# HIV-Associated Lung Disease

## Case related questions.

**Scenario:**
A 49-year-old woman presents for evaluation of cough with abnormal chest imaging. She has a history of IV drug use and was diagnosed with HIV 1 year ago by routine screening. At diagnosis, her CD4 count was 145 cells/mm$^3$. Intereron gamma release assay (IGRA) testing was negative and her CXR was normal. She was initiated on a boosted protease inhibitor-based regimen (darunavir/ritonavir/tenofovir/emtricitabine), with eventual improvement in CD4 counts to >400 cells/mm$^3$.

Over the past 6 months, she noted increased dry cough and mild-moderate dyspnea on exertion that is relatively constant, along with fatigue. She denies hemoptysis, fevers, sweats, headache, rash, GI complaints, or weight loss. She is a Philadelphia native without significant travel history.

She had childhood asthma, which was not active in early adulthood. She would take inhaled fluticasone-salmeterol intermittently prn, but her prescription expired some years ago. She has an albuterol inhaler, which she has been taking more regularly over the past 6 months and thinks may help. She has smoked ~1 pack per day since her 20s.

She is afebrile, with SpO2 on RA = 94%. Her lung sounds are mildly diminished but otherwise clear. There is no wasting, rash, lymphadenopathy, or hepatosplenomegaly on exam.

**Question 1:** How would you approach the differential diagnosis for this case?

**Question 2:** Could this be an immune reconstitution phenomenon?

**Question 3:** How helpful is IGRAtesting in patients with HIV?

**Question 4:** What non-infectious processes should be considered?

**Scenario cont.:**
You obtain a high-resolution chest CT and PFTs. The chest CT shows bilateral hilar adenopathy with mid- & upper zone-predominant micronodular interstitial changes in a bronchovascular distribution. There are no significant ground glass or cystic changes; however, mild upper-lobe centrilobular emphysema is apparent.

PFTs demonstrate a mixed obstructive-restrictive pattern, with an FEV1 of 65% predicted, no bronchodilator reversibility, and DLCO 55% predicted.

**Question 5:** Based on your information, should you put her on other inhalers?

**Scenario cont.:**
You start the patient on an inhaled corticosteroid and long-acting beta-agonist (ICS/LABA), and she undergoes bronchoscopy.

The airways appear mildly inflamed. Transbronchial biopsies reveal non-caseating granulomas, with negative special stains/cytology and negative cultures from a bronchoalveolar lavage, consistent with a final diagnosis of sarcoidosis.

Given her symptoms and PFT impairment, you initiate the patient on prednisone at ~0.5 mg/kg/day. The patient is lost to follow-up for a period but returns 4 months later.

Her breathing and cough have improved, but now she displays Cushingoid features.

Repeat PFTs show improved FEV1 and FVC. A repeat CT chest shows improved peribronchovascular nodularity but a new 8mm ground glass nodule in the RUL. You begin to wean her steroids and plan for follow-up imaging.

**Question 6:** What is the impact of HIV infection on the epidemiology of lung cancer?

**Question 7**: The patient mentions that she has read online that HIV is a risk factor for pulmonary hypertension. What is this patient's risk of developing pulmonary arterial hypertension? How does ART impact this risk?

# HIV-Associated Lung Disease

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*Literature review current through June 2025*
*Last updated June 2025*

**Citation**

Maximous S, Cene-Kush C, Hinkle L, et al., HIV-Associated Lung Disease. In: Fritz J. Association of Pulmonary and Critical Care Program Directors Ambulatory Care Teaching Scripts. 2025. https://apccmpdscholars.org/tool/ambulatory-care-curriculum/.

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**Educational Objectives:**

1. Develop a general approach to the patient with Human Immunodeficiency Virus (HIV) presenting with pulmonary complaints in the ambulatory setting
2. Understand the changing epidemiology of classic Acquired Immunodeficiency Syndrome (AIDS)-defining illnesses with pulmonary involvement in the antiretroviral therapy (ART) era
3. Understand how HIV may impact the natural history of and risk for non-HIV-related pulmonary conditions

A 49-year-old woman presents for evaluation of cough with abnormal chest imaging. She has a history of IV drug use and was diagnosed with HIV 1 year ago by routine screening. At diagnosis, her CD4 count was 145 cells/mm$^3$. Intereron gamma release assay (IGRA) testing was negative and her CXR was normal. She was initiated on a boosted protease inhibitor-based regimen (darunavir/ritonavir/tenofovir/emtricitabine), with eventual improvement in CD4 counts to >400 cells/mm$^3$.

