Tracheoesophageal Fistula in an HIV-1-Positive Man Due to Dual Infection of Candida albicans and Cytomegalovirus

Stefano Rusconi, M.D., F.C.C.P.; Luca Meroni, M.D.; and Massimo Galli, M.D.

We report a 35-year-old HIV-1-positive man who presented with severe dyspnea and a nonproductive cough. Three fiberoptic bronchoscopic examination revealed an infiltrating and vegetating tracheal mass that was diagnosed as necrotizing candidiasis of the trachea. The lesion resulted in the formation of a tracheoesophageal fistula that eventually led to the death of the patient.

Postmortem examination showed cytomegalovirus vascultis in the esophageal wall. (Chest 1994; 106:254-55)

CMV = cytomegalovirus; TE = tracheoesophageal

Tracheoesophageal (TE) fistulas caused by Candida albicans are rare, even in HIV-1-positive patients. Despite a high frequency of oral and esophageal candidiasis in HIV-1-positive patients, respiratory tract infections caused by Candida species are quite rare. We present an HIV-1-infected man who died as a result of complications of a TE fistula.

CASE REPORT

A 35-year-old HIV-1-positive man was hospitalized in January 1991 for evaluation of worsening dyspnea. The diagnosis of HIV-1 infection had been made 5 years prior to hospital admission.

In October 1989, the patient presented with an interstitial pneumonia. Although Pneumocystis carinii could not be isolated, he responded to therapy with trimethoprim/sulfamethoxazole. He had no other AIDS-defining illnesses prior to this hospital admission and was receiving no medication. On physical examination, the blood pressure was 130/80 mm Hg, the pulse was 80/min, the respiratory rate was 20/min, and he was afebrile. Head and neck examination revealed purulent discharge from the right ear, but no evidence of oral thrush. Findings from examination of the chest and abdomen were unremarkable. No skin lesions or lymphadenopathy was noted.

Results of hematologic laboratory evaluation were normal, with the exception of a CD4+ lymphocyte count of 3/mm³. Serum biochemistry study revealed the following: a glucose level of 67 mg/dl, K+ of 2.5 mEq/L, Na+ of 135 mEq/L, Cl− of 90 mEq/L, Ca²⁺ of 6.6 mg/dl; total bilirubin of 2.7 mg/dl; and direct bilirubin of 2.1 mg/dl. A chest radiograph revealed a right basilar infiltrate. Culture of sputum and the right ear discharge were positive for Streptococcus pneumoniae.

Antibiotic therapy was initiated with amoxicillin, 1 g every 8 h orally. Fiberoptic bronchoscopy revealed the presence of a vegetating and infiltrating mass involving the upper third of the trachea. On the eighth hospital day, his temperature spiked to 39°C and his dyspnea continued to worsen.

Hydrocortisone therapy, 20 mg intramuscularly every 24 h, was started and the amoxicillin was changed to ceftriaxone, 1 g intramuscularly every 24 h. Three consecutive tuberculin skin tests using 10, 50, and 100 IU of PPD were nonreactive at 48 h. X-ray tomography of the larynx and trachea demonstrated a gross filling defect starting 3 cm below the beginning of the trachea and extending inferiorly for 10 cm. The lumen of the trachea was irregular and almost completely occluded.

On the 25th hospital day, viral culture of peripheral blood polymorphonuclear cells was positive for cytomegalovirus (CMV) and he was started on a regimen of intravenous ganciclovir, 250 mg every 12 h. As well, because the patient was still febrile (39 to 39.5°C), imipenem therapy, 500 mg every 6 h, was started and the ceftriaxone therapy was discontinued.

Tracheoscopy revealed a necrotic mass extending into the wall of the trachea and eroding the cartilage commencing 3 cm below the beginning of the trachea and extending inferiorly for 7 cm. Collapse of the walls of the trachea was also noted. A tracheal prosthesis could not be placed due to the length of the lesion. Histologic examination of biopsy specimens showed necrotizing candidiasis of the trachea. Examination for Mycobacterium tuberculosis and Mycobacterium avium-intracellulare was negative. The patient was started on a regimen of fluconazole, 400 mg intravenously every day. The chest radiograph was unchanged at this time.

A third fiberoptic bronchoscopic exam revealed no change. Microscopic examination of the bronchoalveolar aspirate was positive for Staphylococcus aureus for which the patient was treated with vancomycin, 1 g intravenously every 24 h.

