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.

Legionelliosis 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...
stenotic lesion in the proximal segment of a dominant right coronary artery and a left coronary artery free of a significant occlusive disease. Percutaneous transluminal coronary angioplasty (PTCA) of the proximal right coronary artery was successfully performed in July 1990; however, 3 months later, symptoms recurred and thallium stress testing demonstrated inferior wall ischemia. Repeated cardiac catheterization revealed restenosis and PTCA was again successfully performed in October 1990. The patient remained asymptomatic until January 1991, when unstable angina recurred. Repeated thallium stress testing demonstrated an inferior wall perfusion defect, following which coronary angiography was performed. The right coronary artery was cannulated with a standard Judkins right 4-cm catheter. During the first injection (manual) of nonionic dye into the right coronary artery, a migrating filling defect was noted. The first impression was that a small air bubble had been injected, but, on closer observation on video replay, it was observed that the filling defect was propagating retrogradely from a stenotic site to the coronary ostium. No symptoms or signs of systemic embolization were observed. The procedure was completed without complications. Cineangiograms were evaluated using electronic calipers: the right coronary artery displayed a tubular lesion in its proximal third with the greatest stenosis comprising the lumen diameter by 55 percent (1.9-mm lumen) and a 1.2×2.8-mm filling defect moving upstream from the lesion to the coronary ostium and into the aortic root (Fig 1). The left coronary artery was unchanged from the previous study. Thallium stress testing repeated 4 days after cardiac catheterization showed a significant improvement in inferior wall perfusion, and a repeated study 3 months later showed no evidence of perfusion defects (Fig 2). Following this diagnostic catheterization, the episodes of chest pain abated and treatment with medication was gradually withdrawn. Twelve months after the procedure, the patient remains asymptomatic.

**DISCUSSION**

Unstable angina illustrates an acute ischemic syndrome with high prevalence of complex lesions and intracoronary thrombi. Intraluminal filling defects overriding atherosclerotic plaques are frequently found during angiography in patients with unstable angina, and angiography has visualized thrombi associated with these lesions. Dislodgement and distal propagation of these thrombi during diagnostic or interventional procedures usually result in ischemic events. Several studies attest to the poor results of coronary angioplasty when performed immediately after thrombolytic therapy in patients with large residual thrombus. Similarly,
coronary embolization of thrombotic and atheromatous material has been recognized as a complication of balloon angioplasty of saphenous vein grafts.\(^7\)

In this patient with unstable angina, forceful injection of contrast medium caused significant reflux resulting in retrograde propagation of a filling defect. This filling defect probably represented thrombus associated with an active coronary lesion. Migration of the filling defect toward the systemic circulation argues against injection of an air bubble. Since the procedure was performed using nonionic dye, one must consider the possibility of injection of a small thrombus formed in the syringe or catheter. However, this would not explain the direction of defect motion or the subsequent improvement in symptoms. The phenomenon of gradual resolution of perfusion defect following reperfusion, as seen in our patient, is well documented in the literature.\(^5\) The significant improvement and eventual normalization of thallium stress images after catheterization, despite reduction in medical therapy, support a causal relationship between the expulsion of suspected thrombus and the disappearance of unstable angina.

**REFERENCES**


**Pulmonary Metastatic Disease in Ameloblastoma***

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**Ameloblastoma** is a rare disease of odontogenic origin with indeterminate metastatic potential. The first site of metastatic disease is usually the lung. We report aggressive surgical treatment of a patient with bilateral disease with five subsequent recurrences. A review of the literature suggests that in the absence of effective chemotherapy or radiation, surgery should be considered the treatment of choice for metastatic ameloblastoma confined to the lung.

(Chest 1993; 104:1933-35)

Ameloblastoma is a rare tumor of odontogenic origin that comprises 1 percent of all tumors and cysts of the jaw. The malignant potential of ameloblastoma cannot be predicted due to the lack of well-defined morphologic criteria. When metastases occur, the most frequent site is the lung. To date, effective chemotherapy or radiation therapy has not been developed and surgery remains the mainstay of any curative option. A case of aggressive management of pulmonary metastases for ameloblastoma and a review of the literature reveals that in the absence of randomized studies, repeated metastasectomy is justified for this difficult situation.

**CASE REPORT**

A 28-year-old man was first diagnosed as having ameloblastoma of the left mandible in 1980. He underwent wide excision, bone graft, and strut placement without incident and was free of disease for 5 years. In 1985, a routine chest radiograph demonstrated bilateral pulmonary metastases and the patient was referred to the National Cancer Institute.

The patient had a median sternotomy in April 1985 and had resection of a 4-cm right middle lobe lesion, 1-cm, 2-cm, and 4-cm right lower lobe lesions, a 0.5-cm left upper lobe lesion, and three 1.5-cm lesions in the left lower lobe. All lesions were consistent with metastatic ameloblastoma. The patient recovered uneventfully but had a recurrence in his lungs 6 months later. At that time, he refused surgery and received 150 mg/m\(^2\) bolus methotrexate followed by 30 mg/m\(^2\)/h for 24 h and 500 mg/m\(^2\)/d of fluorouracil. Despite six cycles of treatment, his condition progressed and he then reconsidered the surgical option.

In May and June 1986, staged thoracotomies were performed. The right side of the chest was explored surgically first, and upper lobe and lower lobe lesions were removed. In June, a left lower lobectomy was performed due to the presence of disease near the hilum. Three nodules were removed with the specimen.

The patient had a recurrence in January 1987 (6 months later) and again refused surgery. This time he underwent five cycles of 80 microg/m\(^2\)/d of cisplatin over 24 h followed by fluorouracil, 800 mg/m\(^2\)/d for 5 days as a bolus. Again, his disease progressed and he agreed to surgery. In January 1988, he underwent right thoracotomy with resection of an upper lobe lesion and two lower lobe lesions. He remained disease free until a recurrence was noted in November 1988. The patient then had staged bilateral thoracotomies in 1989-1990.

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