic regression model to determine predictors of a false positive CT scan, including HIV status (and CD4 cell count strata), age, race/ethnicity, sex, smoking status, and pack-years of smoking as covariates. As a sensitivity analysis we then limited this multivariable analysis to participants meeting NLST inclusion smoking history criteria (≥30 pack-years of smoking in current smokers or former smokers who quit within 15 years of study enrollment).

Based on the prevalence of pulmonary nodules observed among patients in the cohort, we estimated that the study had an 80% power to detect 15% difference in nodules meeting NLST criteria by HIV status at a 0.05 significance level. All analyses were performed using STATA 11 (College Station, TX).

Results

Our study included 299 participants, 54% (n=160) of whom were HIV infected. HIV infected participants were older (p=0.03) and more likely to be male (p<0.001) than HIV uninfected participants (Table 1). There was no difference in the distribution of race/ethnicity, smoking habits, or rates of baseline chronic lung disease between HIV infected and uninfected persons (p>0.05 for all comparisons). The majority of cohort subjects were either current or former smokers (>80%) with significant pack-year exposure (median 24.6 pack-years for current or former smokers in cohort). HIV infected participants were more likely to have been diagnosed with prior pulmonary infections including pneumonia (19% vs. 4%; p<0.001) and tuberculosis (7% vs 1%; p=0.007). Most HIV infected participants were prescribed cART and had well-controlled HIV viremia (Table 2; 80% for patients with CD4 ≥200 cells/mm³ versus 64% for patients with CD4 cell counts <200 cells/mm³; p=0.08).

CT Findings

Prevalent lung cancer was diagnosed by lung biopsy in 3 of the HIV infected and 1 of the HIV uninfected participants (2.0% vs. 0.7%; p=0.3). However, a substantially larger number of scans had incidental findings. The proportion of CT scans that met the positive criteria as defined in the NLST did not differ when comparing HIV infected to HIV uninfected persons (Table 3; 29% vs. 24%; p=0.3). HIV infected participants with baseline CD4 cell counts <200 cells/mm³ had a greater frequency of positive scans than HIV infected participants with CD4 counts ≥200 cells/mm³ (55% vs. 25%; p=0.008). There was no significant difference in other clinically significant findings on study CT scans including adenopathy and pleural effusions, but there was a trend towards more emphysematous changes in HIV infected participants (41% vs. 30%; p=0.05).

In our multivariable analysis, being HIV infected with a CD4 count <200 cells/mm³ was independently associated with increased odds of a false positive scan (Table 4; odds ratio [OR]: 3.6, 95% confidence interval [CI]: 1.4-9.4), as was increasing age (OR 1.3 for each 5-

year age increase, 95% CI: 1.0-1.6). In a sensitivity analysis using an identical model limited to participants meeting NLST inclusion criteria for smoking history, HIV infection with CD4 count <200 cells/mm³ was the only significant predictor of scan false positivity (OR: 4.9, 95% CI: 1.3-19.0).

Clinical Evaluation Following Study CT Scans

Clinical follow-up recommendations and patterns after study CT scan completion were similar among HIV infected and uninfected cohort participants (Table 5). Follow-up recommendations by the interpreting radiologists (23% vs. 30%; $p=0.2$) and subsequent follow-up completion rates (50% vs. 54%; $p=0.8$) were similar in the HIV infected and uninfected groups. HIV infected participants were more likely to have a routine medical visit in the 6 months after the study CT scan (89% vs. 63%; $p<0.001$). Positive study CT scans led to similar rates of follow-up procedures including subsequent CT scans, PET scans, and biopsies ($p>0.05$ for all comparisons). No bronchoscopies were performed in response to CT findings in either study group. All biopsies led to lung cancer diagnoses (all adenocarcinomas). Final diagnoses triggered by study CT scans did not differ by HIV status.

Discussion

HIV infected smokers are a unique high-risk group that may benefit from CT based lung cancer screening. However, chronic lung changes resulting from immunosuppression-related pulmonary infections have raised concerns as to whether the frequency of false positive tests may be higher in this patient group. In this study, we found a similar likelihood of pulmonary nodules meeting NLST criteria for a positive CT scan among asymptomatic HIV infected and uninfected persons. We also found similar patterns of clinical evaluation triggered by study CT scans, suggesting that follow-up may not be more aggressive in HIV infected patients with abnormal chest imaging findings. This provides preliminary evidence that lung cancer screening may have a favorable harm/benefit profile in certain HIV infected smokers.