The patient continued to be dyspneic and febrile and on the 50th hospital day, the antifungal therapy was changed to amphotericin B, 5 mg/d intravenously, gradually increased to 30 mg/d. A barium esophagogram performed on the 61st hospital day revealed passage of the barium into the trachea and bronchi (Fig 1).

![Figure 1. The barium esophagogram demonstrated a passage of the barium into the trachea and bronchi.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21696/ on 06/26/2017)
His clinical status continued to deteriorate with worsening dyspnea, and he died on the 67th hospital day of cardiorespiratory arrest. Post-mortem examination showed a TE fistula of 5.0 cm in diameter (Fig 2) and CMV vasculitis in the esophageal wall.

**DISCUSSION**

The patient reported herein is unique in that the TE fistula can be correlated both to a deep esophagitis due to CMV and to an invasive tracheal candidiasis.

Organisms causing lower respiratory tract infections in HIV-1-infected persons include fungi such as *Cryptococcus neoformans*, *C albicans*, and *Histoplasma capsulatum*; bacteria, protozoa, and viruses. *Candida albicans* is considered to be the most common fungus isolated from respiratory secretions of patients with HIV-1 infection, in many cases representing oropharyngeal colonization.

*Candida albicans* and viruses, such as herpes simplex virus and CMV, may produce esophagitis in HIV-1-positive patients. In our case, histologic findings at autopsy showed CMV inclusions with associated vasculitis in the esophageal wall.

Another report in the literature has described the development of a TE fistula due to candidiasis and aspergillosis of the esophagus with subsequent spread into the respiratory tract. Candidal pneumonia was also found to be secondary to aspiration through a TE fistula in a patient with AIDS.

To the best of our knowledge, our patient represents the first description of a TE fistula caused by a dual infection with CMV and *Candida albicans*, as supported by the histologic examination of the trachea and esophagus. This case indicates the potential development of a TE fistula in an HIV-1-infected patient with both infectious tracheitis and esophagitis.

ACKNOWLEDGMENTS: The authors thank Tony Mazzulli, M.D., for helpful comments, and Lua Vago, M.D., for review of the post-mortem findings.

**REFERENCES**


**Intrapleural Omentum Simulating Pleural Effusion**

Yohet Sekiguchi, M.D.; Sanae Shimura, M.D.; and Tamotsu Takishima, M.D., F.C.C.P.

We report herein a case of Morgagni hernia of omentum into the pleural space, simulating a pleural effusion on a routine chest radiograph. A 62-year-old man was referred to our clinic for close examination of a pleural effusion-like shadow at the right costophrenic region. He had no history of trauma and no symptoms. Chest computed tomographic scan showed a pleural effusion-like shadow with a fat density. Thoracoscopy revealed a movable omentum-like mass and no significant fluid in the right pleural space. Magnetic resonance imaging and celiac angiography confirmed the herniation of omentum into the right pleural space. This case suggests that a Morgagni hernia must be excluded in a patient with a fat density effusion-like shadow in the pleural space.

Herniation of omentum through the formation of Morgagni is rare and represents only 3 percent of diaphragmatic hernias. Patients with hernia of Morgagni are usually asymptomatic and present with right cardiothoracic angle opacification on routine chest radiograph, since a hernia sac contains colon or omentum. We report herein a case of omentum herniation through the foramen of Morgagni into the right pleural space, simulating effusion on a routine chest radiograph.

**CASE REPORT**

A 62-year-old man had spontaneous right upper abdominal pain and thereafter abdominal discomfort; he was believed to have a gallstone in 1988. In another hospital, he had received various examinations, including a chest radiograph that failed to find a definitive cause. A year later, in 1989, an abnormal shadow appeared in the right costophrenic region on a routine chest radiograph. Various examinations failed to find its cause. A thoracentesis was attempted, but no fluid could be obtained. For pleural effusion of unknown origin, however, he received diuretics and glucocorticoids (prednisolone, 40 to 10 mg/d for 1 month). The treatment with these drugs did not produce any significant alterations in the apparent pleural effusion in the chest radiograph, although the shadow did change independently of any treatments.

*From the First Department of Internal Medicine, Tohoku University School of Medicine, Sendai, Japan.

Reprint requests: Dr. Shimura, 1st Department of Internal Medicine, Tohoku University School of Medicine, 1-1-1 Seiryo- machi, Aobaku, Sendai 980, Japan.*