Endobronchial Lesions in HIV-infected Individuals*

Marc A. Judson, M.D., F.C.C.P.; and Steven A. Sahn, M.D., F.C.C.P.

Endobronchial manifestations of HIV infection are rare. The endobronchial appearance and clinical presentation of these lesions may suggest the correct diagnosis. Establishing an appropriate differential diagnosis at the time of visualization of the endobronchial lesion is important because some lesions require specific biopsy techniques or special stains. The bronchoscopist must consider the risks vs benefits of biopsy when confronted with an endobronchial lesion. With the notable exception of pseudomembranous necrotizing tracheobronchial aspergillosis, there are no specific endobronchial lesions associated with HIV infection which increase the risk of complications when they are biopsied. Although EKS is a vascular lesion and an early case report suggested that endobronchial biopsy might result in excessive bleeding, this complication was not observed in two subsequent series. Fortunately, a presumptive diagnosis of EKS can usually be made without biopsy by the characteristic appearance of the lesion. EKS is the most common endobronchial lesion associated with HIV infection; however, its incidence will probably decline as the incidence of KS declines. Many of the other endobronchial lesions described herein have been reported recently. We suspect these and other lesions will be found more frequently, as the epidemic of HIV continues to evolve.

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AFB=acid-fast bacilli; ARC=AIDS-related complex; EKS=endobronchial Kaposi’s sarcoma; EBV=Epstein-Barr virus; ETB=endobronchial tuberculosis; KS=Kaposi’s sarcoma

The discovery of an endobronchial lesion during bronchoscopy in a patient infected with the human immunodeficiency virus (HIV) is a rare and often surprising finding. Knowledge of the clinical presentation and endobronchial appearance of these lesions is important because several require specific biopsy techniques. In addition, the presence of some of these lesions has prognostic significance.

The purpose of this review is to describe the clinical presentation, endobronchial appearance, and diagnostic techniques of the various endobronchial lesions known to be associated with HIV infection. These lesions are listed in Table 1.

**Kaposi’s Sarcoma**

Kaposi’s sarcoma (KS) occurs frequently in HIV-infected individuals who are homosexual or bisexual, and its presence establishes the diagnosis of the acquired immunodeficiency syndrome (AIDS). The proportion of AIDS cases with KS appears to be declining, possibly because the proportion of AIDS cases with homosexuality as a risk factor is declining.

Thoracic KS usually involves the lung parenchyma, pleura, intrathoracic lymph nodes, and the airways.1 KS of the thorax has been found in up to 49 percent of patients with cutaneous KS.2-4 and it rarely occurs without KS involvement of another site.5-7 Fouret and coworkers5 found the mean duration of cutaneous disease before the diagnosis of bronchopulmonary KS was 10 months.7

Naidich and coworkers8 reviewed 114 patients with bronchopulmonary KS and found the incidence of endobronchial Kaposi’s sarcoma (EKS) to be 28 percent. Presenting complaints of patients with EKS include dyspnea and cough.6,9 Life-threatening upper airway obstruction may occur.10-11 Fever is common, even in the absence of a concomitant opportunistic infection.6,9 Auscultation of the chest may reveal wheezing or stridor.12 Almost all patients with EKS have lesions in the oropharynx.2,5,9

Chest radiographs with EKS are nonspecific, and the bronchoscopic extent of EKS does not correlate with the degree of radiographic abnormality.5 The chest radiograph may show perihilar infiltrates, diffuse nodular infiltrates, diffuse interstitial infiltrates, pleural effusions, or it may be normal.5,9,12