Over the past 6 months, she noted increased dry cough and mild-moderate dyspnea on exertion that is relatively constant, along with fatigue. She denies hemoptysis, fevers, sweats, headache, rash, GI complaints, or weight loss. She is a Philadelphia native without significant travel history.

She had childhood asthma, which was not active in early adulthood. She would take inhaled fluticasone-salmeterol intermittently prn, but her prescription expired some years ago. She has an albuterol inhaler, which she has been taking more regularly over the past 6 months and thinks may help. She has smoked ~1 pack per day since her 20s.

She is afebrile, with SpO2 on RA = 94%. Her lung sounds are mildly diminished but otherwise clear. There is no wasting, rash, lymphadenopathy, or hepatosplenomegaly on exam.

**Question 1: How would you approach the differential diagnosis for this case?**

Initial consideration of the degree of immunosuppression (i.e., CD4 count), as shown in Table 1, is a reasonable place to start. When cross-referenced with key symptoms and radiographic patterns, this approach can reliably narrow the differential.[1]

Geographical location should also be a consideration, as infectious diseases can vary significantly with geography. For example, tuberculosis (TB) is the most common pulmonary complication in HIV in Africa, while in the developing world, bacterial pneumonia is the most common.[2,3]

**Table 1.** Differential Diagnosis of Respiratory Disease Based on CD4 Count

| CD4 Count | Respiratory Illness |
|---|---|
| **Any** | Acute bronchitis<br>Bacterial pneumonia<br>Bronchogenic carcinoma<br>COPD<br>Lymphoma<br>Nonspecific Interstitial Pneumonia<br>Pulmonary Arterial Hypertension<br>Pulmonary Embolism<br>Sarcoidosis |
| **≤500 mm$^3$** | LIP (usually not until CD4 <350 mm$^3$)<br>Recurrent bacterial pneumonia<br>"Typical" pulmonary TB |
| **≤200 mm$^3$** | "Atypical" pulmonary TB & possibly disseminated infection<br>Cryptococcal pneumonia<br>PJP |
| **≤100 mm$^3$** | Pulmonary Kaposi sarcoma<br>Toxoplasma pneumonitis |
| **≤50 mm$^3$** | *Aspergillus* pneumonia<br>CMV pneumonia<br>Disseminated *Coccidioides*<br>Disseminated *Histoplasma*<br>Disseminated MAI<br>Lymphoma<br>*Nocardia*<br>*Rhodococcus* |

*Abbreviations: CMV, cytomegalovirus; COPD, Chronic Obstructive Pulmonary Disease; LIP, Lymphocytic Interstitial Pneumonia; MAI, Mycobacterium Avium Intracellulare; TB, Tuberculosis; PJP, Pneumocystis jirovecii pneumonia*

Adapted from Tokman S, Huang L. Evaluation of respiratory disease. *Clin Chest Med* 2013.

Endemic mycoses (histoplasmosis, blastomycosis, coccidioidomycosis) should be considered based on local risk and prevalence or relevant travel history. Clinical and imaging findings will offer valuable information and can be supplemented by laboratory tests. Various antigen tests (cryptococcal antigen, urine Histoplasma antigen, blastomyces antigen) in the right clinical context can be a simple starting point to evaluate suspected infection by endemic mycoses. High levels of blood beta-D-glucan have demonstrated a correlation with HIV-related PJP, although the precise role of this test remains to be defined.[6]

The following tables summarize the differential diagnosis when considered from a radiographic standpoint.[4,5]

**Table 2.** Radiologic Findings and Associated Differential Diagnoses in Patients with HIV Infection

| | **Consolidation** | **Ground-Glass Opacity** | **Cysts** | **Peribroncho-vascular Opacities** |
|---|---|---|---|---|
| Infectious Causes | - Bacterial<br><br>*CD4 <200 cells/mm$^3$*<br>- Fungal<br>- Mycobacterium | - Viral<br>- Atypical bacterium<br><br>*CD4 <200 cells/mm$^3$:*<br>- PJP<br><br>*CD4 <100 cells/mm$^3$*:<br>- CMV | *CD4 <200 cells/mm$^3$:*<br>- PJP | |
| Interstitial Lung Diseases | | - LIP<br>- NSIP | - LIP | - LIP<br>- Sarcoidosis |
| Neoplasms | - Lung Cancer<br>- Lymphoma | | | *CD4 <200 cells/mm$^3$*:<br>- Lymphagitic carcinomatosis<br>- Lymphoma<br>- Kaposi sarcoma |

