cases of septic pulmonary emboli, our case is unique for such a source being clinically occult. However, antecedent dental symptoms, periodontal disease, bacteriology of the nodules, and the absence of any other infectious source all suggested the gingiva as the source for the pulmonary abscesses in our patient. Toothbrushing, dental flossing, and use of dental irrigation devices are all important predisposing factors for gingiva-related bacteremia. The occurrence of infective endocarditis from dental foci in the absence of any history of dental procedures supports the notion that gingiva-related bacteremia could indeed be clinically significant. In our patient, the history of toothache antedating his illness was the harbinger of a localized periodontal infection. We believe that the latter could have set the stage for subsequent bacteremia resulting in seeding of the lungs that ultimately led to multiple pulmonary abscesses. Finally, anaerobic infections of the lung are characteristically polymicrobial, but the demonstration of only a single isolate from the pus might be related to the intercurrent antibiotic therapy.

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Simultaneous Legionellosis and Invasive Aspergillosis in an Immunocompetent Patient Newly Treated With Corticosteroids

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Invasive pulmonary aspergillosis (IPA), although unusual, has been recognized in the immunocompetent host. Several cases of IPA with rapidly progressive respiratory failure have been reported in patients receiving short-term corticosteroid therapy for chronic obstructive pulmonary disease. Atypical pneumonia caused by dual infection with Legionella pneumophila and Mycoplasma pneumoniae has also been reported. We report an unusual case of simultaneous L. pneumophila pneumonia and IPA in an asthma patient with suspected allergic bronchopulmonary aspergillosis newly treated with corticosteroids.

(Ches 1993; 104:1929-31)

IPA = invasive pulmonary aspergillosis
ABPA = allergic bronchopulmonary aspergillosis

Invasive pulmonary aspergillosis (IPA) typically occurs in immunocompromised hosts and is frequently fatal.

Case Report

A 71-year-old man was admitted to the Medical ICU with a 4-day history of increasing dyspnea, wheezing, and cough productive of whitish sputum that did not respond to inhaled bronchodilator therapy with ipratropium bromide, theophylline, 400 mg every day, oral amoxicillin, 500 mg 3 times daily, and 40 mg of prednisone daily for 5 days. His medical history was significant for bronchial asthma for 10 years, chronic sinusitis, and nasal polypectomy. He had not received previous corticosteroid therapy. He was a non-smoker and denied alcohol consumption. Initial chest radiograph showed a left mid lung field infiltrate. Sputum cultures showed normal flora with light yeast and mold. Empiric treatment with

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intravenous (IV) ampicillin/sulbactam sodium, 1.5 g every 6 h, for presumed bacterial pneumonia, methylprednisolone, 40 mg every 6 h, and albuterol sulfate aerosol every 2 h was initiated. Subsequent chest radiograph 5 days after hospital admission showed worsening left mid lung field infiltrate and partial atelectasis of the left upper lobe (Fig 1). Sinus radiographs showed an air fluid level in the right maxillary sinus. Sputum fungal culture grew *Candida albicans* and *Aspergillus flavus*. A fiberoptic bronchoscopy confirmed the left upper lobe atelectasis was due to mucous plugging. Culture of the bronchoalveolar lavage fluid grew both *A. flavus* and *fumigatus* serotype 1. The patient improved on a regimen of IV corticosteroids and was discharged home after 3 weeks of receiving 20 mg of prednisone twice daily. Hospital discharge diagnosis was an exacerbation of bronchial asthma secondary to sinusitis, mucous plugging, and suspected allergic bronchopulmonary aspergillosis.

A week later, he was readmitted to the hospital with worsening dyspnea, wheezing, and a fever of 39.3°C. Physical examination revealed an acutely ill elderly man in moderate respiratory distress with a respiratory rate of 24/min. He had oral thrush. Lung examination revealed diffuse wheezing and bibasilar rales. There was mild hepatomegaly. No skin lesions were noted. Chest radiograph now showed bilateral lower lobe infiltrates (Fig 2). Initial leukocyte count was 3,900 with 56 percent neutrophils, 31 percent band forms, 8 percent lymphocytes, 4 percent atypical lymphocytes, and 1 percent monocytes. Arterial blood gases on 2 L of oxygen by nasal prongs were pH of 7.53, PAO2 of 37 mm Hg, and PaO2 of 50 mm Hg. Empiric antibiotic therapy with piperacillin sodium and gentamicin was instituted. Treatment with methylprednisolone, 40 mg every 6 h, was begun. Repeated sputum cultures showed normal flora, rare *Pseudomonas aeruginosa*, and yeast. There was no growth on blood cultures. A computed tomographic scan of the sinuses revealed right maxillary mucosal thickening, bilateral ethmoid and sphenoid opacification, and a questionable mass involving the sella turcica. He was transferred to another hospital for magnetic resonance imaging of his sinuses and possible surgery.