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*From the Medical University of South Carolina, Division of Pulmonary and Critical Care Medicine, Charleston. Manuscript received June 9, 1993; revision accepted November 29.
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<table>
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<th>Disease</th>
<th>Presentation</th>
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<th>Endobronchial Appearance</th>
<th>Biopsy Technique</th>
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<tr>
<td>Kapoši's sarcoma</td>
<td>Skin lesions; oropharyngeal lesions; fever</td>
<td>Peripheral infiltrates</td>
<td>Erythematous, Macular</td>
<td>Not required if appearance and clinical presentation characteristic</td>
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<td></td>
<td></td>
<td>Diffuse nodular infiltrates</td>
<td>Violaceous, Papular, not discrete in distal airways</td>
<td>Highest yield from lesions draping distal airway carinae</td>
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<td>Intestinal infiltrates</td>
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<td>Send biopsy for AFB fungal stain and culture</td>
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<td></td>
<td>Pleural effusions</td>
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<tr>
<td>Mycobacterium tuberculosis, Atypical mycobacteria</td>
<td>May occur on appropriate antimycobacterial therapy</td>
<td>Hilar or mediastinal adenopathy adjacent to endobronchial lesion</td>
<td>Simulates bronchogenic carcinoma</td>
<td>Airway obstruction</td>
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<td>Send biopsy and BAL for AFB stain and culture</td>
</tr>
<tr>
<td>Aspergillus airway disease</td>
<td>Fever, Cough, Dyspnea; Hemoptysis</td>
<td>Localized or diffuse infiltrates</td>
<td>Blackened ulcerations, creamy-white plaques, pseudomembranes, exophytic, mucoid impaction</td>
<td>Send biopsy and BAL for AFB stain and culture</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>If pseudomembranes seen, biopsy potentially dangerous and mechanical attempts to enlarge airway very dangerous</td>
</tr>
<tr>
<td>Non-Hodgkin's Lymphoma</td>
<td>Evidence of past EBV infection, Dyspnea, wheezing; Fever</td>
<td>Subtle tracheal abnormalities</td>
<td>Exophytic, friable</td>
<td>Rigid bronchoscopy required</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Adenopathy (adjacent to bronchial lesion)</td>
<td></td>
<td>Endobronchial biopsy suggestive</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>&gt;20 pack-year smoking history</td>
<td>Peripheral nodule/infiltrate</td>
<td>Probably similar to non-HIV patients</td>
<td>No difference between HIV+ and HIV-</td>
</tr>
<tr>
<td></td>
<td>Present at younger age than non-HIV lung cancer patients</td>
<td>Hilar mass</td>
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<tr>
<td>Purulent (bacterial) bronchitis</td>
<td>Fever, cough, dyspnea</td>
<td>Bronchiectasis</td>
<td>Mucosal edema, erythema, friability, purulent secretions</td>
<td>BAL culture for bacteria: commonly positive for strept species, staph species, or H. Influenzae</td>
</tr>
<tr>
<td>Bacillary angiomatosis</td>
<td>Violaceous skin papules, fever, cough</td>
<td>Parenchymal pulmonary infiltrates</td>
<td>Polypoid lesions</td>
<td>Send biopsy for Warthin-Starry silver stain, electron microscopy</td>
</tr>
<tr>
<td>Bacterial tracheitis*</td>
<td>Fever, stridor, upper airway obstruction</td>
<td>Chest radiograph: normal</td>
<td>Obstructing tracheal mass</td>
<td>Rule out CMV tracheitis: send biopsy for CMV stain, culture</td>
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<tr>
<td>CMV tracheitis*</td>
<td>Dyspnea, stridor, upper airway obstruction</td>
<td>Tracheal mass</td>
<td>Obstructing tracheal mass</td>
<td>Send biopsy for CMV stains and cultures</td>
</tr>
<tr>
<td>Actinomyicosis*</td>
<td>Dyspnea, fever, cough</td>
<td>Localized infiltrate</td>
<td>Exophytic obstructing mass</td>
<td>Endobronchial biopsy, BAL gram stain and cytology</td>
</tr>
<tr>
<td>Pneumocystis carinii*</td>
<td>Fever, cough, hemoptysis</td>
<td>Cavitary mass</td>
<td>White endobronchial lesion completely obstructing a segmental orifice</td>
<td>Send biopsy for PC stain. BAL and TBB may be negative</td>
</tr>
<tr>
<td>Granular cell myoblastoma*</td>
<td>Dyspnea, wheezing, fever</td>
<td>Bilateral hilar adenopathy</td>
<td>Plaque-like lesions in central airways</td>
<td>Endobronchial biopsy diagnostic</td>
</tr>
</tbody>
</table>

*Single case reported. Italics indicate common presentation or common biopsy technique.