*Abbreviations: CMV, cytomegalovirus; LIP, lymphocytic interstitial pneumonia; NSIP, nonspecific interstitial pneumonia; PJP, Pneumocystis jirovecii pneumonia*

Adapted from Lichtenberger JP et al. "What a Differential a Virus Makes: A Practical Approach to Thoracic Imaging Findings in the Context of HIV infection--Part 1, Pulmonary Findings," *Am J of Roentgenology* 2012; 198(6): 1295-304.

**Table 3.** Features of Pulmonary Nodules and the Associated Differential Diagnoses in Patients with HIV Infection

| | **Micronodules (<1 cm)** | **Macronodules (>1 cm)** | **Cavitary Lesions** |
|---|---|---|---|
| Infectious | Centrilobular ("tree-in-bud")<br>- Bacterial<br>- Viral<br><br>Miliary Distribution<br>- TB<br>- NTM<br>- Fungal<br>- Toxoplasma<br><br>*CD4 <200 cells/mm$^3$:*<br>- Mycobacterial<br>- Fungal | -Mycobacterial<br>-Fungal<br>-Septic emboli | -Bacterial pneumonia<br>-Bacterial abscess<br>-Mycobacterial<br>-Fungal<br>-Septic emboli |
| Non-infectious | Centrilobular ("tree-in-bud")<br>- LIP<br><br>Perilymphatic Distribution<br>- LIP | | |
| Neoplasms | - Metastatic disease<br><br>Perilymphatic Distribution<br>- Sarcoidosis<br>- Lymphangitic carcinomatosis | - Lymphoma<br>- Lung cancer<br>- Metastatic disease | -Necrotic carcinoma<br>-Lymphoma |

*Abbreviations: LIP, lymphocytic interstitial pneumonia; NTM, nontuberculous mycobacterium; TB, tuberculosis*

Adapted from Lichten Berger JP et al. "What a Differential a Virus Makes: A Practical Approach to Thoracic Imaging Findings in the Context of HIV infection--Part 1, Pulmonary Findings," *Am J of Roentgenology* 2012; 198(6): 1295-304.

### Question 2: Could this be an immune reconstitution phenomenon?

Possibly. IRIS (immune reconstitution inflammatory syndrome) can occur in the context of multiple pre-existing opportunistic infections, including TB and NTM, PJP, and Cryptococcus, as well as non-infectious conditions like Kaposi sarcoma. The timing of symptoms can be helpful; in one prospective study, the median onset of IRIS was 48 days (IQR 29-99 days) after ART initiation.[7] Fever is typical, and neurologic symptoms are common, particularly with cryptococcus and TB. Neurologic symptoms and abnormal chest imaging should prompt a directed evaluation for mycobacterial or fungal pathogens, including CSF analysis. When endemic mycoses present with pneumonia in HIV, it is usually in the context of disseminated disease. Lymphadenopathy with a *low-attenuation center* strongly suggests an infectious etiology (mycobacterial or fungal).[5]

In this case, the onset of symptoms (~6 months after ART initiation) is a bit long for IRIS, and the pre-treatment CD4 count is typically lower than this patient's (often <100 cells/mm$^3$, though preexisting TB infection is an exception). This, along with the patient's lack of fever, makes traditional IRIS less likely.

### Question 3: How helpful is the IGRA test in patients with HIV?

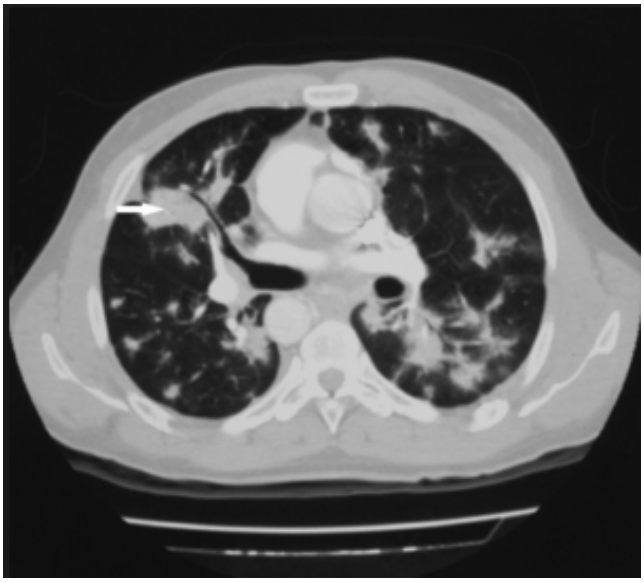
Interferon-gamma release assays (IGRAs) appear to have similar (imperfect) sensitivity compared with tuberculin skin testing (60-70%) for identifying TB infection. The T-SPOT TB

