Figure 2. Magnetic resonance imaging.

in the left precordial leads (V1 and V6) with giant negative T waves strongly suggests that his apical hypertrophic cardiomyopathy is primary with hypertension playing only a small role toward the development of his left ventricular hypertrophy.

Although this is the first description of a patient with apical hypertrophic cardiomyopathy associated with a left atrial myxoma, it is the second report of hypertrophic cardiomyopathy and left atrial myxoma.12 The coexistence may be more than a chance occurrence so that the association of left atrial myxoma should be ruled out when one sees a patient with a hypertrophic cardiomyopathy.

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Spontaneous Pulmonary Hemorrhage Following Coronary Thrombolysis*

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Excessive bleeding is a major concern during the administration of thrombolytic therapy. Although the great majority of these events occur at sites of vascular interrup tion, major gastrointestinal, retroperitoneal, genitourinary, and central nervous system hemorrhage are known to occur: We present a patient who developed spontaneous pulmonary hemorrhage during thrombolytic therapy. Lack of recognition that the lungs too may be a site of spontaneous hemorrhage during thrombolytic therapy may lead to a considerable diagnostic and therapeutic delay. Pulmonary hemorrhage should be considered in the differential diagnosis of patients who receive thrombolytic therapy in whom new roentgenographic pulmonary infiltrates present accompanied by decreases in hematocrit value. (Ches 1992; 101: 1150-52)

Coronary thrombolysis, has become a standard mode of therapy for early treatment of acute myocardial infarction, and bleeding is a well described complication of this therapy. Although most of these events occur at vascular puncture sites, severe hemorrhage from gastrointestinal, retroperitoneal, central nervous system, and genitourinary sites are known to occur. The following report describes a patient who received thrombolytic therapy for myocardial infarction, and subsequently developed new bilateral pulmonary infiltrates, accompanied by a dropping hematocrit value and respiratory embarrassment. Clinical, laboratory, and roentgenographic data indicated pulmonary hemorrhage.

Case Report

A 52-year-old man presented to St. Vincent's Hospital with an acute anterior wall myocardial infarction. Chest pain had begun approximately one half hour prior to presentation. Physical examination demonstrated blood pressure of 130/80 mm Hg, pulse rate of 84 beats per minute, and respiratory rate of 16 per minute. Chest examination revealed clear heart sounds, an atrial gallop, and no murmurs or rubs. Rales were heard at both lung bases. The ECG was significant for an acute anterolateral wall myocardial infarction, sinus rhythm, and a right bundle branch block pattern. Approximately 1 h after presentation, nitroglycerine, *From the Division of Cardiology (Dr. Nathan), Long Island College Hospital, Brooklyn; the 11Department of Medicine, and the 111Division of Pulmonary Medicine, St. Vincent's Hospital and Medical Center of New York (Dr. Torres, Smith, Gagliardi, Rapoport); and the UNY Health Science Center at Brooklyn.

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Spontaneous Pulmonary Hemorrhage following Thrombolysis (Nathan et al)
heparin, and lidocaine were administered by vein. The patient subsequently received 325 mg of aspirin, 25 mg of metoprolol, and 100 mg of rt-PA. Relief of pain and resolution of ST elevations on ECG were noted soon thereafter.

Initial laboratory data revealed a hemoglobin concentration of 15.7 g/dl; hematocrit value, 0.49 percent; white blood cell count, 7.7 x 10^3; mean corpuscular volume, 89.4 NM; platelets, 313,000; prothrombin time, 12.9 s (control 10 to 13 s); and activated partial thromboplastin time, 27.1 s. The initial chest roentgenogram demonstrated a left basilar infiltrate without cardiomegaly or pulmonary vascular congestion.

In the coronary care unit, because of hypotension and persistent sinus tachycardia, a pulmonary artery catheter was inserted without incident. Initial hemodynamic data revealed a central venous pressure of 12 mm Hg; pulmonary artery pressure, 52/19 mm Hg (mean 30 mm Hg); pulmonary capillary wedge pressure, 25 mm Hg; and cardiac index, 3.9 L/min/m².