Approximately half the patients with EKS have normal pulmonary function, while half demonstrate an obstructive ventilatory defect. The endobronchial appearance of KS is almost always erythematous in contrast to the violaceous appearance of the skin and oropharyngeal lesions (Fig 1). The lesions may be macular or papular. Lesions tend to be discrete in the trachea, while
they may appear as diffuse confluent hyperemic areas (Fig 2) in distal airways.6 Careful examination of the bronchial tree is essential, since small focal areas of erythema may be overlooked.7 These EKS lesions are usually numerous and widespread throughout the tracheobronchial tree.5,13 Pozniak and coworkers reported 75 percent of their patients with EKS had more than 15 visible lesions.13

Knowledge of the bronchoscopic appearance of EKS is essential, since a presumptive diagnosis can be made based on the characteristic appearance of the lesions.1 Biopsy probably has clinical utility in KS patients with uncharacteristic endobronchial lesions who have an abnormal chest radiograph or pulmonary symptoms. Biopsy may be necessary in the unusual patient with EKS who has no other known organ involvement with KS. Although an early report suggested that endobronchial biopsy of EKS might result in excessive bleeding,14 this complication is rare.9,15

Proper endobronchial biopsy technique of EKS may be important in increasing the diagnostic yield. Ideal locations for biopsy of EKS lesions are at carinae subdividing segmental orifices.9 Although such EKS lesions are usually not as discrete as lesions in the proximal airways, the former sites are preferred because the angle of the forceps at the moment of biopsy is less acute than in the trachea or mainstem bronchus. In addition, a lesion at a small bronchial division has a better fit between the forceps jaw than a lesion in the trachea or mainstem bronchus. Using this technique, Hamm and associates5 were able to obtain diagnostic biopsies of EKS through a flexible bronchoscope in five of seven patients presumed to have EKS by endobronchial inspection, representing an improvement in diagnostic yield compared to previous series.16,17 In our experience, the most difficult aspect of the biopsy technique is the identification of an EKS lesion in this desired location, because such lesions often appear as subtle erythematous patches that may be mistaken for bronchoscope-induced trauma; EKS lesions should be suspected in these cases if the patient has risk factors for EKS, such as a history of KS (especially oropharyngeal KS), evidence of airway obstruction, or a chest radiograph that is suggestive of KS.

The prognosis of patients with EKS is poor. Miller and associates5 reported a median survival of less than 5 months. Gill and coworkers3 found that 60 percent of their EKS patients had a response to chemotherapy; the median survival of the respondents was 10 months vs 6 months in the non-responders.3 Palliation with radiation therapy has been recommended,18 and Nd:YAG laser has been useful.19

**Tuberculosis**

The number of cases of tuberculosis reported annually in the United States has increased since 1986, reversing a 40-year trend. This unprecedented resurgence of tuberculosis is largely related to the HIV epidemic.20

There are theoretical reasons to suspect that an endobronchial location should be more frequent in tuberculosis patients infected with the HIV virus.
First, endobronchial tuberculosis (ETB) is a frequent accompaniment of primary tuberculosis and it is thought that the defect in cell-mediated immunity caused by HIV results in a higher incidence of primary tuberculosis. Second, one common mechanism of development of ETB is by direct airway infiltration from an adjacent tuberculosis mediastinal lymph node, and chest radiographs of tuberculosis patients with HIV infection frequently reveal hilar and/or mediastinal adenopathy.