A limited number of studies have systematically described imaging findings on chest CTs in HIV infected patients.[20, 25] Jasmer et al. reviewed 242 lung CTs (mostly obtained in the pre-cART era) describing the frequency of pulmonary nodules among other findings noted on imaging. In this study of younger patients (mean age 40), the authors found 36% of CTs had pulmonary nodules, however patients were often symptomatic from opportunistic infections at the time of their CT scans.[25] A more recent study described the prevalence of incidental findings on lung CTs obtained for the calculation of coronary artery calcification scores in a cohort of asymptomatic HIV infected patients. The proportion of patients with incidental findings requiring additional workup or medical referral was 43% in that cohort, the majority of whom were on stable cART.[20] Our study is the first to compare rates of incidental chest imaging findings in HIV infected persons to a matched, HIV uninfected control group, and we find similar rates of pulmonary nodules and other incidental findings in both groups. This is also the first study to investigate the applicability of lung cancer screening using NLST positivity criteria in HIV infected persons. In our cohort, HIV

infected participants with CD4 counts ≥ 200 cells/mm³ had a similar frequency of positive scans to the NLST (25% vs. 24%),

Inflammation has been linked to lung cancer risk in HIV infected persons.[26] However, higher rates of previous pulmonary infections did not increase the rate of suspicious pulmonary nodules or eventual inflammatory or infectious final diagnoses in our HIV infected participants compared to uninfected participants. We did, however, observe higher rates of emphysematous changes on imaging in HIV infected patients. The development of emphysema has been tied closely to inflammation, especially in HIV infected patients[27], and these findings continue to support a potential role of inflammatory processes in the development of lung nodules in these patients. Emphysema is also a known risk factor for lung cancer. Future analyses will include standardized quantitative and semi-quantitative assessment of emphysema severity and other markers of inflammation and their associations with lung nodules.

The rate of false positive tests in asymptomatic smokers undergoing chest CT scanning is a critical determinant in establishing the harms and cost-effectiveness of lung cancer screening.[28, 29] Positive screening tests can lead to invasive procedures such as bronchoscopy, needle biopsy, and surgical lung biopsy, which can all have serious complications, including respiratory failure and death.[10] Moreover, the morbidity associated with diagnostic tests for lung nodules may potentially be higher in HIV+ patients. [30] Specifically, the increased risk of emphysema in HIV+ patients[31-33] may confer greater risk of pneumothorax, the most common serious complication of lung biopsy.[34] No published data exist regarding lung cancer screening with chest CT in HIV infected persons, and therefore our findings represent a preliminary evaluation of the safety of this potentially important screening modality. The overall efficacy and cost-effectiveness of lung cancer screening in HIV infected persons could be further explored using simulation modeling, and our findings are critical to inform such models.

We found that HIV infected participants with CD4 counts <200 cells/mm³ were more likely to have a positive CT scan, even after adjusting for smoking exposure. This finding is likely related to a higher risk of lung infections associated with significant immunosuppression. It is also possible that radiologists reviewing the study CTs were aware of patients' CD4 count, potentially biasing the interpretations of the scans. This finding suggests that patients with CD4 counts <200 cells/mm³ may have higher lung cancer screening false positivity rates, and therefore may be subject to increased harms associated with screening. This finding deserves further investigation in prospective screening studies.

Clinicians caring for HIV infected persons may be aware of their increased risk of lung cancer. This may affect their pretest probability estimates for cancer when evaluating chest imaging. Despite this, the patterns of clinical evaluation triggered by CT findings in our study did not differ by HIV status. Although HIV infected participants had more frequent healthcare contact, abnormal CT findings were not investigated with more frequency. As radiologists often base their follow-up recommendations for pulmonary nodules on Fleischner society criteria,[35] which does not incorporate HIV infection as a prognostic factor, it is possible that clinicians in our study based their follow-up evaluations on

recommendations made by the radiologists interpreting our CT scans. HIV infected participants were also more likely to have had prior chest CT scans, which may have allowed interpreting clinicians to determine the stability of existing nodules, thereby limiting the need for further evaluations.