assay may be slightly more sensitive (62-81%) than the Quantiferon testing.[8] If the initial test was done with a CD4 count <200 cells/mm$^3$, some recommend repeating the test once the count surpasses this threshold on therapy.

**Question 4: What non-infectious processes should be considered?**

**Kaposi sarcoma (KS)** and **non-Hodgkin lymphoma (NHL)**[9] are two classic AIDS-defining malignancies; however, their incidence has declined in the ART area, corroborating a link between risk and level of immunosuppression. KS occurs largely in men who have sex with men and at very low CD4 counts. CT findings include peribronchovascular nodularity and thickening (classically, in a "flame" shape), interlobular septal thickening, and fissure nodularity; adenopathy can also be seen as shown in Figure 1. The parenchymal abnormalities tend to have a lower-lobe predominance, and lymph nodes may enhance. Pulmonary disease can exist in the absence of cutaneous disease

**Figure 1.** Kaposi Sarcoma



*Peribronchovascular flame shaped densities seen in Kaposi Sarcoma (white arrow).*

Image borrowed from Vummidi D, Siripurapu, R, Allen, C. Non-infectious pulmonary complications of HIV infection: A pictorial review. *European Society of Radiology*; 2008. Accessed March 18, 2025. https://epos.myesr.org/poster/esr/ecr2008/C-218

The incidence of NHL has decreased in the ART era. However, the link with a low CD4 count is perhaps less clear than with KS; thus, a relatively preserved CD4 count cannot rule out the possibility of lymphoma. Most HIV-related NHL is of the diffuse large B-cell type, many of which are associated with EBV infection. While not a likely consideration for this patient, primary effusion lymphoma is a rare type of NHL that is associated with HIV infection and can present in the pleural or pericardial space as well as the peritoneum.[9]

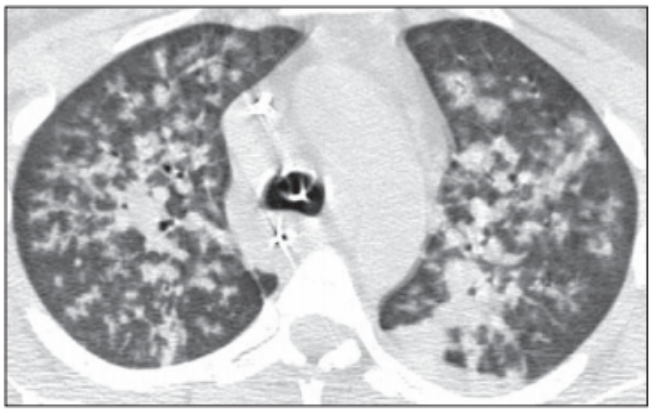
**Multicentric Castleman Disease (MCD)** has been reported in HIV. About 50% of HIV-associated cases will have mediastinal adenopathy (much rarer in non-HIV MCD). MCD is also associated with a LIP-like pattern of the lung parenchyma. Peripheral lymphadenopathy and splenomegaly are usually present, and fever is nearly universal. Our patient lacks these additional features, making this unlikely.[10]

**Scenario cont.:**
You obtain a high-resolution chest CT and PFTs. The chest CT shows bilateral hilar adenopathy with mid- & upper zone-predominant micronodular interstitial changes in a bronchovascular distribution. There is no significant ground glass or cystic changes; however, mild upper-lobe centrilobular emphysema is apparent.

PFTs demonstrate a mixed obstructive-restrictive pattern, with an FEV1 of 65% predicted, no bronchodilator reversibility, and DLCO 55% predicted.