However, despite these theories, ETB rarely has been reported in HIV-infected patients. Kennedy and coworkers reported no cases of endobronchial tuberculosis among 67 HIV-infected tuberculosis patients who underwent bronchoscopy. We recently reviewed the medical literature and found only four reports of ETB in an HIV-infected patient and seven reports of endobronchial disease related to atypical mycobacteria; Mycobacterium avium complex in three, Mycobacterium kansasii in three, and Mycobacterium fortuitum in one. All patients had either AIDS, ARC, or a CD4 count under 300/min. The appearance of the endobronchial lesion varied, as did the lobar segments involved. The endobronchial lesions often were mistaken for bronchogenic carcinomas and frequently completely obstructed segmental airways. Patients with endobronchial Mycobacterium kansasii infection had obstruction of a lobar bronchus. Although all three were described as “responding favorably” to three- or four-drug chemotherapy because their mycobacterial infections did not progress clinically, their chest radiographs continued to show lobar infiltrates. The patient with Mycobacterium fortuitum had right middle lobe syndrome; a chest radiograph showed right middle lobe collapse, bronchoscopic exam revealed inflammation of the bronchus intermedius without a distinct endobronchial lesion, and pathologic specimens of resected lung revealed a fibrotic right middle lobe with destruction of the middle lobe bronchus by caseous nodes that had extended into the airways.

Several generalizations can be made concerning these patients. First, 8 of 11 had evidence of hilar or mediastinal adenopathy which was adjacent to the endobronchial lesion. These lymph nodes were proven to be involved with mycobacteria in the two instances where lymph nodes were sampled. This suggests that the mechanism involved in the development of these lesions is erosion of hilar lymph nodes into a bronchus. Second, all the patients with ETB had sputum smears negative for acid-fast bacilli (AFB). This is consistent with a recent study of 20 non-HIV patients with ETB in which 85 percent were AFB smear-negative or could not produce sputum. These authors postulated that proximal endobronchial granulation tissue might prevent caseous AFB-positive secretions from being expectorated. Whatever the mechanism, it is clear that a negative AFB smear of sputum does not exclude ETB. Third, endobronchial disease progressed in three of the patients receiving appropriate therapy. This is a known phenomenon thought to be the result of a hypersensitivity reaction related to the killing of mycobacteria and release of mycobacterial antigens after initiation of chemotherapy. Fourth, in two of these patients residual airway stenosis was documented, a well-recognized sequela of ETB.

One explanation for the low incidence of ETB in HIV-infected patients is that fiberoptic bronchoscopy is not indicated in a patient with ETB if acid-fast bacilli are detected on sputum smear; therefore, ETB may go undetected. We think this explanation is insufficient because it fails to explain the low incidence of ETB in HIV-infected patients compared to HIV-negative tuberculosis patients, especially since HIV-infected patients with signs or symptoms of pulmonary disease frequently undergo fiberoptic bronchoscopy to exclude a variety of pulmonary pathogens.

Because chest radiographs of tuberculosis patients with HIV infection frequently reveal hilar and/or mediastinal adenopathy, extrinsic airway compression may frequently occur. Although mediastinal or hilar adenopathy in patients infected with...
AIDS is commonly the result of tuberculosis, it may occur with KS, lymphoma, bronchogenic carcinoma, fungal infections, and atypical mycobacterial infections. Therefore, an attempt should be made to diagnose these extrinsic lesions definitively. Mycobacterium tuberculosis has been cultured by endobronchial needle biopsy of an AIDS patient with extrinsic airway compression from a tuberculous mediastinal node. Tuberculous mediastinal lymph nodes are usually of low density (necrotic) on chest computerized tomography (CT), an appearance that should suggest the diagnosis of tuberculosis.

**Aspergillus Airways Disease**

Invasive tracheobronchitis from Aspergillus species has been reported in several HIV infected patients. Kemper et al. described four AIDS patients with ulcerative and plaque-like tracheobronchitis from Aspergillus. Common presenting symptoms included fever, dyspnea, and cough. CD4 counts were less than 100/mm³ in the three patients tested. Two of four had an absolute neutrophil count less than 1,000/mm³. Two of four had pulmonary infiltrates on chest radiographs, while the other two had normal film findings. Bronchoscopic examination revealed 2 mm to 1.5 cm blackened, necrotic bronchial ulcerations and 2 to 3 mm raised creamy white plaques. One patient had a 1.0 cm exophytic lesion involving the mucosa at the juncture of two lobar bronchi. Endobronchial biopsies all revealed acute and chronic inflammation with evidence of mucosal invasion by Aspergillus. Fungal cultures or stains revealed Aspergillus species in all cases, and cultures from bronchoalveolar lavage in all patients grew Aspergillus. All patients were treated with amphotericin preceded or followed by a course of therapy with itraconazole. Two of four patients had a clinical response.