Approximately 12 h after presentation, the patient experienced recurrent chest pain and was taken to the cardiac catheterization laboratory. At that time, a repeat hematocrit was noted to be 0.36 percent. Coronary angiography disclosed total occlusion of the left anterior descending coronary artery in its proximal portion. The angiographic appearance of the vessel was consistent with thrombus.

Intracoronary urokinase (250,000 units) was infused into the LAD with resultant relief of chest pain, decrease in left ventricular end-diastolic pressure, and improvement in distal coronary blood flow. Thirty-six hours after presentation, a further decrease in hematocrit to 0.32 percent was noted. There was no oozing from either arteriotomy or venipuncture sites. Urinalysis was negative for red blood cells and hemoglobin. Serial stool samples were negative by hemoccult test. Abdominal examination was benign. Sputum was blood tinged without evidence of gross hemoptysis. Forty-eight hours after admission, a repeat hematocrit was noted to be 0.28 percent, and two units of packed erythrocytes were transfused. An arterial blood sample with the patient breathing room air was obtained which displayed a partial pressure of carbon dioxide (PaCO₂) of 39 mm Hg and partial pressure of oxygen (PaO₂) of 58 mm Hg (P[A-a]O₂, 43).

A chest roentgenogram was obtained and displayed bilateral posterior segment infiltrates (Fig 1). Supplemental oxygen was administered by nonbreather mask, and the patient was maintained in the upright position in bed. The pulmonary capillary wedge pressure was 8 mm Hg and right sided cardiac pressures were within the normal range. Additional laboratory results revealed the following values: total bilirubin, 0.8 mg/dl; reticulocyte count, 1.5 percent; haptoglobin, 133 mg/dl; prothrombin time, 15.3 s; partial thromboplastin time, 89.1 s; and fibrinogen, 360 mg/dl. The CT scan of the abdomen failed to disclose any evidence of intraabdominal or retroperitoneal blood.

On the sixth hospital day, the patient still required a nonrebreathing mask to maintain an oxygen saturation of 90 percent (by pulse oximetry). A repeat chest roentgenogram showed worsening of the bilateral infiltrates (Fig 2). The patient was afibrile. Heparin and aspirin were discontinued.

By the ninth hospital day, the patient improved, and an arterial blood gas drawn with the patient breathing room air disclosed a PaO₂ of 90 mm Hg with a corresponding saturation of 97.3 percent. Repeat chest roentgenogram revealed substantial clearing of the infiltrates.

**DISCUSSION**

The majority of complications involved with thrombolytic therapy are primarily related to bleeding. The first TIMI trial reported bleeding, ecchymosis, or hematoma at the catheterization site in approximately two thirds of the patients. A major bleeding event defined as a reduction of hemoglobin of 5 g/dl or more or any intracranial bleed occurred in about 15 percent of patients studied.1 Our patient sustained a 40 percent reduction in hematocrit value during the 48 h period after administration of thrombolytic therapy which we believe is a manifestation of spontaneous intrapulmonary hemorrhage.

The cardinal manifestations of alveolar hemorrhage include hemoptysis, pulmonary infiltrates on chest roentgenogram, anemia, dyspnea, and hypoxemia. It should be noted, however, that life-threatening intrapulmonary hemorrhage can occur in the absence of hemoptysis.2 The chest roentgenogram is a more accurate assessment of the amount of progression of pulmonary hemorrhage than is the amount of hemoptysis.2 The anemia, important diagnostically, should be followed with serial hematocrit determinations to afford a monitor for continued or recrudescent bleeding, especially in cases where hemoptysis is absent.2

**FIGURE 1.** Chest roentgenogram showing wedge shaped peripheral opacities in both mid lung fields.

**FIGURE 2.** Chest roentgenogram showing progression of the midlung infiltrates.
Plasmin-induced platelet dysfunction and the concomitant use of other medications such as heparin, aspirin, beta blockers, calcium channel blockers and intravenous contrast media, as well as the breakdown of physiologic clot, hypo-fibrinogenemia, and the consumption of factors V and VIII may influence the tendency to bleed. Our patient received intravenous rt-PA and intracoronary urokinase, as well as heparin and aspirin and had a prolonged activated partial thromboplastin time, all of which probably contributed to the diathesis. A reptilase time which was not performed might have been useful in differentiating heparin effect from the thrombolytic effect. The possibility that the balloon flotation catheter had a causal role seems unlikely in that the radiologic infiltrates were apparent before insertion of the catheter and they appeared simultaneously. Finally, position of the catheter tip was confirmed by x-ray and the continuous pressure monitoring never suggested "over-wedging."