**Lymphocytic interstitial pneumonia (LIP)** is the "classic" ILD associated with HIV infection, but it has been described most frequently in infants and children. Interestingly, LIP tends to be seen with higher CD4 counts, suggesting that a certain level of immune competency is required to drive the lymphocytic infiltration that characterizes the disease. HRCT findings include scattered ground glass opacities, nodules, and possibly cysts, often in a predominant lower-lobe distribution as seen in Figure 2. This remains a possibility in our case; however, bulky adenopathy is not expected in LIP.

**Figure 2.** LIP with Peribronchovascular Nodules



John P. Lichtenberger I, Sharma A, Zachary KC, et al. What a Differential a Virus Makes: A Practical Approach to Thoracic Imaging Findings in the Context of HIV Infection-- Part 1, Pulmonary Findings. *American Journal of Roentgenology*. 2012;198(6):1295-1304. doi:10.2214/ajr.11.8003

**Non-specific interstitial pneumonitis (NSIP)** was described frequently in the pre-ART era, though the definition of the disease has evolved over the years. The appearance of NSIP in HIV is similar to non-HIV patients. It also tends to occur with higher CD4 counts, though lower than that seen in LIP; however, there is a wide overlap.

**Sarcoidosis** is characterized by abnormalities in cell-mediated immunity and was considered rare in HIV in the pre-ART era; however, there has been a "resurgence" of this diagnosis, potentially related to ART-induced restoration of CD4-related immunity, allowing the disease to manifest in susceptible patients.[11]

*While considering non-infectious causes, never forget about PJP as a cause of a sub-acute pulmonary presentation – this can cause a variety of CT patterns, many of which overlap with the above non-infectious entities. The risk of PJP increases in patients with CD4 counts <200 cells/mm³, especially in the absence of adequate prophylaxis.*

Given the clinical context, sarcoidosis becomes your leading differential diagnosis. You discuss proceeding with bronchoscopy, and the patient agrees. She asks if anything can be done to make her feel better.

**Question 5: Based on your information, should you put her on other inhalers?**

The PFTs suggest an element of obstruction without bronchodilator reversibility, and the CT reveals emphysema in addition to the interstitial changes.

**Despite the increasing use of ART, people living with HIV (PLWH) have a higher risk of emphysema (OR 1.46), obstructive lung disease (OR 1.29), gas exchange impairment (OR 2.63), and a faster decline in pulmonary function compared to people living without HIV (PLWOH)**.[12,13] In one study, 23% of PLWH who smoke (without a history of pulmonary infections) had emphysema on either PFTs or CT scans compared with 2% of PLWOH (controls) matched for age and smoking history.[14] COPD can also present at an earlier age, and PLWH may be more likely to experience acute exacerbations of COPD.[15] Multiple cohorts have suggested a 15-20% prevalence of airway obstruction using typical spirometric criteria; associated factors include age, smoking, IV drug use, and history of PJP pneumonia. A 2024 systematic review and meta-analysis found that diffusion impairment was the most common pulmonary function abnormality in PLWH and frequently occurred with normal spirometry.[13] An abnormal DLCO was associated with increased mortality in PLWH even after controlling for emphysema and obstructive lung disease.[13] Therefore, DLCO should be measured in addition to spirometry as part of a first-pass work-up for PLWH.[13]

The link between HIV and asthma is less well-established. Still, in one study, 21% of HIV-infected persons carried a self-reported asthma diagnosis, compared with ~9% in the general population.[16]

Given that our patient's CT and PFT findings make sarcoidosis our leading differential, initiating an ICS/LABA would be reasonable as a first step for symptomatic control.[17,20] There are no HIV-specific guidelines for sarcoidosis management, so treatment generally mirrors the approach used in people without HIV. Therefore, in addition to starting an ICS/LABA, additional treatment with steroids and steroid-sparing agents should be considered given the pulmonary parenchymal involvement noted on her CT scan. Drug-drug interactions should always be kept in mind; a particular concern when selecting an ICS is the boosting effect of protease inhibitors on fluticasone, which can lead to frank Cushing syndrome or adrenal insufficiency.