Three AIDS patients with obstructing bronchial aspergillosis have been described. All presented with progressive cough, and two were witnessed to cough up fungal casts of the airways. Chest pain, dyspnea, fever, and hemoptysis were each seen in two patients. Chest radiographs showed bilateral lower lobe infiltrates in all three. Microscopic examination and culture of sputum casts were diagnostic in all three. Bronchoscopy was normal in one patient, revealed mucoid impaction with multiple fungal casts in the second, and showed the formation of a pseudomembrane 2 weeks before death in the third. Chemotherapy with itraconazole resulted in a clinical response in two patients. The patient with endobronchial pseudomembrane formation was treated only after this endobronchial lesion was discovered and he subsequently died of disseminated Aspergillus infection.

Pseudomembranous necrotizing bronchial aspergillosis has been reported in four additional HIV-infected patients. All developed wheezing and dyspnea and died of respiratory failure. An autopsy of one of these patients revealed a pseudomembrane that completely covered the mucosa of the lower trachea, mainstem, lobar, segmental, and subsegmental bronchi. Although the lumens of the trachea, mainstem, and lobar bronchi were patent, there was progressive luminal narrowing with occlusion of several subsegmental branches. Histologically, the necrotic bronchial mucosa was replaced by a pseudomembrane, which consisted primarily of Aspergillus hyphae. The necrosis was transmural and extended for a limited extent into peribronchial tissues. Hyphal invasion and fibrin thrombi were present in the peribronchial blood vessels. Necrotizing tracheobronchial aspergillosis with airway obstruction has since been described in two additional immunocompromised patients. One of these patients died from uncontrollable hemorrhage during an attempt to remove a large obstructing mycetoma bronchoscopically. The authors suggest that bronchoscopy should be performed only for diagnosis and that manipulation of mycetomas is potentially dangerous because necrosis could extend transmurally. Therefore, it may be in the best interest of the patient not to pursue endobronchial therapy.

**Lymphoma**

There is an increased incidence of non-Hodgkin lymphomas in HIV-infected individuals. The incidence of these malignancies rises exponentially with increasing duration of HIV infection. The role of Epstein-Barr virus (EBV) in the development of these lymphomas has been strongly suspected. It is thought that T-cell suppression by HIV allows EBV-infected B lymphocytes to proliferate, enhancing the risk of chromosome translocation that could lead to activation of oncogenes.

Thoracic involvement of non-Hodgkin lymphoma is rare with HIV infection. In most large series, the incidence of thoracic involvement has varied from 0 to 25 percent. Two cases of endobronchial lymphoma (EL) have been reported in HIV-infected patients. Both patients presented with cough, dyspnea, fever, wheezing, and low CD4 counts (50 and 90/mm³). Both had tracheal involvement: in the upper trachea and at the main carina (Fig 4a). In both patients, the chest radiograph revealed subtle tracheal abnormalities; and a rigid bronchoscope was required to make the diagnosis, as endobronchial biopsies obtained through a flexible bronchoscope were inadequate. In one
served between HIV-seropositive and HIV-seronegative patients (0.09 percent vs 0.15 percent, respectively).\textsuperscript{50} Braun and colleagues\textsuperscript{62} suggested a 14-fold increased risk of lung carcinoma in HIV-infected patients based on the frequency of lung cancer detected in a population of known HIV-positive individuals. However, the estimation has been criticized because no adjustment was made for age, sex, race, or smoking status.\textsuperscript{63}