Our study has several strengths. This was a multi-site, prospective study design with demographically similar HIV infected and uninfected individuals with comparable smoking exposure. The imaging studies performed in our protocol were similar to the low dose CT scans used in lung cancer screening trials. The study collected data on “real-world” processes and therefore represents how clinicians would follow-up lung cancer screening scans in HIV infected patients in actual practice. Our cohort was small compared to other studies of lung cancer screening, which may have limited our ability to find differences in the overall rates of suspicious nodules between HIV infected and uninfected participants. Another limitation of our study was the exclusion of persons with prior lung diseases, besides COPD and asthma. This criterion likely excluded more HIV infected persons than HIV uninfected, potentially affecting the generalizeability of our findings. However, patients with prior lung diseases were also excluded from the NLST, which improves the validity of the extrapolation of our findings to the screening results of that trial. Other limitations include the unavailability of details on follow-up studies performed at non-VA medical centers. Additionally, not all patients in our study were smokers, and therefore would not meet NLST inclusion criteria; in exploratory analyses limited only to smokers, however, we found similar results to those from the overall cohort. Last, this study used data from a longitudinal study of lung function and was not a formal lung cancer screening protocol. Although the study CTs were included in participants’ electronic medical record, there were no study procedures that dictated a protocol for follow-up of CT results. Rather, follow-up was at the discretion of the patient’s medical provider. This may have affected the rates of clinical evaluation of CT findings as well as the determination of final diagnoses in patients with abnormal scans.

HIV infected patients with CD4 counts >200 in our study did not have higher likelihood of findings meeting NLST criteria for a positive chest CT compared to uninfected controls, despite more previous lung infections, and a greater risk of lung cancer. These data suggest a similar frequency of positive screening scans among well controlled HIV-infected compared to HIV-uninfected persons, suggesting that lung cancer screening could be favorable in HIV infected smokers. The risk/benefit profile of lung cancer screening requires further evaluation in high-risk HIV-infected persons.

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Table 1
Baseline Cohort Characteristics by HIV status with NLST Baseline Characteristics for Comparison

	HIV+ (n=160)	HIV- (n=139)	P-value**	NLST (n=53,454)
Age, median (IQR)	55 (50-59)	52 (48-58)	0.03	62
Male, n, %	157 (98)	122 (88)	<0.001	31,532 (59)
Race/ethnicity, n, %			0.1	
Black	115 (72)	89 (64)		2,376 (4)
White	19 (12)	28 (20)		48,549 (91)
Hispanic	26 (16)	22 (16)		935 (2)*
Other	0 (0)	0 (0)		2,529 (5)
Smoking Status, n, %			0.5	
Never Smoker	23 (14)	26 (19)		0 (0)
Former Smoker	34 (21)	31 (22)		27,677 (52)
Current Smoker	103 (64)	82 (59)		25,779 (48)
Pack-years by self-report, median (IQR)	26 (14-42)	22 (10-38)	0.4	48 (39-66)
COPD, n, %	22 (14)	24 (17)	0.4	5137 (10)
Asthma, n, %	32 (20)	24 (18)	0.5	5,228 (10)
Hepatitis C, n, %	58 (36)	27 (19)	0.001	--
Previous Infections or Opportunistic Illnesses				
Prior pneumonia, n, %	30 (19)	6 (4)	<0.001	--
Tuberculosis, n, %	11 (7)	1 (1)	0.007	--
Histoplasmosis, n, %	2 (1)	0 (0)	0.2	--
Cryptococcal Disease, n, %	1 (1)	0 (0)	0.4	--
Pneumocystis Jirovecii Pneumonia, n, %	2 (1)	0 (0)	0.2	--
Kaposi's Sarcoma, n, %	7 (4)	0 (0)	0.01	--
HIV Viral Load, median (IQR)	48 (48-185)	--		--
Baseline CD4 Count, n, %				
<200 cells/mm ³	22 (14)	--		--
200 cells/mm ³	138 (86)	--		--
Anti-retroviral Therapy Current Use, %	84.0	--		--

IQR: Interquartile range,

* The NLST did not report ethnicity in combination with race, therefore Hispanic ethnicity is not mutually exclusive.