Smoking cessation should always be recommended, especially in patients with HIV. A national cohort study suggested that the attributable risk of smoking-related death in PLWH is double that of PLWOH. PLWH who smoke tobacco lose more life years to smoking than to HIV infection.[13] Both behavioral and pharmacologic methods of smoking cessation are effective in patients with HIV, similar to other smokers. Varenicline is effective for smoking cessation and well tolerated in patients with HIV.[18]

**Scenario cont.:**
You start the patient on an ICS/LABA, and she undergoes bronchoscopy.

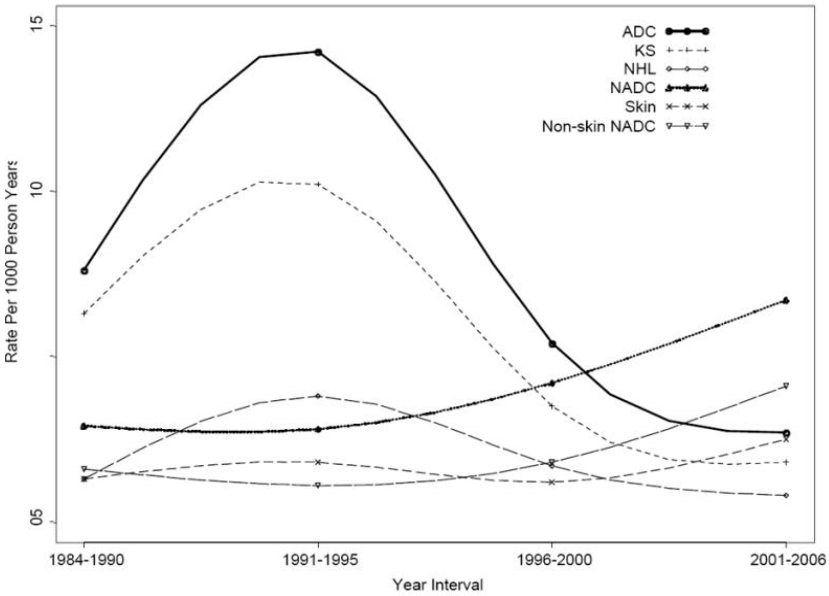
The airways appear mildly inflamed. Transbronchial biopsies reveal non-caseating granulomas, with negative special stains/cytology and negative cultures from a BAL – consistent with a final diagnosis of sarcoidosis.

Given the symptoms and PFT impairment, you initiate the patient on prednisone at ~0.5 mg/kg/day. The patient is lost to follow-up for a period but returns 4 months later. Her breathing and cough have improved, but now she displays Cushingoid features.

**Question 6: What is the impact of HIV infection on the epidemiology of lung cancer?**

The incidence of AIDS-defining malignancies, such as KS and NHL, has decreased in the ART era. However, the incidence of other cancers, including lung, has increased as shown in Figure 3.[19,4]

**Figure 3.** Cancer Incidence Over Time in Patients with HIV Infection




*Abbreviations: ADC, AIDS defining cancer; KS, Kaposi sarcoma; NADC, non-AIDS defining cancer; NHL, Non-Hodgkin's lymphoma.*

Borrowed from Crum-Cianflone N, et al. "Trends in the Incidence of Cancers among HIV-Infected Persons and the Impact of Antiretroviral Therapy: A 20-Year Cohort Study," *AIDS* 2009; 23(1): 41-50.

While it is true that smoking prevalence is higher among PLWH, similar to the COPD population, the presence of HIV infection appears to independently increase the risk of developing lung cancer (2 to 4-fold increased risk) after adjusting for tobacco use. Low CD4 counts and prior pneumonia are associated with increased lung cancer risk.[20] Cigarette smoke, HIV infection, and chronic inflammation can increase oxidative stress, leading to oxidative DNA damage. This has been postulated as a possible mechanism for increased cancer risk in PLWH. Early initiation of ART has been associated with reduced risk of lung cancer in this population.[21]

There are limited data on lung cancer screening for people living with HIV, given that PLWH were not included in the NLST. The American Thoracic Society has advocated for more research to determine whether lung cancer screening for PLWH who do not have tobacco exposure is warranted.[22,4] For patients with a sufficient smoking history, standard lung cancer screening criteria should be applied, and the presence of HIV as an independent risk factor should be discussed when counseling patients on the risks/benefits of screening. Additionally, it should be noted that there will likely be a higher percentage of incidental findings in this population than in patients without HIV.