Although our diagnosis of pulmonary hemorrhage was one of exclusion, Ewan and associates have elegantly shown that measurement of carbon dioxide uptake can be a very sensitive and specific indicator of acute pulmonary hemorrhage. Unlike other alveolar filling processes, alveolar hemorrhage is associated with an increase in carbon monoxide uptake by the lung because of binding of the carbon monoxide to the erythrocytes in the alveoli with a decrease in delivery of carbon monoxide to the arterial blood. This results in an increased ratio of pulmonary uptake to clearance of carbon monoxide.

The approach to the postthrombolysis patient with a waning hematocrit without an obvious etiology should include attention to vascular puncture sites, careful examination of the abdomen, and possibly computerized tomography to image the retroperitoneum. If this does not yield useful information and new infiltrates are present on chest x-ray film, carbon monoxide diffusion capacity or bronchoscopy looking for hemosiderin laden macrophages could be used for further diagnostic information.

We believe this is the first reported case of spontaneous pulmonary hemorrhage during thrombolysis. The incidence of such events may be much higher than realized as infiltrates on chest roentgenogram may be attributed to a pneumonic process or pulmonary vascular congestion and small decrements in hematocrit to phlebothrombosis or hemodilution.

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REFERENCES

Penicillium decumbens* A New Cause of Fungus Ball
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We report the first documented case (to our knowledge) of fungus ball due to Penicillium decumbens in an immunocompetent Japanese farmer, using immunologic and histopathologic techniques.

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IPA = indirect fluorescent antibody

Genus Penicillium consists of more than 900 species and comprises some of the most common airborne fungi. However, its association with human disease has generally remained elusive except for a few instances. Because of a lack of understanding of its potential pathogenicity, it is considered as a contaminant. We herein present the first documented case (to our knowledge) of a fungus ball due to Penicillium decumbens and discuss the pathogenicity.

CASE REPORT

A 57-year-old Japanese female farmer continued to have occasional blood-tinged sputum for several years until April 1987, when she developed 20 ml of hemoptysis. A lateral chest roentgenogram and a computed tomographic scan of her thorax at that time revealed a round density within a thin-walled cavity in the left lower lobe. Results of physical examination and routine laboratory studies were normal. She had no history of pulmonary tuberculosis, chronic obstructive pulmonary disease, asthma, or immunosuppressive illnesses. Histologic examination of a transbronchial biopsy specimen of the lesion revealed fungal hyphae. No growth was obtained on cultures of the biopsy specimens and her sputa. Assessment of her immunologic status showed a normal white blood cell count without eosinophilia, normal serum immunoglobulin levels, and a positive purified protein derivative skin test. The serum precipitating antibodies to Aspergillus antigens were negative. Because of continuing hemoptysis, a left lower lobectomy was performed on Oct 20, 1987. Gross sectioning of the surgical specimen showed a bronchietatic cyst of the posterior basal bronchus that contained a 3 × 1.5 × 1.5-cm brown fungus ball consisting of a dense network of fungal hyphae lying free in the cavity (Fig 1). Microscopically, the inflammatory response in the wall was minimal, and neither fungal invasion nor granulomatous changes were seen. Portions of fungus ball were cultured on Sabouraud's agar and potato dextrose agar at 25°C and at 37°C, respectively. The culture showed a pure and heavy growth at 25°C and a slight growth at 37°C that was identified as P decumbens at Toyo University Research Center for Medical Mycology. To further characterize the pathologic role of the P decumbens isolate, the following immunologic studies were done.

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