Although lung cancer may not be more frequent in HIV-infected individuals, its clinical course appears to be different. Fraire and Awe\textsuperscript{63} reviewed 21 cases of lung carcinoma associated with HIV infection reported between 1983 and 1991. Only one patient was a nonsmoker (smoking history was not reported in four). Although the age of the patients was not reported in all cases, at least half were under age 40. Patients smoked 20 to 30 pack-years in the five cases in which detailed smoking histories were reported. Eleven lung cancers (50 percent) were adenocarcinoma, six (27 percent) were small-cell carcinoma, three (13.5 percent) were squamous cell carcinomas, and one (4.5 percent) was an adenosquamous carcinoma. Ten of the 11 cases reporting data on staging with non-small cell carcinoma had stage III or stage IV disease. Sridhar and coworkers\textsuperscript{64} performed a retrospective case-control study involving 19 HIV-positive lung cancer patients. Sixteen of the 17 HIV-positive patients reporting a smoking history were smokers. The median amount of smoking was 60 pack-years. Compared to an HIV-negative historical control group, the HIV-positive lung patients were younger (median age 47 years vs 61 years, \(p<0.02\)) and had a shorter survival (median 3 months vs 10 months, \(p<0.05\)). There was no significant difference in the stage of disease at presentation. There was a trend toward a greater incidence of adenocarcinoma in the HIV-positive patients. The above data suggest that lung carcinoma in HIV-positive patients occurs at a younger age, is more aggressive, has smoking as a necessary risk factor, and has a greater incidence of adenocarcinoma than in HIV-negative lung cancer patients.

Presently, there are inadequate data to determine if there is a different incidence of endobronchial lesions in HIV-positive lung cancer patients with lung cancer compared to HIV-negative patients. In the series of Fraire and Awe,\textsuperscript{63} where the location of the tumor was described, three of the ten patients had an endobronchial lesion. Several of these patients had adenocarcinomas which tend to be peripheral. Interestingly, this series included two patients with small-cell lung carcinoma who had no endobronchial lesion identified on bronchoscopy.\textsuperscript{31,65}

**Lung Carcinoma**

Malignancies associated with HIV infection include KS, non-Hodgkin lymphoma, and several types of squamous cell carcinomas.\textsuperscript{30,61} It is unclear whether lung cancer is more frequent in HIV-infected individuals. In a recent study of 1,701 hemophiliacs followed for several years, no difference in the development of lung cancer was observed between HIV-seropositive and HIV-seronegative patients (0.09 percent vs 0.15 percent, respectively).\textsuperscript{50} Braun and colleagues\textsuperscript{62} suggested a 14-fold increased risk of lung carcinoma in HIV-infected patients based on the frequency of lung cancer detected in a population of known HIV-positive individuals. However, the estimation has been criticized because no adjustment was made for age, sex, race, or smoking status.\textsuperscript{63}

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OTHER MISCELLANEOUS ENDOBRONCHIAL LESIONS

Other endobronchial lesions in HIV infected patients have been reported in the literature. Purulent bronchitis has been observed in 19 of 422 HIV-infected patients who underwent bronchoscopy because of suspicion of opportunistic infection.\textsuperscript{66} Mucosal edema, erythema, friability and purulent secretions were seen in the airways. Fourteen of the 19 had opportunistic infections excluded by bronchoscopy. All 14 patients presented with fever, dyspnea, and cough. Bronchiectasis was detected on chest radiograph in seven (50 percent) of the patients, \textit{S viridans} and \textit{H influenzae} were commonly isolated from bronchoalveolar lavage (BAL), and ten of these patients had a rapid clinical response to antibiotic therapy (trimethoprim-sulfamethoxazole, penicillin, or ampicillin) on average of 2 days. Three patients developed adult respiratory distress syndrome and two of these died. Patients with purulent bronchitis had a significantly higher percentage of neutrophils in BAL (mean 56 percent) than HIV-infected patients with \textit{Pneumocystis carinii} pneumonia (mean 7 percent).

Bacterial tracheitis presenting as fever, stridor, and eventually life-threatening upper airway obstruction has been reported in an HIV-infected patient.\textsuperscript{67} Emergency fiberoptic bronchoscopy revealed an intratracheal mass originating from the posterior tracheal wall that obstructed 80 percent of the lumen. Gram stain and culture of endotracheal biopsies revealed \textit{Staphylococcus aureus} and \textit{Pseudomonas aeruginosa}. The patient required intubation and eventually responded to antibiotics.