At this time, he developed a right plural effusion. A diagnostic thoracentesis revealed a serosanguineous exudative effusion with lactate dehydrogenase level of 4,983 IU/L, WBC of 20,275 cells per cubic millimeter, predominantly segmented neutrophils, and RBC count of 20,875. Pleural fluid culture grew *Legionella pneumophila* serogroup 1 (a community-acquired species). Treatment with IV erythromycin was initiated and a chest tube was placed into the right pleural space for drainage. Worsening respiratory distress and hypoxemia resulted in intubation and mechanical ventilation. He required inotropic support with dopamine hydrochloride. Turbinate biopsy specimen and sinus drainage showed no invasive aspergillosis or granuloma, but the culture revealed *P. aeruginosa*. Repeated computed tomographic scan of the head revealed a left cerebellar lesion and a pituitary mass with extension into the sphenoid sinus. After 4 days of intravenous amphotericin B, treatment with it was discontinued. He showed clinical improvement and remained afebrile on a regimen of erythromycin although the radiographic appearance of bibasilar pneumonia was unchanged. Treatment with other antibiotics was discontinued. On the 11th hospital day, the patient unexpectedly developed acute massive hemoptysis and fatal respiratory arrest. Autopsy revealed disseminated invasive aspergillosis involving the lungs (Fig 3), myocardium, thyroid, brain, and adrenals. An incidental pituitary adenoma was found.

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21678/ on 04/29/2017)

**Figure 1.** Posteroanterior chest radiograph demonstrating left mid lung field infiltrate and atelectasis of left upper lobe with elevation of left hemidiaphragm.

![Figure 2](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21678/ on 04/29/2017)

**Figure 2.** Posteroanterior chest radiograph showing bilateral lower lobe infiltrates.

![Figure 3](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21678/ on 04/29/2017)

**Figure 3.** Photomicrograph of involved lung showing extensive invasion with *Aspergillus* hyphae (methenamine silver stain, original magnification × 180).
DISCUSSION

This case is of interest for three reasons. First, it illustrates the diagnostic and therapeutic dilemma encountered when Aspergillus species are recovered from respiratory secretions; second, the patient is suspected of having had ABPA that progressed to IPA; and last, it illustrates that two infections can occur simultaneously in an immunocompetent patient receiving corticosteroid therapy for a short time.

Isolation of Aspergillus from a nasal smear or culture strongly suggests active infection, although rare false-positive results have been reported in patients with acute leukemia. Recovery of Aspergillus organisms by culture of respiratory secretions has low sensitivity and uncertain specificity for active infection. However, in immunosuppressed but nonneutropenic individuals either with a transplant, lymphoma, or receiving corticosteroid therapy, isolation is worrisome and invasive diagnostic measures are indicated. Recent data examining the role of bronchoalveolar lavage in immunosuppressed hosts suspected of having IPA reveals the finding of hyphae in the lavage fluid to be a specific but not a sensitive indicator of IPA. In our case, Aspergillus species was recovered by bronchoalveolar lavage and ultimately represented an invasive infection.

We suspect this patient had ABPA that became invasive while he was receiving high-dose corticosteroid therapy. Although the usual diagnostic laboratory criteria of ABPA of peripheral eosinophilia, elevated serum IgE levels, and serum precipitins to Aspergillus were not done as the patient was already receiving high-dose corticosteroid therapy, we believe the clinical presentation, chest radiograph, and bronchoscopic findings are consistent with the diagnosis.

Evidence of tissue invasion by Aspergillus either on presentation or as a possible complication of corticosteroid therapy has been reported previously in cases of Aspergillus-induced hypersensitivity pneumonitis. Recently, a case of rapidly progressive respiratory failure and fatal disseminated aspergillosis was reported in a patient with emphysema receiving short-term corticosteroid therapy (60 mg of prednisone for 2 to 3 weeks) by Crean et al. To our knowledge, although limited tissue invasion has been seen in ABPA, a previous report of IPA evolving from ABPA has not been reported in the literature.

Legionellosis is now recognized as a common cause of community-acquired pneumonia while at the same time it has also emerged as a common cause of nosocomial pneumonias. Acute Legionella pneumonia is associated with high mortality. Concomitant infection with both bacterial and opportunistic organisms in an immunocompromised host must be considered and dealt with aggressively. Our patient initially appeared to respond to treatment.

In conclusion, we believe that the compromised condition of our patient coupled with the difficulty in making the diagnosis accounted for the patient's death due to invasive aspergillosis. Amphotericin B remains the treatment of choice for Aspergillus infection.

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Resolution of Coronary Ischemic Syndrome due to Dislodgement of Intraluminal Thrombus During Diagnostic Cardiac Catheterization*

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Dislodgement of an intraluminal coronary thrombus occurred in a patient with unstable angina during diagnostic cardiac catheterization. The thrombus propagated into the systemic circulation without clinical manifestation of embolism. The procedure rendered the patient asymptomatic. The case illustrates the role of intraluminal coronary thrombus in unstable angina. (Chest 1993; 104:1931-33)

PTCA = percutaneous transluminal coronary angioplasty

The role of intraluminal coronary thrombus in acute ischemic syndrome is now well recognized. Most transmural myocardial infarctions are caused by thrombotic occlusion of a coronary artery. Although the relationship of thrombus and unstable angina is less clear, there is a body of information pointing to an important role of thrombosis in this syndrome. We describe a case in which dislodgement of an intraluminal coronary thrombus occurred during diagnostic cardiac catheterization with subsequent resolution of ischemic symptoms.

CASE REPORT

A 61-year-old man developed exertional chest pain during the spring of 1990. Coronary angiography demonstrated an 80 percent

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