**Question 7: The patient mentions that she has read online that HIV is a risk factor for pulmonary hypertension. What is this patient's risk of developing pulmonary arterial hypertension? How does ART impact this risk?**

PAH has an incidence of 0.5% in HIV. PAH occurring in HIV[5] appears histologically identical to idiopathic PAH, placing it within Group 1 of the World Health Organization classification system.[23] There has been no consistent association with CD4 count, which suggests that the level of immune suppression (or treatment thereof) does not modify the risk of PAH. Risk factors associated with the development of PAH include female sex, IV drug use, detectable HIV viremia, and co-existing HCV infection.[23]

The presentation of PAH in HIV and the diagnostic approach is similar to other forms of PAH. Patients with HIV are vastly under-represented (or absent) from the pivotal trials of available PAH agents. Drug-drug interactions are important to consider; protease-inhibitors can boost the serum concentrations of some ERAs (endothelin receptor antagonists) and PDE5 inhibitors. Ambrisentan is the ERA with the fewest interactions. Both PDE5-Is (though perhaps less with tadalafil) and riociguat require close follow-up due to these boosting effects. Of the available oral agents, no interactions exist with the new prostacyclin derivatives (oral treprostinil and selexipag).

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# HIV-Associated Lung Disease

## Post-Test Related Case Related Questions

1. A 36-year-old woman presents to your clinic with worsening dyspnea over the last 2-3 months. She was diagnosed with HIV two years ago. She has taken her ART inconsistently; the most recent CD4 count was 156 cells/$mm^3$. She reports a low-grade fever (Tm 99-100°C) in the last week and a cough productive of white-yellow sputum. She denies recent travel or sick contacts. She is a non-smoker. She is not currently taking any antibiotics for prophylaxis. Given her symptoms, you are concerned about an infectious process and order a chest CT. She has no prior imaging in the system or PFTs. While awaiting the CT scan results, which of the following would you consider as diagnostic possibilities for this patient's condition based on her CD4 count?
    a. Aspergillus pneumonia
    b. Bacterial Pneumonia
    c. CMV pneumonitis
    d. PJP Pneumonia
    e. B & D

2. A 55-year-old man with a history of well-controlled HIV (CD 400 cells/$mm^3$, viral load undetectable) and COPD comes to your office for a routine follow-up. Reviewing his medication list, you note that he is on ART, including a protease inhibitor. To date, he has been maintained on albuterol as needed alone for the management of his COPD, but he notes that he feels that his symptoms have been worse in the last 6 months. You are considering starting him on a long-acting inhaler to help improve his symptom management. Which of the following inhalers should be avoided in this patient?
    a. Fluticasone
    b. Formoterol
    c. Tiotropium
    d. All are safe

3. A 35-year-old woman with a history of well-controlled HIV (CD4 480 cells/$mm^3$, undetectable viral load) and hepatitis C infection comes to your office with progressive dyspnea. She had a chest CT with no parenchymal abnormalities. Her PFTs demonstrated normal spirometry. DLCO was not performed. You are considering possible etiologies for her dyspnea and the possibility of pulmonary hypertension (PH). Which of the following statements is true regarding pulmonary hypertension in this patient?
    a. Because her HIV is well-controlled, her risk for HIV-associated PH is reduced
    b. Her history of HCV is an additional risk factor for HIV-associated PH
    c. PH is common in HIV-infected patients and thus is a likely culprit of her dyspnea

# HIV-Associated Lung Disease

## Post-Test Related Case Related Questions with Answer Key

1. A 36-year-old woman presents to your clinic with worsening dyspnea over the last 2-3 months. She was diagnosed with HIV two years ago. She has taken her ART inconsistently; the most recent CD4 count was 156 cells/mm$^3$. She reports a low-grade fever (Tm 99-100°C) in the last week and a cough productive of white-yellow sputum. She denies recent travel or sick contacts. She is a non-smoker. She is not currently taking any antibiotics for prophylaxis. Given her symptoms, you are concerned about an infectious process and order a chest CT. She has no prior imaging in the system or PFTs. While awaiting the CT scan results, which of the following would you consider as diagnostic possibilities for this patient's condition based on her CD4 count?
    a. Aspergillus pneumonia
    b. Bacterial Pneumonia
    c. CMV pneumonitis
    d. PJP Pneumonia
    e. B & D

2. A 55-year-old man with a history of well-controlled HIV (CD 400 cells/mm$^3$, viral load undetectable) and COPD comes to your office for a routine follow-up. Reviewing his medication list, you note that he is on ART, including a protease inhibitor. To date, he has been maintained on albuterol as needed alone for the management of his COPD, but he notes that he feels that his symptoms have been worse in the last 6 months. You are considering starting him on a long-acting inhaler to help improve his symptom management. Which of the following inhalers should be avoided in this patient?
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