Tracheitis with central airway obstruction from cytomegalovirus (CMV) has also been reported in a homosexual patient with AIDS.\textsuperscript{68} He presented with dyspnea and expectoration of foul-smelling sputum. Stridor was noted on physical examination. Chest radiographs and CT showed a mass encircling the trachea. Flow-volume loop demonstrated an extrathoracic obstruction. A red exophytic mass with severe ulcerations was seen on bronchoscopy that almost totally occluded the distal trachea. Mucosal biopsies of the mass showed intranuclear and intracytoplasmic inclusions in numerous cells and tracheal washings grew CMV. The patient had no previous history of CMV infection. He was treated with ganciclovir, and a follow-up bronchoscopy 3 weeks later showed both the mass and tracheal obstruction had significantly decreased.

Bacillary angiomatosis (BA), a vascular proliferative disorder usually involving the skin and peripheral lymph nodes in HIV-infected individuals,\textsuperscript{69} has been reported with associated endobronchial lesions.\textsuperscript{70,71} Thought to be caused by the organism \textit{Rochalimaea henselae}, BA can be demonstrated on Warthin-Starry silver stains.\textsuperscript{72} Both reported patients had cutaneous KS and presented with fever, chills, and cough. Chest radiographs showed bilateral interstitial infiltrates with mediastinal adenopathy in one case and right-sided alveolar/interstitial infiltrates with bilateral pleural effusions in the other. Bronchoscopy in both cases revealed small polypoid lesions in the airways (Fig 5) that were described as “pink” and “friable.” Warthin-Starry staining of these endobronchial biopsies revealed the organisms that cause BA.

\textit{Pneumocystis carinii} (PC) infection presenting as an endobronchial mass has been reported without any evidence of diffuse parenchymal involvement.\textsuperscript{73} The patient developed fever, cough, and hemoptysis while being treated for pulmonary tuberculosis. A chest radiograph revealed a new, large, cavitary mass in the lingula. A large white endobronchial mass was seen at bronchoscopy completely obstructing the lingula. Biopsy of the mass was remarkable for sheets of PC cysts; there was no evidence of neoplasm or granuloma, and mycobacterial stains and cultures were negative. Bronchoalveolar lavage and transbronchial biopsies were negative for AFB and PC. A gallium lung scan showed uptake only in the area of the endobronchial mass. The patient was treated with trimethoprimsulfamethoxazole, but switched to pentamidine isethionate because of granulocytopenia. Follow-up bronchoscopy 3 weeks later showed the mass to be slightly smaller. The patient died 2 months later from progressive neurologic deterioration. Chest radiographs obtained over this period failed to show significant improvement.

\textbf{FIGURE 5.} Polypoid endobronchial lesion in a patient with bacillary angiomatosis (From Slater, LN Min KW. Chest 1992; 102:972-74, reproduced by permission)
Endobronchial actinomycosis has recently been reported in an AIDS patient. The patient was a homosexual man who presented with dyspnea, fever, and a non-productive cough. His admission chest radiograph was without infiltrates, and he underwent bronchoscopy on the 10th hospital day for persistent fever and development of a lingular infiltrate. Bronchoscopy demonstrated a yellow-white, exophytic mass partially obstructing the left mainstem bronchus. Endobronchial biopsies demonstrated necrotic material, but Gram stain and cytologic studies of endobronchial secretions revealed masses of Gram-positive branching filaments and basophilic Actinomyces granules. Therapy with penicillin resulted in partial resolution of the infiltrate. A repeat bronchoscopy 10 days later demonstrated partial resolution of the endobronchial lesion, although Actinomyces organisms were again recovered from bronchial washings. The patient eventually died from cryptococcal meningitis.

Endobronchial involvement from a granular cell myoblastoma has been reported in a female intravenous drug abuser infected with HIV. Presenting signs and symptoms included dyspnea, fever, and wheezing. A chest radiograph showed bilateral hilar adenopathy, and chest CT revealed small endobronchial lesions in both mainstem bronchi. At bronchoscopy, white plaque-like lesions of both mainstem bronchi and orifices of the upper lobes were seen. Endobronchial biopsies were diagnostic.

ADDENDUM

Since acceptance of this article, a case of an endobronchial mass from *Nocardia asteroides* in an HIV-infected individual has been reported.

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