** p-value values are for comparisons of HIV+ and HIV- study participants.

Table 2

Baseline Cohort Characteristics for HIV+ Participants by CD4 Count Strata

	HIV+ CD4 < 200 (n=22)	HIV+ CD4 200 (n=138)	P-value
Age, median (IQR)	56 (51-59)	55 (50-59)	0.9
Male, n, %	22 (100)	135 (98)	0.5
Race/ethnicity, n, %			0.9
Black	16 (73)	99 (72)	
White	2 (9)	17 (12)	
Hispanic	4 (18)	22 (16)	
Smoking Status, n, %			0.9
Never Smoker	4 (18)	19 (14)	
Former Smoker	4 (18)	28 (21)	
Current Smoker	14 (64)	87 (65)	
Pack-years by self-report, median (IQR)	27 (13-42)	26 (14-42)	0.8
COPD, n, %	2 (9)	20 (15)	0.5
Asthma, n, %	4 (18)	28 (21)	0.8
Hepatitis C, n, %	11 (50)	47 (34)	0.15
Previous Infections or Opportunistic Illnesses			
Prior pneumonia, n, %	7 (32)	23 (17)	0.09
Tuberculosis, n, %	3 (14)	8 (6)	0.2
Histoplasmosis, n, %	0 (0)	2 (2)	0.6
Cryptococcal Disease, n, %	0 (0)	1 (1)	0.7
<i>Pneumocystis Jirovecii</i> Pneumonia, n, %	0 (0)	2 (2)	0.6
Kaposi's Sarcoma, n, %	1 (5)	6 (4)	0.9
HIV Viral Load <400 Copies/mL	14 (64)	111 (80)	0.08
Anti-retroviral Therapy Current Use, %	22 (100)	113 (82)	0.03

Table 3

Findings on Computed Tomography by HIV Status

	HIV+ (n=160)	HIV- (n=139)	P-value
CT Scans Meeting NIST Positivity Criteria, Overall, %	29	24	0.3
By Baseline CD4 Count, %			
<200 cells/mm ³	55	--	0.008
200 cells/mm ³	25	--	
Any Nodules, %	48	48	0.9
Number of Nodules, median, (IQR)	2 (1-4)	1 (1-3)	0.2
Granulomas, %	24	18	0.2
Lymphadenopathy, %	13	6.5	0.1
Carcinoma Suggested, %	4	3	0.8
Emphysematous Changes, %	41	30	0.05
Pleural Effusion, %	0.0	0.7	0.5
Ground Glass Infiltrates, %	15	14	0.9
Bronchiectasis, %	6	6	0.8

IQR: Interquartile range.

Results of Logistic Regression Model Evaluating the Association of Predictors of Computed Tomography Scan False Positivity

Table 4

Characteristic	Multivariable Odds Ratio for False Positive Scan	95% CI
HIV Status		
Uninfected	--	
Infected, Baseline CD4 <200 cells/mm ³	3.1	1.2-8.2
Infected, Baseline CD4 ≥200 cells/mm ³	1.0	0.5-1.8
Age*		
	1.2	1.0-1.4
Male Gender		
	1.2	0.4-4.0
Race/Ethnicity		
White	--	--
Black	1.4	0.6-2.9
Hispanic	0.6	0.2-1.3
Smoking Status		
Never Smoker	--	
Former Smoker	0.5	0.2-1.4
Current Smoker	1.3	0.5-3.0
Pack-years of Smoking**		
	1.0	0.9-1.1

* 5 year increments.

** 1 year increments.

CI: Confidence Interval.

Table 5

Follow-up Patterns After Study Computed Tomography Scan, Follow-up Testing Triggered by Computed Tomography Scan Results and Final Diagnoses by HIV Status

	HIV+ (n=160)	HIV-(n=139)	<i>p value</i>
Follow-up Recommended by Radiologist, %	23	30	0.2
Follow-up Carried Out When Recommended, %	50	54	0.8
Follow-up Carried Out When Not Recommended, %	8	10	0.8
Previous CT Scan, n, %	39 (24)	23 (17)	0.1
Routine Medical Visits in 6 Months After CT, n, %	142 (89)	87 (63)	<0.001
Number of Routine Visits if Performed, median	2 (1-2)	1 (1-2)	0.03
Actions Resulting From CT Scans, n, %			
Follow-up CT Scan	26 (16)	31 (22)	0.2
CT Guided Biopsy	1 (0.7)	0 (0.0)	0.4
Positron Emission Tomography Scan	5 (3)	2 (1)	0.3
Surgical biopsy	2 (1)	1 (0.7)	0.6
Final Diagnoses, n, %			0.3
None	138 (86)	111 (80)	
Lung Cancer	3 (2)	1 (0.7)	
Inflammation, Infection, or Stable Pulmonary Nodules	17 (11)	24 (17)	
Non-cancer Pathology	1 (0.70)	1 (0.6)	
New or Progressed Lesions	1 (0.6)	2 (1)	